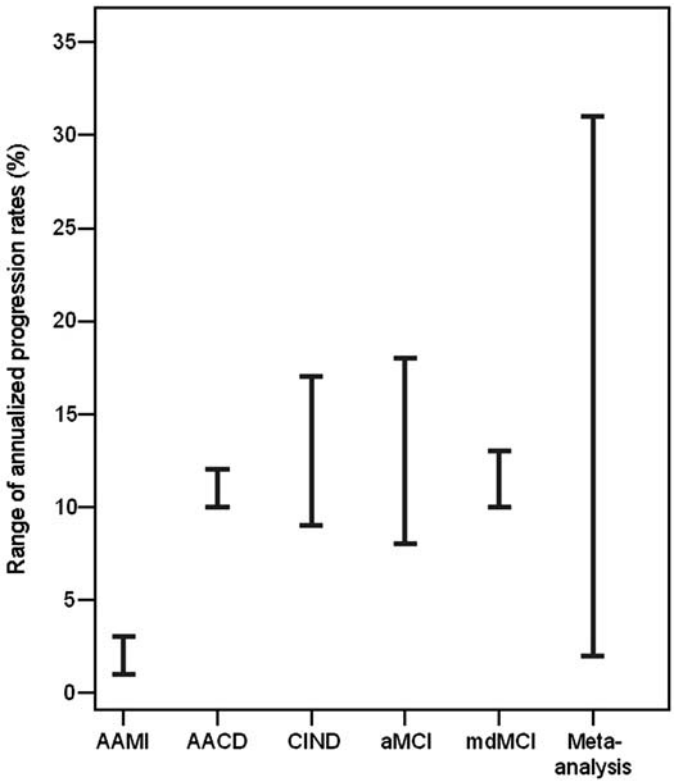

Fig. 8.5.1.1.4 Range of annualized estimates of progression to dementia in various MCI groups, reported in Refs.[13,14,18]

annum, generally over 5-year periods. A meta-analytic study indicated a mean annual conversion rate of 10.24 per cent, with a range of 2 to 31 per cent. The single most important factor accounting for this heterogeneity was the source of the participants, with subjects referred to specialist services, either geriatric or memory/dementia clinics, progressing to dementia at roughly twice the rate found for community volunteers.[18] In studies of CIND annual rates have been 17 per cent for a dementia clinic-referred cohort, 9 per cent for a population-based cohort.[1,9]

An unresolved question is whether all subjects with MCI eventually develop dementia. In a highly selected aMCI sample with CDR 0.5 followed over 10 years, all subjects eventually progressed to meet dementia criteria.[19] This evidence has been proposed to support the belief that aMCI represents early-stage AD.[19] Other studies with similar length of follow-up have not found as high rates of progression and neuropathologic evidence also suggests more heterogeneity, with up to 50 per cent of subjects with aMCI meeting criteria for AD, and the remainder having varied non-AD abnormalities including vascular lesions, argyrophilic grain disease, or other conditions.[20]

(b) Reversion rates to normal

There are consistently proportions of individuals with MCI that will revert back to normal cognitive functioning during follow-up. In population-based studies, this backcrossing has sometimes been particularly high (range 4 to >40 per cent after 1.5 to 5 years).[1] In clinic-referred samples, reversion rates for CIND appear to be lower (14 per cent after 2 years).[9] Reversion may in part be a reflection of an inherent instability in the MCI condition, particularly when it is defined exclusively by psychometric cut points.[1] Reversion appears to be more common when there is no clearly identified aetiology for MCI.[9]

(c) Mortality risk

The mortality risk of MCI is near twice the risk of cognitively normal individuals. The estimated relative risk has been 1.5 for AACD, 1.5–1.9 for CIND, and 1.3–1.7 for aMCI while there has been no increased risk reported for AAMI. There is no clear explanation for this increased mortality risk. It is independent of health conditions such as cardiac disease, cerebrovascular disease, diabetes, and malignancies, and it may be related to incipient and eventually full-blown dementia.[21]

Diagnosis: clinical approach

Overview

Figure 8.5.1.1.5 depicts a flow chart for the clinical diagnosis of MCI.[2] The diagnostic process begins with an expressed concern about cognitive functioning from the patient and/or informant. The assessment then requires a careful history from the patient and informant, as well as the mandatory administration of objective cognitive testing. An evaluation of social function and ADLs, both instrumental and basic, is performed to determine whether there is impairment sufficient for a dementia diagnosis. A clinical judgement must be made of whether the impairment falls outside of the normal range for age and whether it falls within MCI or dementia. The cognitive profile can further be classified into single amnestic, multiple domains, or single non-memory domain. The final step in the diagnostic process is the determination of the aetiology of MCI.

Cognitive assessment

The identification of MCI requires evidence of impairment on objective cognitive testing. Generally, a cognitive screening test is needed as a first step. It should be recognized that the most widely used Mini Mental State Examination (MMSE) lacks sensitivity for MCI diagnosis. In turn, two novel instruments (the Montreal Cognitive Assessment, MoCA, and the DemTect) have been shown to reliably discriminate MCI from normal ageing.[22] The effects of age and educational achievement on test performance and the consequent risk of misclassifications must be kept in mind in the clinical assessment. Irrespective of the instrument that is chosen, there should be coverage of episodic memory, executive functioning and language, which are the most frequent presenting problems in MCI.

Neuropsychological testing (NPT) can be helpful where a clear-cut determination of MCI is difficult following the initial assessment. NPT provides information on the pattern of impairment, with identification of the domains affected and reference to standardized scores. Serial NPT may be particularly useful in defining the progression from MCI to mild dementia. The rate of decline on tests of memory, executive functioning and language accelerates during the 3- to 5-year time window before diagnosis of full-blown dementia.[22] NPT also has some predictive utility in addressing the risk of progression from MCI to dementia. Deficits on tests of episodic memory and executive functioning have consistently been found to characterize those with MCI that will develop dementia.[22]

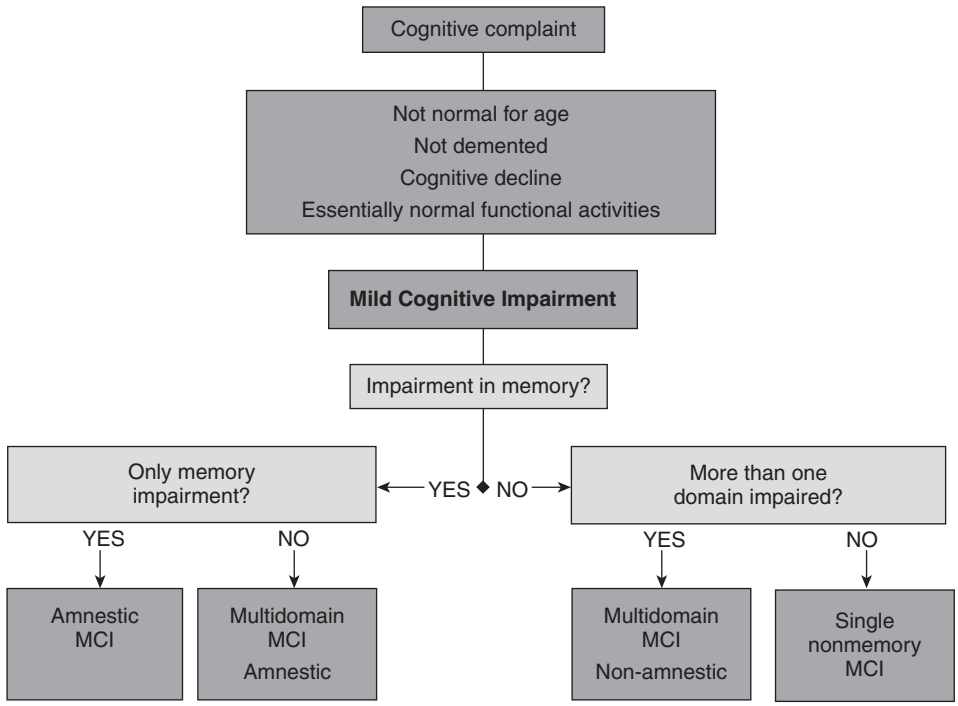

Fig. 8.5.1.1.5 Diagnostic flow chart proposed by the International Working Group on MCI. (Reproduced from B. Winblad *et al.* (2004), Mild cognitive impairment—beyond controversies, towards a consensus: report of the International Working Group on mild cognitive impairment, *Journal of Internal Medicine*, **256**, 240–6, copyright 2004, John Wiley & Sons, Inc.)

Neuropsychiatric symptoms (NPS)

NPS may provide a very useful additional domain for evaluation. An estimated 50 to 70 per cent of subjects with MCI have informant-reported NPS. Symptoms of depression, agitation/aggression, anxiety, apathy, and irritability, while at a low level of intensity, nevertheless each have a frequency of >30 per cent. NPS are associated with greater MCI severity and may be predictive of progression to dementia.[23] The evaluation of NPS is recommended within an MCI assessment.

Aetiology

The determination of aetiology is a final key step in the assessment of MCI. It requires additional laboratory studies as well as consideration of neuroimaging results. Within a cohort of individuals with CIND, the most prevalent aetiologic subtypes were pre-AD, vascular cognitive impairment, cognitive impairment with psychiatric illness, and not otherwise specified (NOS) (Table 8.5.1.1.1). These CIND aetiologic subtypes differed in their functional and psychobehavioural profiles, and in their 2-year prognosis. Pre-AD and vascular CIND had the highest rates of progression to dementia (~40 per cent), with pre-AD subjects developing exclusively probable AD. Psychiatric CIND and CIND NOS had the highest rates of reversion to normal (20 to 30 per cent). Progression to dementia occurred in all aetiologic subtypes (Table 8.5.1.1.1).[9]

Biomarkers

There are no widely accepted MCI biomarkers at the present time. Research has focused on neuroimaging with MRI and FDG-PET as well as on tau and β-amyloid (Aβ) protein levels in cerebrospinal fluid (CSF).

Neuroimaging

On structural MRI, individuals with MCI may show volume loss in the medial temporal lobe (MTL) that may presage AD. The key structures of the MTL include the hippocampus and the entorhinal cortex. MTL structures can be rated on visual scales that may

Table 8.5.1.1.1 Aetiologic subtypes of CIND and their 2-year progression to AD/dementia

Subtype	Prevalence (% within cohort)	Dementia at 2-years (% within subtype)	AD at 2-years (% within dementia)
Pre-AD	24.6	40.9	100
Vascular	18.1	40.0	33.3
Psychiatric	17.3	25.0	75
Non-AD degenerative	2.3	33.3	0
Neurological	7.3	25.0	25
Medical	3.5	60.0	100
Mixed	7.6	33.3	100
Not otherwise specified	19.3	24.2	62.5

(Reproduced from G.Y. Hsiung et al. (2006), Outcomes of cognitively impaired not demented at 2 years in the Canadian cohort study of cognitive impairment and related dementias. *Dementia and Geriatric Cognitive Disorders*, **22**(5–6), 413–20, with permission from S. Karger A.G, Basel.)

reasonably predict AD.[24] MRI-based predictive algorithms may be better when MTL measures are combined with measures of lateral temporal lobe or anterior cingulate structures, or with performance scores on episodic memory.[24] Both whole brain and MTL atrophy rates are greater in MCI subjects that progress to AD than in those who do not.[25]

The characteristic pattern of AD is to have metabolic reductions in temporoparietal and posterior cingulate regions on [^{18}F-]-fluorodeoxyglucose (FDG) PET. This pattern may be seen in individuals with MCI ahead of full-blown disease. Serial PET may show further metabolic deterioration in these areas as well as abnormalities in the ventrolateral prefrontal cortex in subjects who progress to AD.[1] Recently, PET radioligands that bind to cerebral amyloid and potentially to tau proteins have been developed. These include Pittsburgh compound B (PiB) (*N*-methyl-[^{11}C]2-(4′-methylaminophenyl)-6-hydroxybenzothiazole) for amyloid and FDDNP (2-(1-[6-[(2-[^{18}F]fluoroethyl)(methyl)amino]-2-naphthyl]ethylidene)malononitrile) for amyloid and tau. Studies with these ligands have revealed higher than normal retention in subjects with MCI, with a pattern that follows the anatomical distribution of AD pathology.[26,27] If longitudinal follow-up confirms that these MCI cases develop AD, PET with PiB or FDDNP could become a diagnostic test for the early identification of AD.[24]

Cerebrospinal fluid markers

CSF markers can reflect the AD pathogenic process including a reduction in Aβ42 as it aggregates into senile neuritic plaques, as well as an increase in total tau (t-tau) and phospho-tau (p-tau), which signals the hyperphosphorylated state of tau. In MCI, the combination of abnormal Aβ42, t-tau, and p-tau 181 is associated with a 17–20 increased risk of developing AD over 4–6 years. Currently, the utility of these markers is limited by the lack of a standardized assay and the variability in measurements obtained at different laboratories.[28]

Management and treatment

There are no standard therapies for MCI. A clinical management plan is formulated on an individual basis in consideration of the cognitive pattern of MCI and its aetiology. There are two goals for treatment: first, to alleviate the cognitive symptoms of MCI, and second, to attempt to delay the onset of dementia in those at risk.

Symptomatic treatment

(a) Pharmacotherapy

A 24-week trial of the cholinesterase inhibitor (ChEI) donepezil did not benefit subjects with aMCI on its primary endpoints of delayed recall and global impression of change. There were benefits on secondary measures including the ADAS-cog, neuropsychological tests of attention, and patient-rated global function (Patient Global Assessment, PGA).[29]A longer term trial of donepezil with subjects with aMCI (the Alzheimer's Disease Cooperative Study-Memory Impairment Study, ADCS-MIS) demonstrated benefits on the ADAS-cog, memory and language scores, and global measures including the CDR sum of boxes but these were confined to the first 18 months of treatment.[30]

(b) Non-pharmacologic therapies

Cognitive and lifestyle interventions may help the cognitive and behavioural difficulties in MCI. An 8-week cognitive intervention programme to improve memory strategies produced benefits on tests of delayed recall and face-name association, and on self-assessed everyday memory function in subjects with aMCI.[31] A 14-day healthy lifestyle programme with memory training, physical conditioning, relaxation techniques, and a diet plan, showed benefits in subjects with mild self-reported memory complaints. These benefits included improved word fluency and metabolic changes on FDG-PET in dorsolateral prefrontal cortex.[32] Therapeutic approaches of this type require considerable resources, and confirmation in randomized controlled trials (RCTs) is needed before large-scale implementation in MCI can be recommended. Nevertheless these approaches hold some promise.

Delaying the onset of AD

(a) Pharmacotherapy

A considerable number of long and large trials of ChEIs and non-steroidal anti-inflammatory drugs (NSAIDs) have been directed at delaying the time to diagnosis of AD in those with aMCI. In the 3-year ADCS-MIS trial, treatment with donepezil did not have an effect on the primary outcome measure of progression to AD after 36 months. An interesting observation in the study was that carriers of the apolipoprotein E ε4 allele treated with donepezil had a reduced risk of progression to AD at all time points.[30] Similarly, rivastigmine was unsuccessful in delaying the time to diagnosis of AD.[33] A 4-year trial of the COX-2 inhibitor rofecoxib did not show treatment benefits on the primary endpoint of percentage of subjects that were diagnosed with AD nor on secondary measures of cognition and global function.[1] Based on the available data, the long-term use of ChEIs or NSAIDs in MCI, with the goal of delaying AD onset, cannot be recommended.

(b) Non-pharmacologic therapies

An expanding literature supports the hypothesis that a cognitively, socially, and physically active lifestyle in late life may reduce the risk for AD.[34] Evidence from RCTs to support this hypothesis is not currently available and it is premature to recommend organized interventions. There would be little perceived harm in promoting an engaged lifestyle as part of the management of MCI.

The future of MCI

MCI has been a useful construct to focus attention on the cognitive impairment that is going to increase exponentially within our greying societies. It is recognized that MCI is a risk state for developing AD and other dementias. A natural next step will be to develop criteria to diagnose AD earlier. A proposal of research criteria for the early diagnosis of AD has recently been published.[24] The diagnosis builds on a clinical core of early and significant episodic memory impairment and requires in addition the presence of at least one biological footprint of the disease: medial temporal lobe atrophy on structural MRI, abnormal CSF, or reduced temporoparietal glucose metabolism on FDG-PET.[24] Similar frameworks are already in place or will be developed for non-AD dementias. As these frameworks will advance, the concept of MCI will likely be significantly refined and could look quite different by the next edition of this textbook.

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8.5.2 Substance use disorders in older people

Henry O'Connell and Brian Lawlor

Introduction

This chapter is divided into three main sections, focussing respectively on alcohol use disorders (AUDs), medication use disorders (MUDs), and use of illegal substances and nicotine, in older people. In each section we focus in detail on definitions and diagnosis, epidemiology, aetiology, clinical features, investigations, screening, management, and prognosis. More is known about AUDs in older people, hence this section is the longest, but MUDs in older people is also a significant problem and abuse of illegal drugs may become increasingly important in future years.

Alcohol use disorders (AUDs) in older people

Introduction

The ageing of populations worldwide means that the already significant problem of alcohol use disorders (AUDs) in older people is likely to become even more important in future years. However, AUDs in older people are neglected and underdiagnosed, for the reasons outlined in Table 8.5.2.1, and unless these factors are tackled proactively there exists a real danger of AUDs in older people becoming a silent epidemic, with negative impacts on all aspects of health and well-being[1] (see Table 8.5.2.2).

Definitions and diagnosis

AUD is a general and broad term, used to include a wide range of alcohol-related problems, as outlined in Table 8.5.2.3. Alcohol status may also change throughout life, with one-third of older people with AUDs developing such problems for the first time in later life (late-onset AUDs). A more severe course of AUD, higher levels of antisocial personality and stronger family histories of AUDs are seen in those with early-onset AUDs.

Because of the effects of physical and cognitive ageing, pharmacokinetic changes, the increased prevalence of comorbid illness, and interactions with prescribed medication, older people are likely to encounter AUDs at levels of intake lower than the general population. Therefore, the recommended levels of intake for the general population (i.e. up to 21 and 14 units per week for men and women, respectively[2]) may be inappropriately high for older people. However, apart from the NIAAA recommendations of no more than one drink per day for older people,[3] there is a lack of guidance on safe levels of alcohol intake, and the pursuit of obvious and 'down and out' drinkers may lead to a significant amount of more subtle and clinically 'silent' AUDs being missed.

Furthermore, the diagnostic criteria used by ICD-10 and DSM-IV used to describe harmful use and alcohol dependence syndrome may not be applicable to older people, as evidence of diagnostic criteria such as craving, compulsion, tolerance, and withdrawal features may be less clear-cut and masked by other medical conditions, and older people may be less likely to encounter the financial, occupational, family and legal consequences of AUDs (see also Table 8.5.2.1 and section on screening).

Table 8.5.2.1 Reasons for the neglect and underdiagnosis of AUDs in older people

Patient factors
Older people may be less likely to volunteer information on alcohol intake/AUDs
Recall of alcohol intake may be inaccurate due to cognitive impairment
Features of AUDs may be atypical or masked (e.g. presenting as falls, confusion)
Pharmacokinetic changes, comorbid illness, and drug-interactions mean alcohol-related problems may arise even at relatively low levels of intake
Health service factors
Health care professionals less likely to ask older people about alcohol intake and AUDs
Health care professionals less likely to refer older people for treatment, even when AUD detected
Inappropriate screening and diagnostic tools used
Therapeutic pessimism in treating older people
Inappropriately high levels for 'recommended' or 'healthy' levels of intake
Family and societal factors
Family members may be less likely to perceive AUD as a problem in older relatives
Ageist attitudes lead to risk of AUDs in older people being perceived as 'understandable'
AUDs in older people less 'noisy', with less impact on absenteeism, antisocial behaviour, crime

Epidemiology

The prevalence of AUDs in older people varies depending on the screening and diagnostic criteria used, clinical and socio-demographic characteristics (men having levels 4–6 times higher than women) and the level of severity of AUD being defined. In community-based studies, for example, 2–4 per cent of older people have been estimated to have alcohol misuse or dependence,[4] with higher rates of 16 per cent (men) and 2 per cent (women) when looser criteria such as excessive alcohol consumption are used.[5] Clinical populations of older people have higher levels of AUDs, with emergency department, nursing home and psychiatric inpatients being described as having levels of 14, 18, and 23 per cent, respectively.[6–8]

The true prevalence of AUDs in older people is often underestimated, for the reasons outlined in Table 8.5.2.1. It is likely, however, that the actual levels of alcohol consumption and AUDs do decline with age.[9,10] This decline may be due to factors such as premature death of those with AUDs, reduced physiological reserve and comorbid medical illness leading to reduced alcohol intake, age-cohort effects and age-related changes in social networks, occupational, and financial status.

Aetiology, risk factors, and associations

These factors can be broadly described as being biological/medical, social, and psychological in nature. Genetic factors are likely to be important in relation to both early-onset[11] and late-onset AUDs.[12] The genetic risk for AUDs may also overlap with risk for other mental disorders such as antisocial personality disorder, other drug use problems, anxiety disorders, and mood disorders.[13] AUDs may have a cause and effect relationship with medical illness.

Important social aetiological factors are likely to include male gender, bereavement, age-cohort effects, culture and ethnicity, religion, and marital status (higher levels of AUDs in divorced and single). Some social factors, such as marital problems, may have a two-way relationship with AUDs.

Table 8.5.2.2 Physical, neuropsychiatric, and socio-demographic aspects of AUDs in older people

1. Physical factors
Gastrointestinal
Hepatic problems: elevated liver enzymes; fatty liver; alcoholic hepatitis; cirrhosis; malignancy
Gastritis, peptic ulcer disease, and bleeding
Oesophageal varices
Acute and chronic pancreatitis
Malignancies
Mouth, pharynx, larynx, oesophagus, hepatic, colorectal, pancreatic
Cardiovascular
Ischaemic heart disease
Hypertension
Alcohol-induced arrhythmias
Congestive heart failure
Alcoholic cardiomyopathy
Haematological
Macrocytosis (acute effect of alcohol intake and due to vitamin B12 and folate deficiency in chronic AUD)
Anaemia (due to gastrointestinal problems)
Musculoskeletal
Falls and fractures
Reduced bone density
Myopathy
Metabolic
Hypoglycaemia
Hyperuricaemia
Elevated lipids
Diabetes more difficult to control
2. Neuropsychiatric factors
Cognitive impairment and dementia
Frontal lobe impairment
Wernicke–Korsakoff syndrome
Cerebellar cortical degeneration
Central pontine myelinosis
Marchiafava–Bignami disease
Depression
Psychosis
Intoxication
Withdrawal syndrome (may be more difficult to treat in older people)
Suicide
3. Socio-demographic
Male gender
Divorced, widowed, and single status
Social isolation
Upper and lower ends of socio-economic spectrum
4. Other
Alcohol–drug interactions
Aspiration pneumonia
Road traffic and other accidents

Relevant personality factors include the stronger association between antisocial personality, hyperactivity, and impulsivity in 'early-onset' compared to late-onset AUDs, who may have higher levels of 'neuroticism' and depression.[14]

AUDs in older people, as in all populations, may also have a two-way relationship with psychiatric disorders such as depression and anxiety disorders. For example, an older person may begin drinking in an effort to self-medicate depressive symptoms, or they may become depressed because of their drinking.[15]

Table 8.5.2.3 Types/levels of severity of AUDs

- Excessive alcohol consumption (i.e. drinking above recommended levels)
- Binge drinking (i.e. episodic bouts of excessive alcohol intake)
- Problem drinkers/harmful use/abuse
- Alcohol dependence syndrome
- 'Early-onset' versus 'late-onset'

Clinical features and comorbidity

AUDs in older people are linked to significant morbidity and mortality, affecting practically all aspects of physical, neuropsychiatric and social health and well-being,[1,16] as summarized in Table 8.5.2.2.

Pharmacokinetic changes (reduced physiological reserve, reduced metabolic efficiency, and increased volume of distribution due to a higher fat to lean muscle ratio, leading in turn to relatively higher blood alcohol concentrations in older people) along with the general effects of physical and cognitive ageing, increasing frailty, reduced functional ability, and higher levels of concomitant prescription drug use means that alcohol is relatively more toxic to older people than younger people. Furthermore, as outlined earlier, such toxic effects may be subtle and may be missed or mistaken for other conditions.

AUDs in older people are associated with a wide range of mental disorders, such as depression, psychosis, withdrawal syndromes, cognitive impairment, and dementia[17] (see Table 8.5.2.2) and are also associated with an increased risk of suicide.[18] The relationship between alcohol use and brain damage and dementia is complex,[19] in that AUDs may increase the risk for different types of dementia[20] and there also exist diagnostic entities known as 'amnesic syndrome associated with alcohol use' (ICD-10) or 'alcohol-induced persisting dementia' (DSM-IV). In contrast, light to moderate alcohol use may protect against dementia.[21]

(a) Clinical assessment

The assessment of AUDs in older people begins with a thorough clinical interview and history of alcohol use (quantity and frequency of drinking, beverage type, drinking context), mental state examination, physical examination and collateral history if available, and with the patient's consent. If indicated by the initial history, additional questions should be asked about features of alcohol dependence syndrome, as they relate to the patient's physical and psychosocial health. Questions should be framed in a sensitive and non-judgemental way, as patients may disengage and be lost to treatment and follow-up if they feel threatened by the assessment procedure.

(b) Further investigations in AUDs

Following a detailed history and examination, other investigations may be indicated and should be directed by the patient's clinical status. These may include: blood tests to check the following: urea and electrolytes; full blood count; liver function tests; vitamin B12 and folate levels; neuroimaging (CT or MRI brain); gastrointestinal investigations such as ultrasound, CT, or MRI examinations of the abdomen, upper gastrointestinal endoscopy, and liver biopsy; basic cardiovascular investigations such as electrocardiogram and other

more detailed investigations if indicated, e.g. echocardiogram and 24 h blood pressure monitoring.

(c) Screening for AUDs in older people

Screening programmes should aim to detect both clear-cut and subtle cases of AUDs in older people. It must be remembered that screening tests are not diagnostic in themselves, but positive results should lead on to further investigations. Screening methods may be based on self-report alcohol screening instruments such as the CAGE,[22] the AUDIT,[23] and biophysical measures such as blood tests checking mean corpuscular volume and liver function tests.

A systematic review of self-report alcohol screening instruments in older people[24] revealed that the CAGE was the most widely studied, but that sensitivity and specificity varied depending on the clinical characteristics of the population in question. However, the CAGE is the most well recognized alcohol screening instrument and is quickly and easily administered, so the authors would recommend use of at least this instrument, along with further investigations and assessment scales if problems are detected.

The utility of biophysical screening measures such as carbohydrate-deficient transferrin, liver function tests or the mean corpuscular volume may be less reliable in older people,[25] because of higher levels of comorbid physical illnesses leading in themselves to false-positive and abnormal results. However, they may prove useful when combined with other clinical information, both in the detection of AUDs and monitoring of progress through treatment.

Management and prevention

Primary prevention strategies should focus on individual older people (especially considering that one-third of older people have late-onset AUDs) and can also be directed at the entire population, targeting factors such as ease of access to alcohol, restrictions on alcohol advertising, and education about the adverse effects of drinking. Such primary prevention and public health initiatives tend to be directed towards younger individuals, but they should also take into account the more clinically 'silent' AUDs that may develop in older people.[26]

Secondary prevention strategies should focus on older people who already have 'at-risk' drinking, either currently or in the past, and who are at risk of developing worsening problems in the context of diverse factors such as bereavement, social isolation, adjustment to retirement, and physical or psychiatric health problems.

Tertiary prevention involves treatment of existing AUDs. Treatment modalities can be divided into biological/medical, social, and psychological. Biological/medical treatments are most important in the acute setting, where detoxification may be required.

Care should be taken with benzodiazepine-assisted withdrawal in older people, in view of the elevated risk of oversedation, confusion, and falls. There are no elderly-specific guidelines on benzodiazepine-assisted alcohol withdrawal in older people. Lorazepam has been identified in one review[27] as the safest choice of benzodiazepine for treatment of alcohol withdrawal in older people, in view of the fact that advancing age and liver disease have little impact on its metabolism, and absorption by the intramuscular route is predictable. In practice, however, it is likely that there is more clinical experience with use of long-acting benzodiazepines such as chlordiazepoxide. The choice of benzodiazepine used should be based on individual patient characteristics such as previous treatments, current medical status (e.g. degree of hepatic impairment) and an objective measure of withdrawal may help guide the dosing regimen (see Table 8.5.2.4).

Parenteral or oral thiamine should be given to prevent development of the Wernicke–Korsakoff syndrome. A recent review has concluded that, in the emergency department setting, oral thiamine administration is as effective as parenteral administration.[28] Again, however, there are no elderly-specific guidelines, and individual patient characteristics must be taken into account, such as general health, ability to take oral medication, and compliance.

The three medications that are approved by the US Food and Drug Administration to promote abstinence and reduce relapse are Disulfiram, Acamprosate, and Naltrexone.[29] However, the limited efficacy of Disulfiram, combined with the potential for a more severe side-effect profile means it is best avoided in this age group. In contrast, Naltrexone and Acamprosate have been suggested as suitable agents for use in older people.[30]

Psychosocial aspects of treatment should also be explored. This may include addressing social circumstances that may be contributing to the AUD (e.g. personal finances and housing). There is a dearth of evidence on psychotherapeutic approaches to AUDs in older people, but there is some evidence that older people may respond better to psychotherapy in same-age settings,[31,32] and consideration should also be given to support groups such as Alcoholics Anonymous.

Prognosis

The available literature on the topic suggests that older people are at least as likely, if not more likely, to benefit from treatment of AUDs as younger people.[33,34] However, prognosis in older people is likely to vary widely depending on a number of factors relating to the individual themselves and the nature of their AUD, the presence of family and other support systems and the availability of treatment services, particularly services that are tailored to older people.

Table 8.5.2.4 The Clinical Institute Withdrawal Assessment for Alcohol-Revised Version (CIWA-Ar) (Reproduced from The South London and Maudsley NHS Trust Prescribing Guidelines, 2005–2006, copyright South London and Mandsley NHS Foundation Trust)

1. Nausea and vomiting
2. Tremor
3. Paroxysmal sweats
4. Anxiety
5. Agitation
6. Tactile disturbances
7. Auditory disturbances
8. Visual disturbances
9. Headaches and fullness in head
10. Orientation and clouding of sensorium

Severity of alcohol withdrawal	
Mild:	<10
Moderate:	10–20
Severe:	20+

(Items 1–9 are scored from 0–7 and item 10 from 0–4. Maximum possible score is 67)

Medication use disorders (MUDs) in older people

Introduction

High levels of prescribing of all types of medications for older people, which may at times be inappropriate, along with factors such as variable compliance, altered pharmacokinetics, reduced functional ability, and increased levels of physical, psychiatric, and cognitive morbidity mean that older people are at higher risk of developing MUDs than any other age group.[35] As with AUDs, clinical features of MUDs may be atypical and masked by other conditions and thus go undetected and untreated.[36]

Definitions

As with AUDs, older people are affected by a wide range of types and severity of MUD.

The ICD-10 uses the same general principles of intoxication, harmful use, dependence, and withdrawal state that apply to alcohol for use of sedative and hypnotic medications. As with AUDs, elderly-specific criteria are not cited, but the same general principles apply: older people are likely to experience harm at lower levels of use and clinical features guiding diagnosis are more likely to be atypical and masked by other health problems.

Iatrogenic factors are also important, as drugs may be inadequately or underused for treating or preventing conditions, or drugs may be overused, leading to unnecessary exposure of the older individual to adverse effects.[36]

Epidemiology

Older people comprise 13 per cent of the US population, but they have been estimated to use more than 30 per cent of prescription[37,38] and 35 per cent of OTC drugs:[37] it has been estimated that older people use prescription and OTC medications approximately three times as much as the general population. Furthermore, we know that the risk of MUDs increases with polypharmacy, which is common in older people.[39,40]

Benzodiazepines are the most commonly prescribed psychotropic drugs in older people, with one study of community-dwelling older people in Ireland demonstrating that 17 per cent of participants were prescribed benzodiazepines, with use in females being twice that in males, and 18 per cent of benzodiazepine users taking at least one other psychotropic drug. Furthermore, 52 per cent of benzodiazepine users were prescribed a long-acting benzodiazepine.[41] It has also been reported that depression in older community-dwelling people is more likely to be detected if accompanied by anxiety symptoms, and such individuals are at risk of inappropriate treatment with benzodiazepines.[42]

Use of opiate analgesia is common in older people and is liable to give rise to MUDs. Therefore, use of these medications should be carefully monitored, with due consideration of dose and careful tapering.[43]

Aetiology, risk factors, and associations

The general principles for aetiology, risk factors, and associations for substance misuse outlined above (see Table 8.5.2.5) also apply to MUDs. Further MUD-specific factors are outlined below in Table 8.5.2.6.

Table 8.5.2.5 Risk factors for substance abuse in the elderly (Reproduced from R.M. Atkinson (2002), Substance abuse in the elderly, In *Psychiatry in the elderly* (3rd edn.) (eds. R. Jacoby and C. Oppenheimer), copyright 2002, with permission from Oxford University Press).

Predisposing factors
Family history (alcohol)
Previous substance abuse
Previous pattern of substance consumption (individual and cohort effects)
Personality traits (sedative–hypnotics, anxiolytics)
Factors that may increase substance exposure and consumption level
Gender (men-alcohol, illicit drugs; women-sedative–hypnotics, anxiolytics)
Chronic illness associated with pain (opioid analgesics), insomnia (hypnotic drugs), or anxiety (anxiolytic)
Long-term prescribing (sedative–hypnotics, anxiolytics)
Caregiver overuse of 'as needed', medication (institutionalized elderly)
Life stress, loss, social isolation
Negative affects (depression, grief, demoralization, anger) (alcohol)
Family collusion and drinking partners (alcohol)
Discretionary time, money (alcohol)
Factors that may increase the effects and abuse potential of substances
Age-associated drug sensitivity (pharmacokinetic, pharmacodynamic factors)
Chronic medical illnesses
Other medications (alcohol–drug, drug–drug interactions)

Clinical features and comorbidity

Clinical features and comorbidities associated with MUDs in older people will vary widely depending on the drug being used and patient characteristics such as age, gender, and presence of other physical and neuropsychiatric problems. An outline of clinical features and comorbidities are listed in Table 8.5.2.7.

(a) Clinical assessment

As with AUDs, a standard clinical assessment involving a history, mental state, and physical examinations and collateral history will form the basis of an MUD assessment. A list of all prescribed and over the counter medications being used, along with their indications for use, should be recorded. Ideally, the patient should be asked to bring with them all medications in their containers, as this will also give an indication as to levels of adherence or compliance. Any reported adverse effects should be recorded, along with

Table 8.5.2.6 Aetiology, risk factors, and associations of MUDs in older people

Biological/medical factors
Genetic predisposition
Chronic medical conditions (e.g. pain)
Age-related pharmacokinetic changes
Interactions: other medications and alcohol
Type of medication (e.g. benodiazepines, analgesics)
Psychosocial factors
Depression
Anxiety disorders
Personality disorder
Older age
Female gender
Lower educational level
Separated or divorced status

Table 8.5.2.7 Clinical features and comorbidity associated with MUDs in older people

Neuropsychiatric (all psychotropic drugs; benzodiazepines may be particularly problematic)
Delirium
Daytime drowsiness
Sleep disturbance
Depression
Anxiety
Physical
Falls
Fractures
Drug–drug and drug–alcohol interactions
Problems related to drug metabolism (e.g. renal and hepatic impairment)

symptoms and signs indicating underuse, overuse, or intermittent use of medication.

(b) Investigations in MUDs

Blood levels of the patient on some prescribed medications may be checked in order to assess levels of compliance and to establish if the blood level is within the therapeutic window for the drug in question (e.g. lithium, carbamazepine). Other biophysical measures may also be indicated that provide proxy measures of medication compliance, such as random or fasting glucose levels and levels of glycosylated haemoglobin, to assess for level of diabetes control and compliance with hypoglycaemic agents or insulin.

(c) Screening

There are no routinely used screening measures for MUDs in older people. However, use of a measure such as Beers' criteria[44,45] may be a useful addition to the overall assessment of an older person if an MUD is suspected.

Management and prevention

(a) Primary and secondary prevention

Along with patients themselves, health care workers, family members, and carers all have important roles in the primary and secondary prevention of MUDs in older people. Prescriptions should be reviewed regularly with a view to simplification and rationalization if possible, and the practice of giving 'repeat prescriptions' without clinical assessment should be discouraged.

Community pharmacists have an important role in providing education and advice on the use of both prescription and over the counter medications.

As older people may have physical and cognitive disabilities that interfere with appropriate use of medication,[46] devices such as dosette boxes and combination packs may be helpful.[47,48] Secondary prevention of MUDs in older people should focus on those with a past history of MUD.

(b) Tertiary prevention

Tertiary prevention of MUDs in older people will depend on the medication in question and the clinical and socio-demographic profile of the patient. Admission to a medical or psychiatric ward may be required to facilitate reduction or stopping of certain medications, e.g. benzodiazepine detoxification, as outpatient detoxification in older people may be hazardous.

Prognosis in MUDs

Similar prognostic indicators that apply to AUDs are likely to be relevant to MUDs, and centre on the individual's clinical and socio-demographic characteristics, levels of support, and available services. The duration of abuse and the medication or medications being abused is also of relevance.

Illicit drug use and nicotine use in older people

Illicit drug use in older people is far less of a problem in comparison to AUDs and MUDs. Lifetime prevalence rates for illicit drug dependence have been estimated as 17 per cent for 18–29 year olds, 4 per cent for 30–59 year olds, and less than 1 per cent for those over the age of 60.[49] Epidemiological Catchment Area data suggest a lifetime prevalence rate for illegal drug use of only 1.6 per cent for older people.[50]

Several other sources of data suggest similarly low rates of illegal drug use among older people. However, the ageing of the 'Baby-boomer' generation is likely to result in a cohort of older people who are healthier and have higher life expectancies than previous generations of older people, but who also carry with them higher rates of illegal drug use.[51]

Principles similar to those seen with AUDs and MUDs apply, in that lower levels of drug intake are required to cause harm and presentation may be atypical and thus go undetected. There is a dearth of evidence in the literature on detoxification and opiate replacement therapies in older populations.

Nicotine use (primarily through cigarette smoking) in older people arguably causes more significant morbidity and mortality than AUDs and MUDs, but the problem tends not to be addressed by psychiatrists, because of a lack of significant neuropsychiatric effects of nicotine use, and a more obvious impact on many aspects of physical health. As with AUDs and MUDs, smoking in older people is treatable, and any comprehensive approach to improving the health of older people, at the individual clinical level or public health level, should involve education about the adverse effects of smoking and efforts at active treatment through the use of nicotine replacement therapies[52] or antidepressants such as nortriptyline or buproprion.[53] However, an important and circular relationship has been described between depression, smoking, and medical illness that complicates smoking cessation in those who have a history of depression.[54]

Conclusions

In this chapter we have highlighted the importance of AUDs and MUDs in older people, in terms of their prevalence and their important but often underrecognized contribution to morbidity and mortality. AUDs are underdetected, misdiagnosed, and often completely missed in older populations. However, despite ageist and therapeutically pessimistic assumptions, AUDs in older people are as amenable to treatment as in younger people, and treating an AUD in an individual of any age can lead to significant benefits in their quality of life.

Likewise, the wide variety of MUDs in older people may be associated with addiction to medication and the undertreatment and inappropriate treatment of medical and psychiatric conditions. Considering that older people are the highest consumers of

prescription medications, screening and treatment programmes for MUDs should also lead to considerable improvements in quality of life, along with financial and other savings.

Misuse of illicit drugs by older people is not generally a major problem at present, but it is virtually certain that consumption of illegal substances by people over 65 will increase in the future.

Greater awareness amongst physicians and other health care providers of the possibility of AUDs and MUDs in their older patients should lead to the development of more comprehensive and age-appropriate prevention and treatment strategies. At the levels of everyday clinical practice and public health policy, greater emphasis should be placed on AUDs and MUDs in older people and further evaluation of dedicated 'same-age' treatment services and settings should be performed.

Further information

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Website of Royal College of Psychiatrists: www.rcpsych.ac.uk

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8.5.3 Schizophrenia and paranoid disorders in late life

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Introduction

Estimates of the point-prevalence of paranoia and other psychotic symptoms among persons age ≥ 65 years have ranged from approximately 4 per cent to 6 per cent,[1–3] and may be as high as 10 per cent among those age ≥ 85 years.[4] Although the majority of these symptoms occur as secondary psychoses in the context of Alzheimer's disease or related dementias,[5] the population of people with schizophrenia is ageing along with the general 'greying' of the industrialized world, and mental health care for older adults with schizophrenia is expected to be an increasingly important public health concern.[6]

Clinical features

Schizophrenia is typified by the presence of two or more of the following categories of core symptoms: delusions, hallucinations, disorganized or catatonic behaviour, disorganized speech (or formal thought disorder), and negative symptoms (such as affective flattening, avolition, or social withdrawal).[7] Older patients tend to have less severe positive symptoms (hallucinations, delusions, disorganized behaviour) than their younger counterparts, but there are few age-related differences in presence or severity of negative symptoms.[8,9]

Most patients with schizophrenia and related primary psychotic disorders also have mild to moderate neurocognitive deficits.[10] There is considerable interpatient heterogeneity in terms of the severity of neuropsychological deficits, but the level of these deficits is a consistent and strong determinant of impairments in everyday functioning[11] and competence or decisional capacity.[12]

In terms of late-life schizophrenia, one common division is between those with earlier onset in adolescence or early adulthood (prior to age 40 or 45 years) versus later-onset (onset ≥ age 40 or 45 years). The latter group may comprise as many as 24 per cent of people with late-life schizophrenia.[13] Relative to similarly aged patients who had earlier onset, those with later-onset schizophrenia tend to have a higher prevalence of paranoid subtype and persecutory delusions, but better premorbid social-occupational functioning, fewer current disorganized symptoms, less severe (although not an absence of) negative symptoms, and less severe neuropsychological impairment. They are also more likely to be women, and tend to respond to lower doses of antipsychotic medication.[3,14,15] The two groups are similar in terms of severity of thought disorder,[16] although patients with very late onset schizophrenia-like psychosis (age of onset ≥ 60 years) tend to have less severe formal thought disorder.[17]

Classification systems

The term 'schizophrenia' was coined by Eugen Bleuler in the early 20th century, but he wrote of 'the schizophrenias' (plural)[18] as an

explicit acknowledgement of the substantial heterogeneity that characterizes this condition. Efforts to group 'the schizophrenias' into meaningful subtypes have been a key part of efforts to define the syndrome itself.[19] Most of the terms describing different subtypes of schizophrenia in the current *Diagnostic and Statistical Manual* (DSM-IV-TR)[7] and in the International Classification of Diseases (ICD-10)[20] [such as paranoid, catatonic, hebephrenic (disorganized), and undifferentiated (simple) subtypes] overlap with the subtypes identified by Kraepelin and or E. Bleuler a century ago. Other subtyping efforts have focused on a variety of dimensions such as positive and negative symptoms, cognitive functioning and/or course, but as true of the clinical subtypes in the DSM-IV-TR and ICD-10, there is invariably substantial intrasubtype heterogeneity.[21]

In regard to late-life schizophrenia, one of the key nosological controversies over the past century has been whether or not the late onset form is actually schizophrenia. Kraepelin's conception of *dementia praecox* in 1896 was that the disorder was defined by onset in adolescence or early adulthood. By 1913, Kraepelin came to acknowledge that early onset was not a universal feature, but the emphasis on early onset remained a potent belief in the field throughout most of the 20th century.[22] On the other hand, interest in late-onset schizophrenia has a long history, including seminal work by Manfred Bleuler, begun in the early 1940s with patients whose symptoms emerged at or after age 40 years.[23]

The term 'late-onset schizophrenia' has occasionally been used interchangeably with the term *late paraphrenia*, although the latter was originally conceptualized as a more circumscribed psychosis with onset at age 60 or 65.[24,25] Unfortunately, the terms 'late-onset schizophrenia,' and *paraphrenia* (with or without the epithet 'late'), and a variety of age cut-offs have been used interchangeably and inconsistently over the years, resulting in considerable confusion in the literature.[22,25] In a 1998 international consensus meeting on this topic, the group consensus suggestion was that the term 'late onset schizophrenia' be reserved for those with onset between ages 40 and 59 years, whereas the term 'very late onset schizophrenia-like psychosis' be used with those whose symptoms first manifest at age 60 or later.[3]

None of the above schizophrenia onset-related categories is represented in the contemporary formal diagnostic systems. The 1980 version of the American Psychiatric Association's *Diagnostic and Statistical Manual* (DSM-III)[26] arbitrarily excluded the diagnosis of schizophrenia if symptoms did not emerge prior to age 45. This exclusion was dropped from the subsequent revision (DSM-III-R),[27] although the DSM-III-R required the specification of 'late onset' if the prodromal phase of illness developed after 45. The latter is the only instance of 'late-onset schizophrenia' appearing as a named condition in one of the major nosological systems. Based on mounting empirical evidence that 'real' schizophrenia could manifest after age 45,[13] the age of onset restrictions as well as the 'late onset' specifier were dropped in the DSM-IV [28] and DSM-IV-TR.[7] Similarly, there is no age-of-onset related restriction or specification under the ICD-10.[20]

Diagnosis and differential diagnosis

The diagnostic criteria for schizophrenia in the DSM-IV-TR and ICD-10 mention neither current age nor age of onset.[7,20] A key differential diagnosis with older adults is to rule out presence of a secondary psychosis.[29] For instance, among elderly patients, psychotic symptoms most commonly present in the context of dementia, such as Alzheimer's disease, Parkinson's disease, or dementia with Lewy Bodies.[5] The pattern in any one patient may of course vary from normative trends, but in general among those with dementia-related psychotic symptoms, there is a greater propensity for visual over auditory hallucinations, and bizarre content is less common in the delusions than in those of patients with primary psychotic disorders such as schizophrenia.[30]

Delirium may also present as acute psychosis;[31] as with dementia, visual hallucinations and delusions tend to be more common than auditory hallucinations, but the psychotic symptoms associated with delirium can be of any form.[32] Given the high rates of polypharmacy among the elderly as well as age-related changes in pharmacokinetics, it is also important to consider potential acute mental effects of the medications in isolation and in combination.[33] Other differential diagnoses to consider among elderly patients are non-psychotic hallucinations related to bereavement or sensory deprivation.[34,35]

Among the primary psychotic conditions, the standard differential diagnoses and considerations apply in terms of differentiating among schizophrenia, schizoaffective disorder, delusional disorder, brief psychotic disorder, substance-induced psychotic disorder, bipolar disorder with psychotic features, and major depressive disorder with psychotic features.[7]

Epidemiology

As was noted above, prevalence estimates of paranoia and other psychotic symptoms among persons age ≥ 65 years have ranged from approximately 4 per cent to 6 per cent,[1–3] but these symptoms are most commonly in the context of a dementia or other medical condition. Estimating the lifetime prevalence of schizophrenia is a methodologically complex endeavour needing additional research attention; recent estimates have ranged from approximately, 0.4 per cent to 1.0 per cent although estimates as high as 1.6 per cent have also been reported.[36,37] The lifetime prevalence of schizophrenia is similar among men and women, and the majority of patients of either gender experience onset in adolescence or early adulthood.[37] However, a consistent finding noted a century ago by E. Bleuler,[18] is that women tend to show later onset than men.[38]

Estimates of the lifetime prevalence of schizophrenia for persons over age 65 have also varied, although the 95 per cent confidence interval estimate from one recent comprehensive study was 0.58 to 1.45 per cent.[37] There have been some epidemiological studies suggesting that the prevalence (current and lifetime) of schizophrenia among elderly persons is lower than that for the younger population. People with schizophrenia have higher mortality due to suicide and physical disorders,[39,40] so there are probably proportionally fewer people with schizophrenia who survive to older age. However, the prevalence of schizophrenia in elderly patients may also have been underestimated in some of the earlier major epidemiological studies.[41] For instance, the Epidemiologic Catchment Area study used the DSM-III criteria, but as was noted above, the DSM-III criteria for schizophrenia arbitrarily required onset of prodromal symptoms prior to age 45, so any cases of later-onset schizophrenia would have been excluded.[41]

Aetiology

The cause(s) of schizophrenia remains unknown. Both Kraepelin and E. Bleuler correctly suspected that there is a heritable vulnerability to schizophrenia, confirmed by the substantially higher concordance rates among monozygotic twins (estimated at 40 to 50 per cent) relative to dizygotic twins (estimated at 5 per cent to 25 per cent).[42,43] The elevated risk of schizophrenia among first-degree relatives is present among those with schizophrenia onset in middle-age as well as those wither earlier onset relatives.[3] Although some candidate genes have been identified,[44] these efforts remain in an early stage of development. Also, given that even the monozygotic twin concordance rate is substantially below 100 per cent, non-genetic factors clearly have a role in the ultimate expression of the schizophrenia phenotype.

At present, the prevailing model of schizophrenia is that of neurodevelopmentally based aberrations in connectivity of key brain regions and systems.[45,46] Evidence for the neurodevelopment component includes an elevated risk of schizophrenia associated with certain pre- or peri-natal insults or stresses, an increased prevalence of minor facial anomalies among patients with schizophrenia, and an increased prevalence of subtle childhood abnormalities in motor, cognitive, and/or psychosocial development among those who later develop schizophrenia.[45] At the level of neuropathology, Kraepelin expressed some suspicion of involvement of the prefrontal and temporal lobes;[47] these remain areas of interest in schizophrenia research although the current focus is on functional (rather than gross structural) impairments related to the connections among such brain regions or systems.[46]

Course and prognosis

Schizophrenia is generally a chronic condition, but not necessarily a constantly sustained one in that many patients experience one or more periods of several years of sustained recovery over their lifespan, but periods of relapse are also common.[48] As with other dimensions of this disorder, the long-term course of schizophrenia is also characterized by heterogeneity among patients, as well as methodological challenges in interpreting varied findings in the empirical literature.[49] Some of the factors that have been cited as associated with worse prognosis include poor premorbid functioning, very early and gradual/insidious onset of symptoms, male gender, and a relative prominence of negative symptoms.[49,50] In a recent review of ten long-term longitudinal outcome studies, Jobe and Harrow[48] found that the estimates of 'good outcome' ranged from 21 to 57 per cent. Since most of the long-term research on the course of schizophrenia has been of younger patients as they age, there remains a clear need for longitudinal research to document changes among patients as they age from their 60s, 70s, and 80s. Nonetheless, empirical data do not seem to support Kraepelin's initial suggestion that *dementia praecox* is characterized by a course of progressive decline.[49] In fact, there may be some modest age-related improvements in positive symptoms and perhaps other aspects of psychopathology.[8] Although there is a small subset of 'poor outcome'/ chronically institutionalized patients who seem to be at added risk for cognitive and functional decline in older age,[51] for most patients there is generally no increased (beyond age normal) decline in cognitive functioning among those with early or middle-age onset.[52,53]

Treatment

Contemporary treatment guidelines for schizophrenia in older adults parallel those for younger adults in that a combination of pharmacological and psychosocial interventions is recommended.[54] Although treatment with antipsychotic medications is a mainstay of effective treatment and management of late-life schizophrenia, selection of the appropriate type and dose of antipsychotic medication in the elderly may be complicated by age-related factors. One of the primary concerns with conventional neuroleptic medications is that they can cause iatrogenic motor abnormalities; older adults are at even greater risk than younger patients to develop tardive dyskinesia and extrapyramidal symptoms, or EPS, (especially parkinsonism) from conventional neuroleptics.[55] The newer ('atypical' or 'second generation') antipsychotic medications have lower (although not absent) associated risk of tardive dyskinesia and EPS, though risks for these motor side effects also vary from one atypical antipsychotic to another.

The current APA treatment guidelines for schizophrenia indicate '*Second-generation antipsychotics are generally recommended over first-generation agents because of their significantly lower risk of inducing extrapyramidal symptoms and tardive dyskinesia in older persons . . . However, the second-generation agents have other clinically significant and common side effects* (pp. 33–34).' In addition to concerns about potential sedation, orthostatic hypotension, and other potential physical or medical side-effects from the newer medications, there has been recent concern about the potentially serious metabolic and cardiovascular side effects.[56] Overall, atypical agents (with the exception of clozapine) have not shown superior efficacy for schizophrenia in direct comparisons with typical antipsychotics.[57] This fact, combined with the lower cost of typical agents, has prompted some experts to question the use of atypical antipsychotics as first-line pharmacotherapy for schizophrenia. In the absence of more definitive evidence, clinicians should discuss these various advantages and disadvantages of specific drugs with patients when choosing drug therapy.

For most antipsychotics (typical or atypical), older adults with schizophrenia generally respond to 50–75 per cent of the doses needed in younger patients and often cannot tolerate the full young adult dose.[58] Side effects that are especially problematic in older adults include anticholinergic effects (e.g. constipation, confusion, urinary retention), orthostatic blood pressure changes (which may lead to falls), and reported increases in stroke and death among persons with dementia receiving antipsychotics (as dementia, of course, becomes more prevalent with increasing age).

Antipsychotic medications are helpful in managing the psychopathologic symptoms of schizophrenia, especially the so-called 'positive symptoms' such as delusions and hallucinations, but they are not a cure. Medications also tend to have little benefit for the 'negative' (e.g. apathy, anhedonia) and cognitive symptoms of the illness. Furthermore, many patients continue to have residual functional disability despite resolution of positive symptoms of psychosis. Thus, the importance of adjunctive treatment with evidenced-based psychosocial interventions in schizophrenia is being increasingly recognized.[59] For instance, investigators at our Research Center have developed or adapted, and validated a number of effective adjunctive psychosocial interventions for older patients with schizophrenia or related psychoses; these include Cognitive Behavioral Social Skills Training,[60]

Functional Adaptation Skills Training,[61] diabetes management/lifestyle modification,[62] and vocational rehabilitation/supported employment.[63] These efforts address a number of dimensions of schizophrenia that are unaffected by pharmacologic treatment alone.

Management

As noted above, schizophrenia is a chronic condition. In addition, to specific therapies previously described, managing this complex and devastating illness requires careful attention to clinician-patient rapport. This is especially true in persons with severe paranoia and in those who lack insight into their illness. Providing care for someone who does not trust anyone or who sees no reason for treatment can be challenging, but these obstacles can often be overcome by involving family, listening empathically, avoiding premature confrontation about delusions, respecting the increased interpersonal distance many patients require, and identifying the patient's goals and priorities. Management of schizophrenia may often be optimized by multi- and interdisciplinary care to address the many ways in which the illness affects patients' lives. Because there tends to be a high degree of medical comorbidity in persons with schizophrenia (especially older adults), integration of primary care and psychiatric care is often needed. Due to normal age-related physical changes, polypharmacy, and the higher risk of medication side-effects, the long-term management of schizophrenia in older adults demands frequent monitoring of symptoms, overall health, and side-effects. Cross-discipline collaborative care and continuity with the same clinicians can help ensure older persons with schizophrenia achieve the best possible outcomes.

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8.5.4 Mood disorders in the elderly

Robert Baldwin

Introduction

This chapter considers some of the commonly asked questions about mood disorders in later life. Is depression in later life a distinct clinical syndrome? How common is it? Is there an organic link, for example to cerebral changes, and if so, is there an increased risk of later dementia? Is it more difficult to diagnose and treat late-life depression, and once treated, is the outcome good, bad, or indifferent? The emphasis will be on depression but bipolar disorder and mania will also be considered.

Classification

The main mood disorders which older people suffer are classified as: depressive episode, dysthymia, bipolar disorder, and organic mood disorder. Depressive illness and major depression are terms often used synonymously with depressive episode. Current classificatory systems, notably The World Health Organization ICD10 and the DSM Version IV of the American Psychiatric Association, are described in Chapter 4.5.3.

Depressive episode

Clinical features

Other than more frequent somatic and hypochondriacal complaints, patients with depression in later life are little different, symptomatically, to younger adults.[1] An exception may be the recently described 'vascular depression' (depression linked to small vessel disease of the brain), in which depressive ideation is less but cognitive impairment and apathy greater. In most cases then the pathoplastic effects of ageing and ill-health are what mainly influence the presentation of depression in later life (Table 8.5.4.1).

An overlap of symptoms due to *associated physical ill-health* may lead to diagnostic difficulty, and determining whether a symptom has arisen predominantly because of affective disorder or a medical condition can be difficult for those without the necessary experience.

Older depressed patients may *minimize feelings of sadness* and instead become *hypochondriacal* (morbidly preoccupied with a fear of illness).[1] Late-onset *neurotic symptoms* (*severe anxiety*, *phobias*, *obsessional compulsive phenomena or hysteria*) are usually secondary to depressive illness. Any act of *deliberate self-harm* suggests depression, as elderly people rarely take 'manipulative' overdoses. An overdose in an older person should never be dismissed because its effects, in purely medical terms, were trivial. *All* require psychiatric assessment. Severe depression may mimic dementia. Table 8.5.4.2 highlights the main differences between progressive dementia and the pseudodementia of depression. Pseudodementia is a term which is perhaps waning in use as it has become clear that depressive disorder is commonly associated with cognitive impairment, which may not be reversible, even with adequate treatment of depression. Pseudodementia is often applied to an older depressed patient who, on presentation, appears very confused, with frequent 'don't know' responses. However, the onset of confusion is acute, easily dated, the patients convey their despair non-verbally, and, unlike the person with degenerative dementia, complain vociferously about their memory. Cortical signs (aphasia, apraxia, etc.) suggest a primary dementia with a super-added depression rather than depressive pseudodementia. Wandering off and getting lost suggests dementia but occasional cases are seen of fugue states caused by severe depression mimicking disorganized behaviour in dementia. The key is a good history.

An unusual *behavioural disturbance* may occasionally be a leading symptom of depression. Examples include the onset of incontinence in an older person who feels trapped in a situation of resented dependency in a residential or nursing home, late-onset alcohol abuse or, rarely, shoplifting.

Table 8.5.4.1 Factors influencing the presentation of depression in older people

Overlap of symptoms of physical disorder with those of the somatic symptoms of depression
Tendency of older people to minimize a complaint of sadness and instead become hypochondriacal
Late-onset neurotic symptoms (severe anxiety, obsessional compulsive symptoms, hysteria) which mask depression
Deliberate self-harm which seems medically trivial
Pseudodementia
Behavioural disturbance such as alcohol abuse or shoplifting

Table 8.5.4.2 Characteristics distinguishing depression ('pseudodementia') from dementia

Dementia	Depression
Insidious	Rapid onset
Symptoms usually of long duration	Symptoms usually of short duration
Mood and behaviour fluctuate	Mood is consistently depressed
'Near miss' answers typical	'Don't know' answers typical
Patient conceals forgetfulness	Patient highlights forgetfulness
Cognitive impairment relatively stable	Cognitive impairment fluctuates greatly
Higher cortical dysfunction evident	Higher cortical dysfunction absent

(a) Vascular depression

In vascular depression, vascular disease is judged to predispose, precipitate, or perpetuate depressive symptoms. Evidence (summarized by Baldwin[2]) includes the following. There is a high rate of structural brain abnormalities in both white matter and basal ganglia grey matter on imaging and on post-mortem examination of older patients with depressive disorder, notably with a late age of onset. Psychomotor change, apathy, and executive dysfunction (leading to slowed responses, failure of initiation, impersistence in tasks, and inefficient memory) occur characteristically in such patients. Strategic lesion location, sufficient to disrupt subcortical-frontal circuitry, is associated with poorer depression outcomes, and progression of such lesions is associated with later incident cases of depression in those not already depressed. The concept of vascular depression is discussed critically under aetiololgy.

Diagnosis and differential diagnosis

(a) Assessment

The psychiatric history should include a collateral history as well as drug evaluation (prescribed, 'borrowed', and over-the-counter) and alcohol intake. A cognitive screening test should always be undertaken. A physical evaluation should focus on possible disorders causing an organic mood disorder (Table 8.5.4.3), including medication. Non-selective β-blockers, calcium antagonists, benzodiazepines, and systemic corticosteroids were the main culprits in one study.[3]

Screening questionnaires can be used to help diagnose depression, especially in settings such as medical wards where the prevalence is high, but their results must be informed by clinical judgement. The Geriatric Depression Scale (GDS) (Geriatric Depression Scale website http://stanford.edu/~yesavage/GDS.html) is widely used. It focuses on the cognitive aspects of depressive illness rather than physical depressive symptomatology, and has a simple 'yes/no' format (Table 8.5.4.4). It loses specificity in severe dementia but performs reasonably well in mild to moderate dementia. For rapid screening four questions (1, 3, 8 and 9) can be used.

(b) Investigations

Table 8.5.4.5 summarizes investigations appropriate for a first episode of depression and a recurrence. A guiding principle is that elderly people are in a more precarious state of homeostasis with their environment because they have less physiological reserve.

Table 8.5.4.3 Common medical illnesses and drugs that may cause organic mood syndromes

Medical conditions	Central-acting drugs
Endocrine/metabolic	*Anti-hypertensive drugs*
Hypo/hyperthyroidism	β-blockers (especially non-selective)
Cushing's disease	Methyldopa
Hypercalcaemia	Reserpine
Sub-nutrition	Clonidine
Pernicious anaemia	Nifedipine, calcium channel agents
Organic brain disease	Digoxin
Cerebrovascular disease/stroke	*Steroids*
CNS tumours	*Analgesic drugs*
Parkinson's disease	Opioids
Alzheimer's disease and vascular dementia	Indomethacin
Multiple sclerosis	*Anti-parkinson*
Systemic lupus erythematosus	L-Dopa
Occult carcinoma	Amantadine
Pancreas	Tetrabenazine
Lung	*Psychiatric drugs*
Chronic infections	Neuroleptics
Neurosyphilis	Benzodiazepines
Brucellosis	*Miscellaneous*
Neurocysticercosis	Sulphonamides
Myalgic encephalomyelitis	Alcohol
AIDS	Interferon

Severe depression in a 75-year-old may lead to quite serious metabolic derangement which would be unlikely in a fit 35-year-old.

An electroencephalogram (EEG) can help in differentiating depression from an organic brain syndrome such as delirium or an early dementia. A brain scan is only performed if clinically indicated, for example a rapid-onset depression with neurological symptoms or signs. The Dexamethasone Suppression Test (DST) is less specific for depressive illness than was first thought. It cannot reliably differentiate dementia from depression.

(c) Differential diagnosis

Organic mood disorder is diagnosed when a direct aetiological link can be established between the onset of the mood disorder and an underlying systemic or cerebral disorder (including dementia), or an ingested substance such as medication or alcohol.

Bipolar disorder is covered later. **Psychotic illness** (schizophrenia or delusional disorder) may present with marked depressed affect but other symptoms are present. A common depressive delusion in old age is hypochondriasis and sometimes it is difficult to decide whether the patient has a psychotic or an affective disorder. Interpretation depends on which symptoms predominate; if they occur together, it may be appropriate to use the term schizoaffective disorder.

Table 8.5.4.4 Geriatric Depression Scale

Instructions: Choose the best answer for how you have felt over the past *week*.

1. **Are you basically satisfied with your life?** No
2. **Have you dropped many of your activities and interests?** Yes
3. **Do you feel your life is empty?** Yes
4. **Do you often get bored?** Yes
5. Are you hopeful about the future? No
6. Are you bothered by thoughts you can't get out of your head? Yes
7. **Are you in good spirits most of the time?** No
8. **Are you afraid something bad is going to happen to you?** Yes
9. **Do you feel happy most of the time?** No
10. **Do you often feel helpless?** Yes
11. Do you often get restless and fidgety? Yes
12. **Do you prefer to stay at home, rather than going out and doing new things? Yes**
13. Do you frequently worry about the future? Yes
14. **Do you feel you have more problems with your memory than most?** Yes
15. **Do you think it is wonderful to be alive now?** No
16. Do you often feel downhearted and blue (sad)? Yes
17. **Do you feel pretty worthless the way you are?** Yes
18. Do you worry a lot about the past? Yes
19. Do you find life very exciting? No
20. Is it hard for you to start on new projects (plans)? Yes
21. **Do you feel full of energy?** No
22. **Do you feel that your situation is hopeless?** Yes
23. **Do you think most people are better off (in their lives) than you are?** Yes
24. Do you frequently get upset over little things? Yes
25. Do you frequently feel like crying? Yes
26. Do you have trouble concentrating? Yes
27. Do you enjoy getting up in the morning? No
28. Do you prefer to avoid social gatherings (get-togethers)? Yes
29. Is it easy for you to make decisions? No
30. Is your mind as clear as it used to be? No

Notes: (1) Answers refer to responses which score '1'; (2) bracketed phrases refer to alternative ways of expressing the questions; (3) questions in bold are for the 15-item version. Threshold for possible depression: >/=11 (GDS30); >/=5 (GDS15); >=2 (GDS4).

Dysthymia chiefly occurs in younger adults but may occur in later life in association with chronic ill-health. Where there is a clear onset of depressive symptoms within 1 month of a stressful life event without the criteria for a depressive episode being met, then an *adjustment disorder* may be diagnosed.

Epidemiology

In the United Kingdom, pervasive depression (a term denoting a depressive syndrome that a psychiatrist would regard as warranting intervention) is found between 8.6 and 14.1 per cent of elderly people living at home. The prevalence of a depressive episode is between 1 and 4 per cent of elderly people living at home.[4] The finding of a high rate of depressive symptoms but a much lower rate of depressive episodes is an epidemiological dilemma which is discussed in Chapter 4.5.4. It is likely that current classification

Table 8.5.4.5 Investigations for depression in later life

Investigation	First episode	Recurrence
Full blood count	Yes	Yes
Urea and electrolytes	Yes	Yes
Calcium	Yes	Yes
Thyroid function	Yes	If clinically indicated, or more than 12 months elapsed
B_{12}	Yes	If clinically indicated, or more than 12 months elapsed
Folate	Yes	If clinically indicated (for example recent poor diet)
Liver function	Yes	If indicated (for example suspected or known alcohol misuse)
Syphilitic serology	If clinically indicated (for example relevant neurological symptoms)	Only if clinically indicated
CT (brain)	If clinically indicated	If clinically indicated
EEG	If clinically indicated	If clinically indicated

systems overlook many of the late-life depressions found in community studies.

Depression in later life, whether major or 'minor', is associated with worsened medical morbidity, disability, and increased health utilization.[1] Co-morbidity from physical disorder or cognitive impairment is the main determinant of prevalence. Handicap, the disadvantage imposed by a physical impairment and attendant disability, is a further strong predictor of depression.[5] This matters because handicap is amenable to social intervention.

In residential care, nursing homes and medical wards the rates are between 20 and 40 per cent.

Aetiology

The risk factors for depression in later life are discussed in Chapter 4.5.5. A depressive episode usually arises from a combination of vulnerability factors along with a triggering (precipitating) adverse life event. Avoidant and dependent personality types are associated with late-life depression, and a lifelong lack of a capacity for intimacy is another risk factor.[6] Precipitating life events occur at a similar frequency to other age groups, although health-related events are more common among older people.[6]

The concept of vascular depression suggests new aetiological insights.[2] However, criticisms against vascular depression as a distinct subtype include the difficulty in establishing a temporal link between depression onset and vascular disease and that, if causal, vascular disease is likely to have been present well before old age. Furthermore, in studies of vascular depression the direction of causality is unclear since patients with vascular disease have a high rate of depression, and depression appears to worsen vascular disease, perhaps by direct effects on blood vessel endothelial function and indirectly through poor self-monitoring of health and poor adherence to medical drugs.

If confirmed, the vascular depression hypothesis could lead to antidepressant strategies aimed at improving the underlying vascular impairment as well as mood. In the meantime, a patient presenting late-onset depression should be thoroughly investigated for vascular disease as a potential aetiological contributory factor which should be optimally treated along with the depression.

Course and prognosis

Across all age groups depressive disorder is prone to persistence. Beekman *et al.*[7] followed 277 community-dwelling subjects, aged over 55 (most over 75), from a Dutch epidemiological survey. Using multiple assessments of mood over a 6-year period, they found almost half the sample was depressed for more than 60 per cent of the time. Twenty three per cent had true remissions, 12 per cent remissions with recurrence, a third a chronic-intermittent course; another third had chronic depression. Outcomes reported from the community and medical ward patients are worse than those of depressed patients under psychiatric care,[8] possibly linked to undertreatment in the non-specialist settings.

(a) Comparative outcome

Mitchell and Subramaniam[9] reviewed the literature between 1966 and July 2004, finding 24 publications which could be used to assess outcomes between different age groups. Overall the authors concluded that episodes of depression remitted in later life as well as in other times but with a greater risk of relapse. Two factors seemed to explain this: age of onset (recurrent depression from earlier life conferring a poorer prognosis) and medical comorbidity (with a worse prognosis linked to a later onset). Although these mechanisms differ, the high risk of relapse in late-life depression highlights the need for effective continuation and maintenance phases of treatment, regardless of age of onset.

Mortality

A number of studies show that depression is an independent risk factor for increased mortality,[1] not accounted for by suicide. Following a cohort of 652 depressed and non-depressed subjects over 3.5 years, Geerlings[10] found that duration, chronicity, and increasing symptoms from baseline were all linked to a higher risk of death, leading them to suggest that adequate treatment may reduce this effect. Physical illness, occult disease (e.g. a carcinoma), poor self-monitoring of health, inactivity, poor adherence to treatments, and effects on the hypothalamic-pituitary-adrenal axis or other endocrine systems are possible factors.

Factors predictive of outcome

Clinical features that have been shown to be associated with a poorer outcome, include a slower initial recovery, more severe initial depression, duration of illness for more than 2 years, three or more previous episodes, a previous history of dysthymia, psychotic symptoms, and cerebrovascular disease (including vascular depression). Other factors that may affect outcome adversely are chronic stress associated with a poor environment, crime, and poverty as well as a new physical illness, becoming a victim of crime, and poor perceived social support.

The practical message is that to improve the prognosis of depression one must treat episodes early and vigorously and attend to the patient's social supports, and milieu.

(a) Does depressive disorder predispose to later dementia?

There is growing evidence from epidemiological studies that depression is a risk factor for later cognitive impairment or dementia.[11] Why this might be so is not clear but it is known that chronic depression is associated with hippocampal atrophy in older patients,[12] and that depressed patients may adopt unhealthy lifestyles, which can aggravate the risk factors for vascular disease and later dementia.

Treatment

Multi-modal management (pharmacological, psychological, and social) within a multidisciplinary framework is as important in late-life as it is at other times of life. Attending to an elderly depressed person's physical health needs, physical environment, and social needs is essential. The goal is remission of all symptoms and not merely improvement, as residual symptoms increase the chance of chronicity. Ageing and frailty result in increased dependency and less ability to adapt in a flexible way to the kinds of adversity that maintain depression. To give a simple example, good chiropody aimed at optimizing mobility can have a major positive impact alongside medical intervention, but no one aspect of treatment should be prioritized over another. Another important principle is patient-centredness. This should include giving the patients as much choice as possible regarding their treatment. Many older depressed patients, if asked, would prefer a psychological approach to medical management and there is good evidence that psychological treatments work well in older adults.[13] Resource limitations are understood, but age alone should not determine the likelihood of receiving a psychological intervention, if preferred.

Collaborative care, whereby, a depression care manager (usually a nurse, psychologist, or social worker) coordinates the care and works closely with both primary care physician and psychiatrist, is an important model for improving outcomes. In the largest study to date, 'IMPACT' from the United States, involving 1801 depressed primary care patients (major depression, 17 per cent; dysthymia, 30 per cent; or both, 53 per cent), at 12 months the Number-Needed-to-Treat (NNT) was highly significant at four in those receiving the intervention.[14]

General principles of pharmacological management include: building a therapeutic partnership with the patient; explanation of how and when antidepressants work; addressing adherence through building therapeutic concordance; tailoring antidepressants drug to the patient; arranging appropriate follow-up.

Evidence of efficacy

(a) Antidepressants

Altered pharmacokinetics, different pharmacodynamics, a greater chance of polypharmacy and hence drug interactions and reduced compensatory mechanisms are all important factors which bear upon treatment response in late-life depression.[15] An important practical consequence is to be mindful of greater inter-individual variation in drug-handling in older patients. Patients worry about antidepressants being addictive, which they are not, and that depression means 'senility', which it does not. Psychoeducation is important and can improve adherence to medication recommendations. Recommended starting and therapeutic dosages are listed in Table 8.5.4.6. These are average doses. For some drugs, notably the tricyclics, there is a very wide therapeutic range and higher levels may be required, provided they are tolerated.

A Cochrane systematic review of Randomized Controlled Trials (RCT)[16] found efficacy for antidepressants over placebo in late-life major depression. However, the analysis could only find 17 suitable studies (two of SSRIs). The NNT averaged four, but was higher for SSRIs. In a further Cochrane systematic review of studies including patients aged over 55 (29 trials), there was no difference in efficacy between tricyclics and SSRIs, but tricyclics were associated with higher withdrawal rates due to side-effects. Patients receiving tricyclic-related antidepressants (Mianserin or Trazodone) had a similar withdrawal rate to SSRIs, leading the authors to conclude that tricyclic-related antidepressants may offer a viable alternative to SSRIs in older depressed patients. The analysis also investigated 'atypical' antidepressants but it was not possible to make recommendations because of low statistical power. Atypicals included the important antidepressants reboxetine, venlafaxine, and mirtazapine.[17]

More recent RCTs have suggested less positive results.[18] Trials involving older adults and with venlafaxine, fluoxetine, citalopram, and escitalopram showed these drugs to be no more effective than placebo. Sertraline and duloxetine were superior to placebo but remission rates, as opposed to response rates, were relatively low in all these studies. Trials which are of insufficient duration and the use of antidepressants for milder depressions, for which they are ineffective, are possible factors.

(b) Depression in special patient groups

A Cochrane systematic review of antidepressants for depression in dementia found 'weak' evidence for their effectiveness,[19] but there were few admissible trials. This does not mean

Table 8.5.4.6 Suggested starting and therapeutic doses for antidepressants in the United Kingdom

Drug	Average therapeutic doses[a]	Average starting doses
Tricyclics		
Amitriptyline	75–100	25
Clomipramine	75–100	10
Dosulepin (dothiepin)	75–150	25–50
Imipramine	75–100	10–25
Nortriptyline	75–100	10–30
Lofepramine	140–210	70–140
SNRIs		
Venlafaxine	150	37.5 bd
Duloxetine	60	30–60
SSRIs		
Fluoxetine	20	20
Fluvoxamine	100–200	50–100
Paroxetine	20	20
Sertraline	50–150	50
Citalopram	20–40	20
Escitalopram	10	5
Reversible monoamine oxidase inhibitor (RIMA)		
Moclobemide	150–600	150 bd
5-HT2 receptor blocker		
Trazodone	100–300	100
NASSA		
Mirtazapine	15–30	30
Others		
Mianserin	30–90	30

[a] See text.

antidepressants are ineffective in depression-related dementia but there is a high rate of spontaneous recovery of depression complicating dementia so that 'watchful waiting' is reasonable for mild-to-moderate cases.

There is little trial data for individual antidepressants in patients with common medical disorders, but in the previously mentioned IMPACT research, both physical function and pain were improved in participants who received active case management compared to usual care.[20,21]

Depression post-stroke is common. A high rate of spontaneous recovery occurs, especially in the first 6 weeks. Studies are underway to assess whether repetitive Transcranial Magnetic Stimulation (rTMS) may be helpful in post-stroke depression. TCAs and SSRIs in standard dosages are effective in post-stroke emotionalism.

(c) Choice of antidepressant

Tricyclics often cause postural hypotension, which may lead to unpleasant dizziness or dangerous falls. Secondary amine tricyclics are generally safer in this respect than tertiary drugs. Poor left ventricular function is a risk, and so are diuretics or antihypertensive medication. Delirium is more likely in medically ill patients.

Behavioural toxicity (affecting vigilance, reaction times, etc.) has been largely ignored in the elderly. Now that so many older people drive and pursue other activities demanding high levels of vigilance, this must be addressed. The *SSRIs* are safer in this respect than tricyclics, except for lofepramine which causes less impairment than the older tricyclics. *Mirtazepine* enhances noradrenergic and serotonergic function via antagonism at the pre-synaptic α_2 receptor. Differences in pharmacodynamics and pharmacokinetics are minimal with age. The side effect profile is similar to tricyclics; weight gain and sedation can be troublesome. *Duloxetine*, like venlafaxine is a dual-acting drug. The latter has been subject to cautions, regarding heart disease, at present in the United Kingdom, although recent restrictions have been lifted. Duloxetine has some RCT evidence in older depressed adults.[22] Although a special diet with *moclobemide* is not required, patients should be aware of drug interactions with painkillers and other antidepressants. Co-prescriptions of tricyclic and SSRIs should be avoided. A wash-out period of around 4–5 half-lives of the drug and any active metabolite is advised when transferring from a tricyclic or SSRI to moclobemide (but not from moclobemide to a tricyclic or SSRI).

(d) Failure to respond to initial treatment

If a patient has made minimal or no recovery after 4 weeks of treatment at optimal dose, then the chances of recovery are slim.[23] The antidepressant may be changed to one of another class but if the patient shows at least 25 per cent improvement and is on an improving trajectory, then augmentation with lithium or a psychological intervention should be considered (see below). Electroconvulsive Treatment (ECT) is safe and effective in older patients. It is recommended when drug treatment has failed, when the patient is in danger of inanition or is acutely suicidal and is probably the treatment of choice for psychotic depression.

(e) Continuation treatment

Most relapses occur in the first 12 months,[24] so that this is a reasonable time for continuation therapy. Patients must be educated about why they should continue to take medication even when feeling better. In psychotic depression anti-psychotic medication should be continued for 6 months and gradually withdrawn if the patient is well. Following ECT, medication should be continued to avoid relapse. Limited evidence suggests either continuing antidepressants at the acute treatment dose or using lithium.

Older people have been shown to benefit substantially from maintenance therapy,[25] even after a first episode.[24] Maintenance treatment is considered later.

(f) Resistant depression

Data in the elderly are sparse but the most important consideration is a rational stepped care approach. Before moving up the steps of treatment, the following should be addressed: is the diagnosis correct (for example has a psychotic depression been overlooked)? Is poor tolerance a reason for non-recovery? Does the patient take the tablets? Have psychosocial reinforcing factors (for example, family conflict) been addressed? The steps themselves include optimizing the dose of antidepressant (relevant mainly for older tricyclic antidepressants), changing from one class of antidepressant to another, augmentation with lithium or a psychological intervention and combining antidepressants (for example, a SSRI plus mirtazepine). Finally ECT should be considered as it remains the most effective antidepressant treatment. Using such an approach 80 per cent of patients in one study responded.[26]

(g) Psychological therapies

Cognitive behavioural therapy (CBT), Interpersonal Psychotherapy (IPT), and psychodynamic psychotherapy have been shown to work in older people,[13] including in group format. CBT and IPT along with family interventions are discussed elsewhere. Problem-solving Treatment (PST) addresses the here and now, focusing on current difficulties and setting future goals.

Psychological interventions may also be important in relapse prevention. Reynolds *et al.*[27] showed in a study of older patients that monthly IPT given in the continuation phase of treatment was more effective than routine care, with combined IPT and antidepressant therapy being the most effective strategy. However, the same group were unable to replicate this in a later study with a group of patients who were somewhat older.[25]

Anxiety management can be an effective adjunctive treatment for depressed patients, especially those recovering from depression but left with residual anxiety, low confidence, or phobic avoidance, any of which can undermine functional improvement. Techniques include progressive relaxation, either alone with a commercial tape, or in groups. Exercise and activity are important both to avoid depression and counter it. Behavioural activation is a technique which can overcome the withdrawal and apathy that so often exists in late-life depression. It works by helping the patient develop a schedule of activities, agreed with the patient, with or without a written diary to support implementation.

Work to support the family and main caregivers is also important.

Prevention

(a) Primary prevention

Many prevalent diseases of later life are associated both with depression as well as lifestyle factors: diet, exercise, and obesity. Cole and Dendukuri[28] carried out a meta-analysis of risk factors for late-life depression, finding five which were robustly linked to it. These were bereavement, sleep problems, disability, prior depression, and female gender. Some of these are amenable to a public health preventative approach.

Those most vulnerable to depression will often be in touch with a home care services. This is one area where education about detection could be usefully targeted. Another example is the staff of nursing homes where depression is highly prevalent. Postgraduate training of general practitioners is another way of improving detection via the 'filter' of primary care.

(b) Secondary prevention

Maintenance treatment with a tricyclic,[24] the SSRIs citalopram[29] and paroxetine[25] or a combining medication with a psychological treatment[27] are effective prevention strategies.

Expert guidelines[30] recommend a minimum of 12 months continuation treatment for a first episode, 24 months for a second, and at least 3 years for three or more episodes. Some clinicians recommend lifelong treatment with antidepressants following even a single episode of major depression on the grounds that a substantial later period morbidity might be reduced. This must be balanced against an increased risk of side effects as patients age.

(c) Tertiary prevention

Often the emphasis is on basic explanations and on simple instructions about how to manage problems such as frequent hypochondriacal complaints or apathy. Although respite care is usually associated with dementia, there is occasionally a case for it in those with chronic treatment-resistant depression, in order to allow the relative(s) a break.

Bipolar disorder

Practice guidelines for bipolar disorder are available from the internet. Two are: *British Association of Psychopharmacology* (2003)[31]—http://www.bap.org.uk/consensus/bipolar_disorder.html and the *National Institute for Clinical Excellence (NICE) (2006)*.[32] http://www.nice.org.uk/page.aspx?o=CG38.

Mania

Clinical features

Clinical descriptions of mania in late life often portray it as atypical. However, Broadhead and Jacoby[33] found few clinical differences between 35 manic patients over the age of 60 compared to 35 younger manic patients, aged below 40. The younger manic patients were more severely ill but there was no support for the often-held view that there is a greater depressive admixture in older patients.

Diagnosis and differential diagnosis

The main differential diagnosis of mania in late life lies between a late-onset manic episode and bipolar disorder. In later presentations of bipolar disorder the time between depression and first manic episode can be us much as 15 to 40 years or more. Given this long latency it is easy to overlook bipolar disorder unless a thorough history is taken.

Epidemiology

Although only about 10 per cent of new onset cases of bipolar disorder occurs after the age of 50 they account for proportionally greater morbidity. Changing demography makes it likely that there will be more cases of bipolar disorder in later life. Episodes can be misdiagnosed; for example a depressive mood swing presenting with withdrawal or a manic one with irritability.

Aetiology

The phenomenon of conversion to bipolarity after many years of unipolar depression has led to speculation that cerebral organic factors may play a part in the aetiology of late-onset mania. In support of this, cognitive function is significantly impaired in between a fifth and a third of elderly manics.[33,34] Furthermore studies[35] have shown a high rate of neurological disturbance, cerebral deep white matter lesions, and reduced heritability in late-life mania.

The term 'secondary mania' denotes manic illness which starts without a prior history of affective disorder in close temporal relationship to a physical illness or drug treatment and often in the absence of a family history of affective illness. A large number of conditions have been associated with secondary mania, including stroke, head injury, tumours, and non-specific lesions to the right side of the brain.

Course and prognosis

The prognosis for mania is similar to that for late-life depression; that is, there is an 80 to 90 per cent recovery in the acute phase but relapses and/or recurrences occur over time in about 50 per cent of cases.[35]

Treatment

As with younger patients, the mainstays of acute treatment are neuroleptics and mood stabilizers which include lithium and anticonvulsants, with ECT reserved for refractory cases. Increasingly atypical antipsychotics are used. These include olanzapine, risperidone, and quetiapine with aripiprazole as a possible new contender. Valproate preparations are the most widely used anticonvulsant in bipolar disorder.

Some general points to consider are: (1) a greater inter-individual variability in drug metabolism, which makes predicting the therapeutic dose difficult. Emergency rapid tranquillization with haloperidol 5 to 10 mg (often with 1 to 2 mg of lorazepam) can be used, but haloperidol has a long half-life and may lead to sudden immobility after a few days; (2) balancing risks caused by overactivity and exhaustion against an increased risk of falls in the elderly when using sedative tranquillizers; (3) an increased risk of confusion and delirium if anticholinergic drugs are given to counteract side effects; and (4) the higher risk of side effects and toxicity from lithium in older patients (including a risk even at what are considered therapeutic doses in younger patient).[35] The optimal treatment dose of lithium is not known. NICE recommends levels of 0.6 to 0.8 mmol/L for adults requiring maintenance lithium. Some old age psychiatrists use lower dosages but the evidence for low dose treatment in older patients is mixed.

The NICE Bipolar Guidance also covers the treatment of bipolar depression. The main message is that antidepressants, if used, should be combined with a mood stabilizer or an antipsychotic because of the risk of a manic switch.

Prevention

Given the high rate of relapse or recurrence, prophylaxis should be considered in all patients. There has been a steady shift away from lithium to valproate over recent years, although some argue this has occurred ahead of evidence.[36] Also, there is some evidence that lithium may reduce the risk of suicide in bipolar disorder as

well as being neuroprotective, possibly reducing the risk of dementia which some population studies have been shown to be raised in affective disorder.[31]

Further information

CRUSE Bereavement Centre (helpline@crusebereavementcare.org.uk, 0870 167 1677).

Bipolar Organisation (formerly the Manic Depressive Fellowship, http://www.mdf.org.uk/; telephone 08456 340 540 [UK Only]; 0044 207 793 2600 [Rest of world]).

MIND National Association for Mental Health, http://www.mind.org.uk.

Depression Alliance (http://www.depressionalliance.org).

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8.5.5 Stress-related, anxiety, and obsessional disorders in elderly people

James Lindesay

Stress-related, anxiety, and obsessional disorders in elderly people are common, distressing, costly to services, and potentially treatable. However, despite their clinical importance, many patients still go untreated, or are treated inappropriately. The specific conditions covered here are described in detail elsewhere; this chapter focuses on the differences and difficulties that are encountered when they occur in old age.

Classification

The ICD-10 and DSM-IV diagnostic classifications are described in Chapter 1.11. Although the term 'neurotic disorder' is not used in DSM-IV, it is retained in ICD-10 as a collective term for the disorders considered in this chapter. The extensive comorbidity between these conditions and their diagnostic instability over time are also apparent in elderly people, which supports the idea that they are better considered as aspects of a general neurotic syndrome than as discrete diagnostic categories.[1] This model is particularly applicable to elderly patients, whose illnesses are often the result of a long interaction between individual vulnerability, circumstances, and maladaptive responses to distress. Obsessive–compulsive disorder (OCD) is probably not part of a general neurotic syndrome. Although classified with the anxiety disorders in ICD-10 and DSM-IV, it has a number of features that suggest it is a distinct and stable condition with a different aetiology (see Chapter 4.8).

Clinical features

These disorders have psychological, somatic, and behavioural features. In elderly people, these symptoms and behaviours are similar to those seen in younger patients, but there are some important differences in how they manifest themselves or are perceived by others. Although most neurotic disorders in elderly patients are long standing, an important minority of cases have their onset in old age, and it is these that usually cause the greatest diagnostic difficulties.

Psychological symptoms

Symptoms of anxiety and depression occur to some extent in all of these disorders in late life. Depressive symptomatology in old age is described elsewhere (see Chapter 8.5.4). Regarding anxiety, the focus of the worries and fears of elderly people is on those issues that are of general concern in this age group (health, finances, crime). The phobias described by elderly people are similar to those seen in younger adults,[2] although some, such as the fear of falling, are more commonly seen in old age. Clinically significant anxieties and fears in elderly people are often dismissed as reasonable purely on grounds of age. In fact, it is physical frailty and the availability of social support that determine elderly people's perceptions of vulnerability and risk, and these rather than age should be considered when deciding whether or not concerns are reasonable.

The clinical features of OCD in old age are similar to those seen in younger patients. Obsessional symptoms rarely appear for the first time after the age of 50 years, and in such cases the possibility of an organic cause such as dementia or a space-occupying lesion should be investigated. They may also form part of a primary affective disorder.

Somatic symptoms

The somatic symptoms of anxiety are similar at all ages, but in elderly patients there is a greater likelihood of misdiagnosis and inappropriate investigation and treatment. This is particularly true of elderly patients experiencing panic attacks, who tend to be misdirected to cardiologists, neurologists, and gastroenterologists.

Behavioural disturbance

The psychological and somatic symptoms of anxiety have several adverse behavioural consequences, for example, phobic avoidance, the abuse of sedative drugs and alcohol, and the development of troublesome abnormal illness behaviours such as somatization and hypochondriasis. In elderly patients these behaviours are usually of long standing, but they can develop following the onset of anxiety or depression in old age. In cognitively impaired patients, disturbed behaviour may be the main presenting feature.

Diagnosis and differential diagnosis

In old age, these disorders usually present in primary care and the general hospital, and clinicians working in these settings need to be able to identify them, and to distinguish them from the other mental and physical disorders that they may accompany or mimic.

Depression

There is extensive comorbidity between neurotic disorders and depression, and depressive symptoms are an integral component of many neurotic disorders, particularly in old age. It is therefore important to assess to what extent depression forms part of the clinical picture, as this may require treatment in its own right. Depressive disorder that is comorbid with anxiety responds less well to antidepressant treatment, and there is a greater likelihood of relapse and recurrence.

Dementia

In the early stages, dementia may present with symptoms such as anxiety, and obsessionality. More commonly, anxiety and depression cause subjective cognitive impairment, which may be the presenting symptom. Dementia is associated with higher rates of anxiety, unrelated to severity of cognitive impairment. Patients with vascular dementia may be more vulnerable in this respect.

This anxiety may be associated with the implications of the diagnosis in those patients who retain insight, or a response to psychotic symptoms or misinterpretations of the external environment in those who are more severely affected. The caregivers of people with dementia are also vulnerable to developing depressive and anxiety disorders, particularly if they have a previous psychiatric history.

Delirium

Although delirium is a relatively quiet disorder in elderly patients (see Chapter 8.5.1), it may be associated with significant affective disturbances, often in response to frightening visual hallucinations and imagined assaults. Conversely, in vulnerable individuals, severe anxiety may be sufficient to precipitate delirium.

Paranoid states and schizophrenia

Patients suffering from these disorders may experience significant fear and anxiety in response to their psychotic experiences, but this rarely causes diagnostic difficulty. Unusual hypochondriacal ideas may sometimes be difficult to distinguish from monosymptomatic delusional disorders.

Physical illness

There is an important association between physical illness and neurotic disorders in old age. As a life event, an episode of physical illness may be the cause of neurotic disorder, particularly if it is severe or has sinister implications. For example, mild anxiety symptoms are common following myocardial infarction in old age, and vulnerable individuals may develop a disabling 'cardiac neurosis' focused on their somatic anxiety symptoms. Most cases of agoraphobia that develop after the age of 65 years are not induced by panic but arise following an alarming experience of physical ill health.[1] Follow-up studies of stroke survivors show that conditions such as agoraphobia and generalized anxiety are common, tend to become chronic in a significant proportion of cases, and are associated with poor functional recovery.[3] Chronic disabilities that limit mobility and independence, such as arthritis, balance disorders, and sensory impairments, increase the patient's sense of personal vulnerability and are also associated with elevated rates of anxiety and secondary avoidance.

Neurotic disorders can also cause physical illness by direct or indirect effects on the body. In elderly people, this may come about as the result of many years of harmful anxiety-driven behaviours such as smoking and alcohol abuse.

In terms of differential diagnosis, there is also the problem that a wide range of physical disorders may present with neurotic symptoms, and vice versa. In particular, a number of important cardiovascular, respiratory, and endocrine disorders may present with anxiety or depression and little else in old age.[4] Anxiety symptoms may also be caused by prescribed drugs such as oral hypoglycaemics and corticosteroids, or by excessive intake of caffeine and preparations containing sympathomimetics. In view of this, the clinical assessment should always include a drug history and a physical examination. A physical cause for neurotic symptoms should be considered if there is no past psychiatric history and no life event or other circumstances to account for their onset.

Epidemiology

While neurotic disorders are relatively uncommon in clinical populations, there are significant prevalence rates in community samples, indicating that they do not pass easily through the filters on the pathway to care. Surveys using different diagnostic criteria produce different rates of disorder, which makes comparisons difficult. However, some general findings include a female preponderance for most disorders, and a fall in prevalence and incidence rates with age. Most elderly people with neurotic disorders developed them before their fifties, but elderly cases of phobic disorder, panic, and OCD tend to be of later onset.[5,6]

Aetiology

The acquisition and subsequent loss or elaboration of the symptoms of anxiety and depression are determined by the patient's premorbid vulnerability, the particular factors that precipitate the episode of illness (destabilization), and the measures taken by the patient or the doctor to control it (restitution).[7]

Biological factors

Most of the evidence that biological factors play a role in the development of neurotic disorders derives from studies in younger subjects (see Chapters 4.7.1–4.7.3). These studies indicate that genetic factors contribute significantly to premorbid vulnerability, but the role that they play in old age is not known. Neuroimaging studies of elderly depressed patients provide only limited information about neurotic disorders, suggesting that patients with milder forms of depression and higher anxiety scores are more likely than severely depressed patients to have normal CT scans. It has been suggested that some anxiety disorders following stroke may be related to lesion location.[8]

Psychosocial factors

(a) Social adversity

This may have its effect through higher rates of physical illness and exposure to adverse life events, or by inculcating a sense of poor self-esteem. However, the impact of adversity on self-esteem in old age is not clear, since a hard life may in fact equip one to cope better with the difficulties of old age.

(b) Life events

Adverse life events have an important role in determining the onset of depressive and anxiety disorders (see Chapters 4.5.5.1 and 4.7.1–4.7.3). It is the meaning of the event to the individual that is important; loss events lead to depression and threatening events to anxiety. Some types of life events (physical illness, bereavement, retirement, institutionalization) are more common in old age, and are associated with psychiatric morbidity in vulnerable individuals.

Extreme trauma and catastrophe are well known to have adverse psychological consequences, and post-traumatic stress disorder (PTSD) is a recognized diagnosis in ICD-10 and DSM-IV (see Chapter 4.6.2). It is clear from studies of elderly survivors of traumatic experiences such as war and the holocaust that PTSD is often a persistent disorder, and that its onset or recurrence may be precipitated by events many years after the original traumatic

experience. PTSD may also develop following trauma in late life, and as in younger subjects it tends to persist.

(c) Early experience

Early parental loss and childhood physical and sexual abuse are associated with the development of mental disorder in adult life, an effect that persists into old age.[1]

(d) Relationships

There is evidence that both the quantity and quality of social relationships are important determinants of psychological well-being in old age, and that factors such as smaller social networks are associated with anxiety disorders.[9]

Course and prognosis

The limited evidence suggests that neurotic disorders in elderly people tend to become chronic and that older age of onset is a predictor of poor outcome, particularly in men.[10] The pattern of symptoms may change over time.[11] Elderly patients with anxiety disorders have an excess mortality.

Treatment

Evidence

There is little high-quality evidence available about the effects of treatments for these disorders in elderly patients. Cognitive behaviour therapy (CBT) is of proven benefit in younger adults (see Chapter 6.3.2.1), and there is some evidence that it is also effective in later life.[12] There have been surprisingly few trials of anxiolytic drugs in elderly patients, but there is evidence that antidepressant drugs such as SSRIs and venlafaxine are effective in the treatment of generalized anxiety. In the absence of good evidence for a particular treatment, patient preference, and choice are important considerations.

Management

Since most patients are seen in primary care and general medical settings, this is where the focus of management should be. The role of specialist old age psychiatry services should be to provide any advice and support that is necessary, which may include assuming responsibility for the most complex cases. Wherever the patient is seen and treated, the following should need consideration from the outset (see also Chapter 8.6):

- a thorough assessment and accurate diagnosis as the basis for the management plan
- the full range of physical and psychological treatment options, including patient education, lifestyle advice, bibliotherapy, and supportive counselling
- clear goals for the treatment plan, agreed if possible with the patient
- an adequate trial of treatment
- the likely duration of treatment
- frequency of review
- any possible adverse consequences, such as dependency, adverse side effects, risk of self-harm.

(a) Psychological treatments

For the most part, the goals and techniques of CBT are the same for elderly patients as they are for younger adults (see Chapter 6.3.2.1). However, these may require some adaptation to accommodate sensory impairments, physical illness and disability, and cognitive dysfunction.[13] The need to tailor treatment to the individual may limit the value of CBT in a group setting, although this has to be set against the benefits of shared experience and peer support. Group treatment is probably more straightforward with task-centred activities such as anxiety management.

The use of formal psychodynamic approaches to management is currently limited by economic constraints, and the lack of evidence regarding their effectiveness. However, health professionals should have some knowledge of the psychodynamics that underlie the concerns of elderly patients, and the mental defences that they use. They also need to be aware of their own preconceptions and cognitive distortions regarding the experience of old age, the psychological sophistication of elderly people, and their capacity for growth and change.

(b) Physical treatments

Chapter 8.6 describes the general principles of drug treatment in old age. None of the drugs used to treat anxiety in elderly people is entirely without problems, so they should be prescribed with care.[14] Benzodiazepines are the most commonly used drugs, but they are often prescribed inappropriately. Elderly patients are particularly sensitive to their adverse effects, and drug accumulation may lead to delirium, incontinence, and falls. Compounds with short half-lives and no active metabolites, such as oxazepam, are least problematic, although patients may develop withdrawal symptoms if they are discontinued, or taken erratically. Long-term benzodiazepine use should be avoided where possible, although it may be necessary in a few patients unresponsive to other forms of treatment.

Antidepressant drugs are now the first choice in generalized anxiety and panic, particularly if depressive symptoms are prominent. The use of antidepressants in elderly patients is discussed in Chapter 8.5.4. SSRIs also have a specific effect in OCD. Neuroleptics have only a limited role in the management of anxiety, given their potentially disabling extrapyramidal side effects. However, a short course of low-dose treatment with a drug such as haloperidol or zuclopenthixol may be considered in those unable to tolerate benzodiazepines. Alternatively, sedative antihistamine drugs such as hydroxyzine may be useful. β-blockers are used in younger adults to control the sympathetic somatic anxiety symptoms, but contraindications such as chronic obstructive airways disease, sinus bradycardia, and heart failure limit their use in elderly patients. Buspirone is an azapirone anxiolytic that is well tolerated by elderly patients, but it takes about 2 weeks to become effective, so is not useful for the management of acute episodes. It is indicated for severe chronic generalized anxiety and in patients where there is risk of dependence or abuse.

Prevention

The possibilities for primary prevention are limited at present. However, in view of the association with physical illness, there may be an opportunity to intervene and prevent the development of chronic neurotic disability following strokes, heart attacks, and

falls. It remains to be seen if the improved management of these disorders earlier in life will result in lower rates of chronicity and recurrence as cohorts age.

Further information

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8.5.6 Personality disorders in the elderly

Suzanne Holroyd

Introduction

The study of personality disorder (PD) in late life presents conceptual, diagnostic, and methodological difficulties. By definition, PD is considered a group of personality traits that relatively persistent through adulthood. However, the concept of PD persisting throughout the lifespan contradicts widespread clinical belief that they become less severe with ageing. For example, DSM-IV[1] notes that 'some types of personality disorders . . . tend to become less evident or remit with age'.

There are difficulties in studying PD in the elderly. One is the instability of the definition of PD over time, making it difficult to relate earlier studies to those using current definitions of PD. In addition, diagnostic criteria are subject to criticism when applied to the elderly, in that they may be 'age-biased'.[2,3] Finally, the methodology used to diagnose PD has been highly variable and difficult to interpret between studies.

A major issue is whether personality is fully developed by early adulthood and then remains unchanged, or whether personality continues to develop and change throughout life. The work of McCrae and Costa[4] demonstrated that personality characteristics are relatively stable within individuals over a 30-year period with correlations ranging from 0.7 to 0.8. However, this also demonstrates that complete stability is not there and suggests certain aspects of the personality may still develop and change with ageing. This suggests that PD may also change over the lifespan.

Another issue is whether underlying traits that persist throughout the lifespan can rise to the level of a PD depending on the environment. For example, traits that may be personality disordered in young adult life, such as extreme dependency, may be an appropriate and adaptive trait for an older individual with multiple physical disabilities.[5] Conversely, an individual may have a trait of extreme independence that may be adaptive in earlier life but which leads to distress and maladaptive functioning in a setting requiring dependence, such as a nursing home. Thus it is possible to have a PD diagnosed for the first time in late life, which goes against the very definition of lifelong PD.

Clinical features

Clinical features of PD and details of their classification are reviewed in Chapters 4.12.2 and 4.12.3. However, there is difficulty in simply relating these criteria, which were developed for younger individuals, to the elderly. Typical diagnostic behaviours that clinicians associate with PD in younger adults may present as different behaviours in the elderly. This may lead clinicians to overlook personality traits and disorders in older individuals. For example, a criterion of antisocial personality disorder is the repeated failure to sustain consistent work, behaviour not applicable to the older retired individual. Yet it is possible that the personality trait of irresponsibility, which led to the loss of jobs earlier in life continues, now appearing as a behaviour such as medicine non-compliance. Some authors have thus argued that the clinical

features for some PD are age-biased since certain behaviours are less likely to occur in elderly persons despite the persistence of personality traits.[2,3]

Diagnosis of personality disorders

Diagnostic criteria for PD are discussed in Chapter 4.12.3. Because features of PD may change with ageing, diagnosis can be difficult. Overlap with Axis I diagnoses such as depression or dementia make the diagnosis even more challenging. For example, depressed elderly people have symptoms normally associated with PD as they may be more dependent, avoidant, resistant, negative, and somatic.[6] In addition, depressed elderly people may view their lives negatively and overestimate personality psychopathology.[7,8]

Clinicians may be reticent to give a personality diagnosis to an individual with multiple medical problems to which maladaptive behaviours may be attributed even if a lifelong history of personality pathology is established.[8] They may also be concerned about the validity of historical information needed to make a PD diagnosis. Therefore, in making a diagnosis of PD, a clinician should take a thorough history from as many reliable outside informants as possible. If the patient is in a state of acute distress with a current Axis I diagnosis such as depression, it is best to defer diagnosis of the PD until the illness is in remission. Otherwise, it is especially important to ask outside informants to think back to when the individual was a younger person, as current symptoms can colour the perception of lifelong personality traits. Asking for specific examples of history such as, details of relationships and job history, legal history, and the like, will be more helpful than just general descriptions of personality.

If behavioural difficulties and personality problems are found to be recent, the clinician needs to search carefully for a superimposed medical condition, or a psychiatric condition such as depression. Clinicians should carefully screen for illnesses such as dementia, stroke, or other neurological disease, or a systemic medical illness. Those with frontal lobe dementia, Alzheimer's disease, or vascular dementia may have personality changes early in their life course.[9–11]

Epidemiology and aetiology

The prevalence of PD in the elderly varies as to the methodology used and the population studied. It should be noted that no assessment instrument for PD in the elderly has been validated.

Community studies

Community studies have been the most useful to date. A community study,[12] using the Epidemiologic Catchment Area (**ECA**) data, had 841 subjects examined by psychiatrists using the semi-structured Standardized Psychiatric Examination with DSM-III criteria. Comparing those over the age of 55 with those under 55, older individuals were found significantly less likely to have a PD (6.6 to 10.5 per cent) as compared with younger individuals. This finding was almost entirely due to a three-fold higher prevalence of cluster B PD in those under the age of 55, especially antisocial and histrionic PD. Interestingly, in this study none of the older individuals were found to have cluster A PD. Table 8.5.6.1 summarizes the findings of this large community study. The strengths of this study were that it was a community rather than a clinical sample, and subjects were evaluated by psychiatrists using a structured questionnaire. Limitations of this study were those inherent to the study of PD in late life, in that older subjects may have been inaccurate in recalling maladaptive behaviours, outside informants were not used, and lack of non-validated instruments for diagnosing PD in the elderly.

Table 8.5.6.1 Weighted prevalence (%) of DSM-III personality disorders in a large community study

	Age < 55 years	Age > 55 years
Cluster A	0.1	0.0
Paranoid	0.0	0.0
Schizoid	0.1	0.0
Schizotypal	0.1	0.0
Cluster B	6.8	2.2*
Antisocial	2.7	0.1*
Borderline	0.8	0.0
Histrionic	4.3	2.2*
Narcissistic	0.0	0.0
Cluster C	3.8	4.3
Avoidant	0.0	0.0
Dependent	0.2	0.1
Obsessive–compulsive	3.6	3.3
Passive-aggressive	0.0	1.0
Any personality disorder	10.5	6.6*

* $p<0.05$

Reproduced from B.J. Cohen *et al.* (1994). Personality disorders in later life. A community study. *British Journal of Psychiatry*, **165**, 493–9, copyright 1994, The Royal College of Psychiatrists.

A community study of 43 093 persons, examining alcohol and related conditions across the life span, confirmed that those over 65 years had significantly lower rates of all studied PD including avoidant, obsessive–compulsive, paranoid, schizoid, histrionic, and antisocial, using DSM-IV criteria.[13]

A community survey study of DSM-III PD traits using the Personality Diagnostic Questionnaire revealed that 'dramatic' and 'anxious' personality traits declined up to 60 years of age with a slight increase thereafter, but that 'odd' or 'eccentric' traits showed no change with age.[14]

Psychiatric populations

In addition to community samples, specific clinical samples have been examined. Limitations of these studies are the possibility of over diagnosis of PD due to symptoms of Axis I diagnoses.

A retrospective study of 2322 psychiatric inpatients with major depression found the prevalence of PD to be 11.2 per cent in those over the age of 65 as compared with 17.2 per cent for those under 65.[15]

Psychiatric inpatient studies suffer from the limitation of diagnosing PD in the face of an acute psychiatric illness requiring hospitalization, making them likely to overdiagnose. Such studies are of very limited value.[16,17]

Unfortunately, outpatient studies have similar limitations when examining those with concurrent Axis I diagnoses. A study of 36 psychiatric outpatients, including those with bipolar disorder, delusional disorder, and schizophrenia, revealed that 58 per cent had a diagnosis of personality disorder.[18] Arguably, diagnosing

personality disorder in the face of these disorders is likely to be difficult and result in an overestimation.

Prevalence summary

A meta-analysis of 11 articles published from 1980 to 1994 of personality disorders based on DSM-III or DSM-IIIR criteria revealed an approximately 10 per cent prevalence of personality disorders in those aged 50 and over.[19] In comparing these studies it was noted that the method of diagnosis affected the prevalence of personality disorder. In conclusion, the authors felt that there was a definite need for well-designed studies using statistically robust samples to assess the true prevalence of personality disorders in late life.

Taking the best studies together—the ECA community study and the meta-analysis—the prevalence of personality disorder in the elderly, as currently defined, ranges from 7 to 10 per cent, with a decrease in prevalence of cluster B diagnoses.

Course and prognosis

Longitudinal community studies of PD have not been performed. With longitudinal data lacking, only cross-sectional studies are available. However, cross-sectional studies have a variety of limitations, including the possibility of a cohort effect explaining changes in prevalence in late life.

Antisocial personality disorder has been the best studied. The ECA study revealed that antisocial personality disorder declined from a 1-month prevalence of 0.9 per cent for individuals between 25 and 44 years of age to 0 per cent for those over the age of 65. When considering men only, the rate fell from 1.5 per cent in those aged 22 to 44 to 0.1 per cent in those over 65.[20] Supporting this is a study revealing the decline in lifetime prevalence in antisocial personality disorder from between 2.1 and 3.3 per cent to between 0.2 and 0.8 per cent in those aged 65 and older.[21] In addition, antisocial traits, as measured by the Minnesota Multiphasic Personality Index, reveal a decline with ageing.[22] However, a forensic centre study revealed while antisocial PD declined after the age of 27, one-third remained criminally active throughout their lives.[23]

Several hypotheses exist to explain this apparent decline in antisocial personality disorder with ageing. Personality may continue to mature and develop. Early death due to high-risk behaviour or a change in antisocial behaviours to other symptoms including hyperchondriasis, depression, or alcoholism may occur.[23] Also, behaviours such as criminality may decrease in older individuals, but antisocial personality traits remain and are simply more difficult to measure using current diagnostic criteria. Also, decrease in impulsive and aggressive behaviours may correlate with full myelination of frontal, temporal, and parietal cortices that does not occur until 30 or 40 years of age.[24] Changes in brain neurochemistry with ageing, including serotonin and dopamine, may also result in decreased impulsiveness or aggressiveness.[12] Decreased testosterone levels in men with ageing may contribute to a decline in these traits.[25]

Other Cluster B disorders may decline with ageing. The ECA study previously reviewed found a decline in antisocial and histrionic disorder.[23] Another community study supported a decline in histrionic PD with ageing.[26] Interestingly, the pattern of decline varied with gender, with rates remaining constant in women but declining in men. Similarly, a diagnosis of borderline PD is rare in elderly individuals, with only two case reports in the literature.[5] There is conflicting data regarding a decline in cluster A or C diagnoses. A large community study revealed lower rates of both Cluster A and C diagnoses in the elderly.[13] However, a study of schizotypal PD revealed all cases began before 40 years of age and continued lifelong.[27]

Some work has been done on the interaction of PD with Axis I diagnoses. Studies of depressed elderly patients suggest PD is associated with earlier age of depression onset, chronicity, and severity of dysthymia[28,29] however depression may exacerbate or conceal personality traits, thus making firm conclusions of PD in such individuals difficult. In the ECA study, certain Axis I diagnoses were found to be more common with a PD diagnosis. For example, all cases of obsessive–compulsive disorder in older individuals occurred concurrent with a PD.[12] Both generalized anxiety disorder and substance use disorders were more common in the presence of a PD. There were no differences in the prevalence of schizophrenia and major depression in those with or without a PD. The findings need to be confirmed since the group of elderly with PD was small.

A recent interesting study has revealed any PD (DSM-IV criteria) is associated with increased risk of stroke and ischemic heart disease, adding to the possible morbidity of these disorders.[30] However, such results should be viewed with caution as no screening was done to rule out depression or other associated factors that are prevalent in this population and may have led to over diagnosis of PD.

Treatment and management

Given the relative lack of data regarding PD in the elderly, it is not surprising there is a corresponding lack of information regarding treatment and management. In general, clinicians should have a low threshold for suspecting concurrent psychiatric diagnosis, as major depression, anxiety, substance use disorders or dementia may mimic or exacerbate a personality disorder.[5] Physical and medical problems should be thoroughly evaluated and treated to minimize any associated complaints.

Social and family supports should be explored and maximized. Firm and consistent limits must be set by the clinician for both patients and their families in regard to inappropriate behaviour.[5] Clinicians should also try to determine why the disordered behaviour is occurring at a particular time. For example, placement in a nursing home may be stressful to an individual who has had difficulty forming relationships and is now dependent on a group of caregivers. Psychotherapy with the goal of focusing on current life stresses, the individual's vulnerabilities, and adaptive strategies can help the patient adjust to the current circumstance.[5]

Psychotherapeutic treatments used for personality disorders in younger individuals may be tried although little data exists on their effectiveness for elderly. A study of Dialectical Behavior Therapy (DBT) used in combination with medication to treat elderly depressives with personality disorders revealed better results than just medication. The study is limited in that such results are common in other studies using any psychotherapy with medication versus medication alone and does not support a specific usefulness of DBT in elderly personality disorder. However, the study is the first to use DBT in an older population and shows it is tolerable in this age group.

If possible, psychiatric medication should be avoided unless there is a specific diagnosed condition. This will minimize the possibility

of side effects in elderly individuals and avoid dependency and control issues.[5] This is important as elderly individuals with abnormal personality traits have been found to be at higher risk of receiving psychotropic medication.[31]

Possibilities for prevention

There are no data available for preventing the development of a PD in late life. Clearly, more information is needed on the longitudinal course of diagnosed PD in late life so that information regarding treatment and prevention may be realized.

Further information

At the time of writing, there are no books or reports that give an overview of personality disorder in the elderly. Further information about specific aspects of the subject can be obtained from the relevant references in the text.

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8.5.7 Suicide and deliberate self-harm in elderly people

Robin Jacoby

Introduction

Although in some countries suicide rates in young males have risen dramatically in the last decade or so, suicide in old age is important because rates in older people, especially those over 74, are still proportionately higher in most countries of the world where reasonably reliable statistics can be obtained.[1] For example, in 2004 in Lithuania where suicide incidence is currently the highest, the

overall rate in males per 100 000 total population was 70.1, but in men over 74 the rate was 80.2. In the United States, where suicide is neither especially common nor rare, in 2002 the overall rate for males per 100 000 total population was 17.9, but 40.7 in men over 74. Rates for older women are nearly always much lower than for their male counterparts.

A second reason for the importance of suicide in old age is that the proportion of older people in the population is rising worldwide. Indeed, the increase in developing countries is likely to be even greater than in developed countries. Although rates vary from year to year and birth cohort to cohort, it is highly likely that unless suicide prevention becomes a great deal more effective than at present, more and more older people will kill themselves in the coming years.

As with younger people, completed suicide in old age may be seen as part of a continuum from suicidal thinking through deliberate self-harm (which does not lead to death), to completed suicide. An added component within this continuum for older people is that of 'indirect self-destructive behaviour', such as refusal to eat and drink or 'turning one's face to the wall' which is clearly intended to hasten death. Finally, although this section does not deal with euthanasia and related issues, assisted suicide in people with terminal illness such Alzheimer's disease and cancer may also be seen as part of the suicide continuum.

Suicidal thinking in community-dwelling elderly people

A number of studies have explored this issue. Fleeting thoughts of suicide or the idea that life is not worth living occur in up to about 15 per cent of community-dwelling older people,[2] but serious consideration of suicide is very much less.[3] It is those older people with mental disorders, mostly depressive, who show a higher frequency of thoughts that life is not worth living and harbour ideas of committing suicide. It seems logical to suppose, therefore, that depressed elders should be the target of suicide prevention strategies.

Indirect self-destructive behaviour

Unlike the young, some elderly people have the possibility open to them of behaving passively in such a way as to hasten death. This may happen either by refusing medical treatment essential to maintain life, or simply by declining to eat and drink—'turning one's face to the wall'. As regards the latter, many people, especially non-medical, believe that this is reasonable behaviour akin to so-called 'rational suicide', and court rulings have sanctioned it. There is no doubt that there are several cases in which a person's right to refuse treatment or nutrition, for example during the terminal phase of cancer, should and would be respected. However, it has been argued that many of such cases suffer from undiagnosed but treatable depressive illnesses. Some support for this point of view was provided by a questionnaire study of more than 1000 residential and nursing home administrators in the United States.[4] Cognitive impairment, loss events, refusing medication, food, and drink, loneliness, feeling rejected by families are all risk factors for indirect suicidal behaviour in residential homes.[5] It is wise, therefore, that no one should be permitted to turn his or her face to the wall before assessment for the presence of a treatable depressive disorder.

Deliberate self-harm

Incidence

It is less possible to make a clear distinction between deliberate self-harm (DSH) and completed suicide in older than younger people. DSH at all ages has been quite extensively studied, but for obvious reasons mainly in hospital samples, and it is possible that several cases are undetected in the community. Broadly speaking the incidence curve for DSH is highest for the young and declines with age, whereas that for completed suicide rises with age. By the same token suicidal intent behind acts of DSH in older people is significantly greater than in younger adults.[6] In clinical practice it is therefore wise to consider deliberate self-harm in those over 75 as failed suicide.

Sex

As with completed suicide, rates for DSH differ quite widely from country to country. As with younger attempters, females outnumber males at a raw number ratio of approximately 3:2, but the *proportionate* gender ratio is approximately unity because fewer males survive into old age. Contrast this with completed suicide where men clearly outnumber women.

Methods

Deliberate drug overdose is the favoured method for DSH at all ages in Western countries; in some others, corrosive poisons or detergents are used. The most common types of drug for overdose are benzodiazepines, analgesics, and antidepressants. After drugs, self-cutting is the next most frequent method.

Psychiatric diagnosis

Older people are more likely to be assigned a psychiatric diagnosis after DSH, about half suffering from major depressive disorder, up to about a third from alcohol abuse, and under 10 per cent from other disorders.[7] Only about 10 per cent have no psychiatric diagnosis at all. Alcohol abuse together with depressive disorder augments the risk of DSH in older people. The status of cerebral organic disorder is uncertain because selection bias in reported case series reduces comparability. However, mild cognitive impairment and a co-morbid depressive disorder have been considered risk factors, and should be borne in mind by the clinician, if only on common-sense grounds. Personality factors have been implicated in DSH in older people, but research data are too poor and too few to make reliable statements on the subject.

Risk factors

Risk factors for deliberate self-harm in elderly people include: physical illness; widowhood and divorce or separation from a cohabitee; social isolation and loneliness (not the same thing); or simply living alone.[6,7] Unresolved grief, usually after death of a spouse, is a commonly found risk factor. The threat of transfer to a nursing home is, unsurprisingly, a precipitant of deliberate self-harm, although once an elderly patient is transferred to institutional care the risk of an overdose or some other attempt at suicide is reduced, probably because of lower access to the means and higher supervision. Surprisingly perhaps, terminal illness is not commonly found in older patients who attempt suicide but

fail, although hitherto undiagnosed but treatable physical disorders are sometimes revealed.

In keeping with the fact that more older suicide attempters are assigned a psychiatric diagnosis than younger ones is the fact that about 50 to 90 per cent, depending on the case series, undergo some form of psychiatric treatment as a result of the act of deliberate self-harm. Although fewer older people commit DSH than younger ones (about 5 per cent compared with 12 per cent) the risk of subsequent completed suicide is higher, compared with people of all ages (about 7 per cent compared with 3 per cent). Individual risk factors for later successful suicide include being male, having a prior psychiatric history, divorce, and current treatment for a persistent depressive illness.[6,8]

Completed suicide

Rates

The point has already been made in the opening paragraph of this chapter that suicide rates are still highest in the oldest old in most countries. Men outnumber women by about three or four to one in most countries; the exception being rural China.[1] However, suicide rates in the old have in fact been declining in many industrialized countries over the past 25 years, whilst those in young males have been rising—a reminder of the maxim that rates in all groups can and do vary over time and between countries, so that general conclusions about suicide should always take context into account. The reasons for incidence variations are discussed elsewhere, but socio-economic conditions and access to means play their part with the old as well as younger suicide victims.

Suicide at all ages is associated with divorce, widowhood, and single marital status. Widowers are more likely to kill themselves than widows, which has relevance for old age psychiatry, since in the overall population there are more old widowers than young ones.

Methods

Methods of suicide chosen by older people depend to a great extent on availability. In the United States, firearms are used by the majority of older men who kill themselves.[9] Shooting is also commonly chosen in Australia and Finland. In the United Kingdom, which has more stringent firearms control, drug overdose, especially in women and frequently with combination analgesics, hanging (especially in men), suffocation, or jumping from tall structures are preferred methods.[10] In Japan hanging, in Hong Kong jumping from one of the many very high buildings, and in Sri Lanka organophosphate poisoning are the commonest means in use.

Planning

Suicide in older persons is marked by careful planning and about half of the victims leave a note to indicate why or to confirm that they have killed themselves.[10] Suicide pacts are generally rare, but half of those that do occur involve people over 65. A previous history of a suicide attempt (DSH) is found in about a third of those older people who kill themselves.

Psychiatric diagnosis

Studies, including case-control, in the United States, Scandinavia, and the United Kingdom have found that 70 per cent of older suicide victims suffer from a mental illness, most commonly a major depressive disorder at the time they die.[11–14] Chronic symptoms of depression and a first depressive illness in later life are associated with a greater risk of suicide. Untreated or inadequately treated depressive illness is also found more commonly in elderly suicides. By contrast with younger suicide victims, alcohol and drug abuse rates are lower in elderly people, although co-morbid depression and alcohol abuse do occur more frequently than by chance. Schizophrenia or schizophrenia-like disorders are found less commonly in older than younger suicides. Similarly, cerebral organic impairment or dementia are infrequent and even absent from some series of cases. There has been more recent interest in the role of personality in suicide in older people. In various studies obsessional or anankastic traits which researchers have called 'low openness to experience' have been shown to predispose to suicide.[14,15]

Co-morbid physical illness

Co-morbid physical illness is, on common-sense grounds alone, likely to be a risk factor for suicide in older people and this has been confirmed in a number of studies.[16–18] Also, older people are much more likely to have visited their primary care doctor in the month prior to killing themselves than are younger suicide victims, and furthermore more likely to complain of physical than mental symptoms. Nevertheless, suicide to bring about the premature ending of a terminal illness or the avoidance of pain, although found to be a factor in studies of a variety of specific diseases, is not as common as one might imagine.

Social risk factors

Social risk factors for suicide in old age have been found to include: isolation and poor social integration; lack of a person to confide in; and concerns over dependence or a move from home to residential care.[13,18–20] Bereavement by itself is no more of a risk factor in older than the younger suicides, but a grief reaction prolonged for more than a year has been found to increase the risk.

Risk assessment

The study of suicide at any age is primarily for the purpose of prevention. In older people this means that, episodes of deliberate self-harm need to be considered as serious, even and perhaps especially when they do not appear to be so. A quantitatively small overdose of a relatively less lethal drug is frequently no indication of the seriousness of suicidal intent. Primary care doctors should be aware of the suicide risk in those attending with physical disorders, especially where the patient's complaints seem to be out of proportion to the actual evidence of disease. Nor is the identification of a physical illness a reason to relax vigilance over suicide risk. Dismissal of an older person's wish to die as 'rational' is probably wrong in the great majority of cases, but in any case should never be done before a thorough assessment of the mental state concentrating in particular on depressive disorder. Anxiety is frequently so prominently a presenting symptom of depressive disorder in elderly people, that other manifestations, such as suicidal thinking, may be overlooked. Whilst elderly people respond well to antidepressant medication, many live alone. Thus, a prescription for perhaps a month of treatment might be an enhancement of suicide risk. It is therefore prudent either to arrange close supervision or administration of medication by a carer rather than the patient

themselves, or for no more than a week's supply to be dispensed at a time. Pharmacists may be willing to assist in this by providing proprietary boxes with compartments for each dose.

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8.5.8 Sex in old age

John Kellett and Catherine Oppenheimer

Introduction

A Darwinian sees man as devoted to reproducing himself. The decline of fertility with age makes one question the biological purpose of sexuality in the senium. Is it simply the remains of a once useful behaviour, a vestigial characteristic? The fact remains that sexual interest and sexual activity, both as sources of enjoyment and as important components of pair bonding, continue among men and women even into extreme old age. However, as Alex Comfort memorably remarked, 'old people give up sex for the same reasons that they give up cycling—general infirmity, fear of looking ridiculous, no bicycle'.[1] Or, more soberly: the common obstacles to the continued enjoyment of sex in old age are illness, attitudes, and demography.

Surveys of sexuality in old age

A comprehensive discussion of surveys in this field can be found in Bouman.[2] These vary widely in their focus, setting, methods, and target age groups. The details are of great interest but only the main themes and a few illustrative examples can be described here.

Methodology

As with all surveys, one has to consider what factors influenced the selection (and self-selection) of the responders. In general, participants in these studies tend to be better educated and more liberal in their attitudes than their contemporaries; and important groups, such as people with chronic illness, may be under (or over) represented, depending on the setting of the survey.

Cross-sectional and longitudinal studies

Attempts to assess the effects of ageing on sexuality by surveying different age-groups at a single point in time are vulnerable to *cohort effects*—the sexual experience of those brought up before the Second World War is different from that of people whose childhood was in the 1950s, and need have nothing to do with age. Longitudinal studies are demanding and costly, but they reveal the effects of ageing more clearly, and they also allow individual patterns, stable over time, to be identified. For example, the series

of studies conducted at Duke University showed that although the *prevalence* of sexual interest and activity in both men and women decreases with age, *individual* patterns of sexuality tend to be stable until some event (such as illness or loss of a partner) disrupts the pattern.[3]

Sexual interest and sexual pleasure

There is more to sex than actual intercourse, especially in old age. A consistent finding is that in both sexes interest in sex is more resistant to ageing than is sexual activity, even when non-coital activity is included. For example, in a cross-sectional study of volunteer respondents aged 80 or over, living in a residential home in the United States,[4] 88 per cent of the men and 71 per cent of the women enjoyed daydreams and fantasies about sex, 82 per cent of the men and 61 per cent of the women engaged in touching and caressing, while 63 per cent of the men and 30 per cent of the women had sexual intercourse 'at least sometimes'. Janus and Janus[5] surveyed 2765 subjects in the United States by questionnaire, supplemented by 125 interviews. They found less reduction of activity in the older groups than had been found in earlier surveys, probably because they did not confine the question to coitus. In every age group men thought that they were more active than 3 years previously, but women, particularly those over 50, noted a decline. Unlike earlier studies foreplay was discussed, and the authors concluded that older men gain greater pleasure and experience more intimacy and warmth after coitus than younger men.

Obstacles to continued sexual activity

The commonest reasons given by people for a decline in their accustomed level of sexual activity were illness, and the loss of their partner.[3] Interestingly, women (but not men) also gave illness of their partner as a reason. This may reflect the traditional male role in initiating sexual activity, and also perhaps the fact that husbands tend to be older (therefore more at risk of illness) than their wives. For example, in a Swedish community study of 85-year olds,[6] participation in sexual intercourse was reported by 10 per cent of married women but only 1 per cent of the unmarried women, and by 22 per cent of married men compared to 13 per cent of the unmarried men. The rates for sexual interest (as opposed to activity) were higher: 46 and 37 per cent for married and unmarried men respectively; 24 and 15 per cent for married and unmarried women.

Attitudes to sexuality

Attitudes towards sexuality in old age—as revealed in responses to systematically varied vignettes—have become generally more positive over the last half-century, among both younger and older people.[2,7] Probably this is true also of professional attitudes, and where this matters most is among the staff caring for older people in institutional settings. The evidence suggests that care staff who are older, better educated and have had vocational training, and who have more experience of caring, are likely to be more open to the sexual needs of their residents. However as Bouman[2] points out, the same cannot be said of health policies and strategy. Government guidance on the care of older people has ignored their sexuality, and in most official surveys and policies on sexual issues, people aged 60 or more are excluded from consideration.

Demography

With increasing life-expectancy many marriages continue well into the 9th decade of one or both partners. Divorce has now overtaken death as the main cause for the ending of a marriage, though this does not necessarily mean increasing numbers of people left single in old age. Many older people experience second or third marriages (or cohabitations), and the age gap between the partners in these new relationships tends to be larger than that between partners still in their first marriage. The complex effects on family structure of these different social trends are analysed by Harper.[8] She shows that married older people have higher levels of health, social participation, and life satisfaction than those not married, and they live longer; while divorced men (compared to women, and to widowers) are the most disadvantaged in those respects. To this we can add (based on the survey data mentioned above) that in old age married people also enjoy greater opportunities than the unmarried for sexual expression.

Sexual orientation

Much less is known and written about the sexual lives of older people who are not heterosexual in their orientation. Despite the prevailing trend in Western societies towards valuing diversity, most older homosexual people in their earlier lives will have feared—or faced—stigma, discrimination, even the threat of criminal procedure, and may still face such discrimination. Further discussion of this important group of people can be found in Bouman.[2]

Sexuality and dementia

The effect of dementia on sexual interest and activity is unpredictable. Most often it is associated with a decline in interest, but sometimes (probably in less than 10 per cent of cases) a person with dementia may become more sexually demanding, or may lose the ability to judge when the expression of sexual interest is unwanted or out of place.[2,9] The effect of the patient's dementia on the spouse is also difficult to predict. In some couples the physical relationship continues as an important expression of their affection, support, and concern for each other. More commonly, sexual activity declines. For example, Wright[10] followed a group of couples in which one partner had dementia, alongside a control group of couples without dementia. Only 27 per cent of the afflicted couples continued sexual contact over the 5 years after diagnosis, compared to 82 per cent of the control couples.

Patients with dementia who have altered sexual behaviour, or who can no longer make reliable judgments about potentially sexual social situations, become vulnerable to misunderstanding, exploitation, or censure. In marriages the impact of these changes is often borne alone by the spouse, who also keeps them hidden from others. But people with dementia who live alone or who go into institutional care (even if only temporarily, into hospital perhaps) are not so protected, and clinical staff may be asked to intervene—to control behaviour that has offended or is harmful to others, or to reduce risk to a vulnerable patient. It may be crucial to establish whether such a patient is making an autonomous choice (for example, to engage in sexual contact with a fellow resident) or whether their failure of understanding is being exploited; and Lichtenberg and Strzepek[11] describe a helpfully structured approach to this question.

Normal sexual function in old age

The normal human sexual response cycle and the physiology of sexual intercourse are described in Chapter 4.11.1 in this textbook. These descriptions hold true for sexually active older people, although age does bring some relatively minor physical changes (see Table 8.5.8.1, based on data from Masters and Johnson[12])—changes experienced by some but not necessarily by all. The most noticeable of these are probably the much longer refractory period in older men (the interval which follows ejaculation before renewed erection is possible), and the oestrogen-dependent changes in vaginal tissue and lubrication in older women. However, according to the self-reports of older sexually active people, the capacity for sexual pleasure and the quality of orgasms are not at all affected by age.

Sexual dysfunction in old age

The range of sexual dysfunctions is discussed in detail in Chapter 4.11.2 in this textbook. In old age, as mentioned earlier, physical illnesses and their treatments assume increasing importance in curtailing the normal sexual activity and interest of an established couple. Likewise, with ageing, the balance between psychological and physical factors in the causation of a sexual problem, tips towards the physical. An example of this is erectile dysfunction, where a notable increase in research into the physiology of penile erection has led to a number of effective physical treatments, especially relevant to erectile problems associated with illnesses common in old age (such as diabetes and vascular disorders).[13,14] However, a clearer understanding of the physical components of a problem does not diminish the importance of psychological factors. Myocardial infarction, probably accompanied by hypertension and atherosclerosis, provides a good example. After the infarction the couple may misperceive the breathlessness of orgasm as cardiac distress, and they may need encouragement to resume sexual relations which may reduce the risk of further infarction.[15] Even a hospital admission interrupts the couple's sexual routine which may then be difficult to resume.

Some of the most common physical causes of sexual dysfunction are listed in Tables 8.5.8.2, 8.5.8.3, and 8.5.8.4.

Table 8.5.8.1 Physical changes of ageing

	Male	Female
Retained	Nil	Nipple erection Clitoral tumescence and retraction
Reduced	Penile and nipple erection Testicle elevation Power of ejaculation Rectal contractions	Vaginal lubrication and expansion Uterine elevation Bartholin gland secretion Orgasmic contractions
Lost	Flush Scrotal swelling Re-erection Ejaculatory inevitability Prostate contractions	Breast engorgement Flush Swelling of labia majora
Other	Refractory period >24 h	

Table 8.5.8.2 Medical factors affecting sexual function

Drugs reducing sexual drive	Drugs reducing testosterone	Drugs blocking physical arousal
Dopamine antagonists	Digoxin	Thiazide diuretics
Major tranquillizers	Cimetrdine	Some β-blockers
Metaclopamide	Cyproterone	
5-HT_2 agonists–SSRIs except nefazodone, trazodone, fluvoxamine	Finasteride	
Benzodiazepines	Oestrogens Progesterone	

Treatment of sexual problems in old age

The treatment of sexual dysfunction is founded on a comprehensive understanding of the problem. It begins with listening. Perhaps this is a statement of the obvious—except that the emotional power of sexuality makes it hard for patients and their partners to speak about sex, and for others to listen properly. Sexual histories are rarely taken as a routine part of the assessment of older people,[2] and even when patients disclose a sexual problem, their doctor may shrink from embarking on an exploration of the difficulty. Yet there is good evidence that opening up communication about a sexual problem—not only between clinician and patient but also (helped by the clinician) between sexual partners—forms a large part of successful treatment, and sometimes may be all that is needed. Even patients attending a specialized clinic may be satisfied by receiving assessment and information, without necessarily wanting active treatment.[13]

Sexual problems come to the notice of many different medical and surgical specialties (gynaecology, urology, genitourinary medicine, diabetology, endocrinology) who have expertise in the physical treatments now available, and sometimes also in the psychological and relationship components of the dysfunctions that they treat. In other cases the psychiatrist may be helping the couple to work with a physical treatment that one of them is receiving, and to make the most of the improvements in sexual function that it offers.

Behavioural treatment for sexual dysfunction, first described by Hunter,[16] was developed by Masters and Johnson.[17] Their *Sensate focus exercise* (see Chapter 4.11.2) is a simple behavioural technique that can be very effective in helping couples in whom sexual intercourse has become (for whatever reason) difficult, painful, or disappointing, and who have then retreated from all pleasurable physical contact with each other. In this technique attention is removed from intercourse (in fact intercourse is forbidden), and instead the focus is put on renewing the partners' pleasure in mutual touch and caressing for its own sake.

Table 8.5.8.3 Surgical procedures affecting sexual function

Transurethral prostatectomy leads to retrograde ejaculation
Pararectal surgery damages nervi erigentes
Indwelling catheters and pessaries
Mutilation affecting body image
Surgery to genitalia

Table 8.5.8.4 Diseases affecting sexual function

Diabetes
Myxoedema, pituitary tumours
Neurofibromatosis, paralysis, myotonia dystrophica, autonomic neuropathy
Peyronie's disease
Malignancy and infections of genitalia and prostate, vaginal fistulas
Liver failure leading to higher oestrogens
Arthritis
Hypertension, vascular diseases, myocardial infarction
Respiratory distress
Depression, schizophrenia, cortical dementias

There is little scope for pharmacological treatment of most sexual difficulties arising in old age, other than erectile dysfunction.[13,14] Hormonal treatments are no longer thought to be helpful except where deficiencies have been clearly demonstrated. The commonest hormonal treatment in old age is probably the use of oestrogen (topically) in dyspareunia. Treatment of hypersexuality and sexual aggression in the context of dementia is difficult: pharmacological methods have been reviewed by Series and Degano.[18]

Conclusion

Ageing brings increasing diversity. This certainly applies to sexual behaviour. Those who work in the field of old age psychiatry can help their patients by understanding this diversity, making it safe and acceptable for patients to talk about whatever sexual concerns they have, and helping them in the acquisition of all the information they need. Sex in old age is not the frightening imperative of the teenager, but it can still contribute greatly to the quality of life.

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8.6

Special features of psychiatric treatment for the elderly

Catherine Oppenheimer

Introduction

Three themes underlie the topics in this chapter.

Old age is a time of multiple problems

Physical, psychological, and social problems often occur together, linked by chance or causality in the life of the old person. Very rarely can one problem be dealt with in isolation, and many different sources of expertise may be engaged with a single individual. Therefore good coordination between different agents is essential in old age psychiatry, both for the individual patient and in the overall planning of services.

Clear boundaries between 'normality' and 'disease' are rare in old age

Many of the pathologies characteristic of old age are gradual in onset and degenerative in nature, and more due to failures in processes of repair than to an 'external foe', so the distinction between disease and health is often quantitative rather than qualitative. 'Normality' becomes a social construct with fluid borderlines, containing the overlapping (but not identical) concepts of 'statistically common' and 'functionally intact'. Thus the popular perception of normal old age includes the 'statistically common' facts of dependence and failing function, whereas 'intactness' (excellent health and vigorous social participation) is seen as remarkable rather than the norm. But the boundaries of 'old age' are also socially constructed—in developed countries good health at the age of 65 would nowadays be regarded as a normal middle-aged experience, whereas superb health at 95 would still be something noteworthy.

Since some degree of physical dependence, forgetfulness, and vulnerability to social exclusion is expected in old age, meeting those needs is also regarded as a 'normal' demand on families and community agencies such as social services, rather than the responsibility of health care providers. As the severity of the needs increases, however, so also does the perceived role of health professionals, both as direct service providers and in support of other agencies.

Lack of competence is common in old age

Because of the high prevalence of cognitive impairment in old age (especially among the 'older old'), questions frequently arise as to the competence of patients to make decisions. Older people who cannot manage decisions alone may come to depend increasingly on others for help; or, resisting dependence, they become vulnerable through neglect of themselves or through the injudicious decisions they make. When an incompetent person is cared for by a spouse or family member, the danger of self-neglect or of ill-considered decisions is lessened, but instead, there are the risks of faulty decisions by the caregiver (whether through ignorance or malice), and also risks to the health of the caregiver from the burden of dependence by the incompetent person. Legal mechanisms, differing from one country to another, exist to safeguard the interests of incompetent people.

These three themes will be developed further, and with them the following special topics:

1 multiple problems: including sleep disorders in old age, medication in old age psychiatry, and psychological treatments in old age psychiatry;

2 blurred boundaries of normality: including the role of specialist services and support between agencies;

3 incapacity and dependence: including balancing the needs of patients and caregivers, abuse of older people, ethical issues, and medico-legal arrangements for safeguarding decisions.

Multiple problems

Sleep disorders in old age

Useful reviews of this topic may be found in Anconi-Israel and Ayalon,[1] Sivertsen and Nordhus,[2] and Mosimann and Boeve.[3] More detailed general discussion of sleep and its disorders will be found in Chapters 4.14.1–4.14.4 of this textbook.

(a) Normal changes with age

With age, the architecture of sleep changes—in fact, most of the change occurs before the age of 60. Sleep is divided into shorter periods interspersed with wakefulness or brief arousals, there is a decrease in total sleep time and in sleep efficiency (the ratio of time asleep to time in bed), and there is less stage 4 (deep) and more stage 1 and 2 (shallow) sleep, without an increase in the proportion of rapid-eye-movement (REM) sleep. This change in sleep architecture is conventionally associated with changes in circadian rhythms with age, such as decreased amplitude and phase length of these rhythms (but see Monk[4] for a critical review).

Many people adapt to these changes, but others find the altered pattern distressing. Thus the borderline between normal and problematic sleep is blurred, because subjective assessments of sleep quality are not necessarily matched by objective measures (such as polysomnography); consequently the definition of 'insomnia' hinges not only on features of night-time sleep, but on impaired functioning in the daytime.

(b) Comorbidity

The majority of healthy older people have no complaints about their sleep, but there is a strong association between poor sleep and other health problems, and sleep problems make a material contribution to the impaired quality of life suffered by people with comorbid illness. In a study of patients in primary care,[5] a positive answer to even one question about sleep ('do you feel excessively sleepy during the day?') predicted the quality of life related to physical or mental health problems. Attention to improving sleep in these patients can improve their well-being, but too often the sleep problem is missed in the general assessment of the patient. Impaired sleep can have serious consequences: it is associated with symptoms of anxiety and depression, an increased risk of falls, and diminished memory and cognitive functioning.[1]

(c) Causes of disordered sleep

These include the following:

1 **Environmental causes**: e.g. a strange bed, noise, cold or heat, or loss of a familiar bed companion (e.g. through bereavement).

2 **Physical causes**: sleep can be broken by pain, stiffness (e.g. Parkinson's disease or arthritis), limb movement (restless legs syndrome, periodic movements of sleep), breathlessness (cardiac failure or sleep apnoea), the need to urinate (prostatic disease or urinary tract infection), eating too close to bedtime, or dehydration (e.g. voluntary restriction of fluids to prevent nocturia).

3 **Medication**: alcohol, especially if taken to relieve anxiety or to assist sleep (since the rapid metabolism of alcohol leads to rebound anxiety and wakefulness), and antidepressants such as selective serotonin reuptake inhibitors (**SSRIs**) can cause wakefulness or nightmares. Information on the numerous other medications which may impair sleep can be found in Anconi-Israel and Ayalon.[1]

4 **Psychological causes**: for example, anxiety, depression, hypomania, and paranoid illness. Often a sleep problem is triggered initially by a physical cause, but is then maintained by the patient's anxiety about wakefulness.

5 **Sleep in dementia**: changes in sleep rhythm in dementia are similar to those of normal old age, but often more severe: daytime drowsiness or napping, difficulty in falling asleep at night, decreases in slow wave sleep and in rapid-eye-movement sleep. However, another common cause of sleep impairment is the use of benzodiazepines or major tranquillizers to treat behaviour disturbances: the patient may end up drugged in the day and wakeful at night. A partial remedy may be to create an overriding diurnal rhythm (e.g. by attendance at day care or a programme of physical activity in the day), together with the minimal use of medication at night. Patients in institutional care are particularly likely to be deprived of the normal cues for circadian rhythms: quiet and darkness at night, bright daylight in the morning, and physical activity in the day. On the other hand sleep time may be strikingly increased in dementia, especially in vascular dementia where it may be part of the apathy that is common in that disease.

(d) The parasomnias

The parasomnias that are common in old age are obstructive sleep apnoea (or sleep-disordered breathing); restless legs syndrome and periodic limb movements in sleep; and REM sleep behaviour disorder (RBD). They may range in severity from troublesome to severely disabling, and accurate recognition is important for all of them, because of the consequences both to the patients and to their bed partners if their diagnosis and treatment are missed. Further details can be found in the sources mentioned above,[1,3] but RBD warrants some further discussion here. This disorder is defined as an 'intermittent loss of the muscle atonia normally present during REM sleep, and episodes of elaborate motor activity associated with dream mentation'.[6] Typically, in the early hours the patient (usually male) shouts, thrashes around, and may attack his bed partner, without waking, and without any recollection of the episode when he does wake later. The importance of this condition lies in the fact that it is very distressing, and possibly dangerous, for the bed partner; it can often be treated effectively (with clonazepam); and it is strongly associated with the development (sometimes after a very long latent period, of a decade or more) of neurodegenerative disease—especially Lewy body dementia, Parkinson's disease, or multisystem atrophy—the alpha-synucleopathies.[6,7]

(e) Management of sleep disorders

(i) Psychological methods

These are now the methods of choice with insomnia in older adults.[2,8] Trials comparing psychological with pharmacological treatment, and with a combination of the two, show equivalent effects in the short-term, but a longer-term advantage to the psychological methods. More importantly, it has also been shown that in secondary insomnia ('insomnia occurring when a psychiatric condition, a medical condition, a non-insomnia sleep disorder, or a medication appears to precipitate and then appears to maintain insomnia') it is not necessary to wait until the primary condition has been resolved: the insomnia can usually be effectively treated in its own right, and even if it is not completely cured, valuable improvements can be achieved.[8] Likewise, psychological methods can be used to help in withdrawing medication in hypnotic dependent insomnia.[9]

The methods used have generally been 'multicomponent behavioural treatments'. The components include:

- Relaxation training
- Stimulus control (requiring the patient to leave the bedroom if they are not sleeping)
- Sleep restriction and sleep compression (using a fixed time for getting up, progressively shortening the time in bed until it matches the time asleep)
- Cognitive restructuring (modifying the pre-sleep thinking patterns, which in insomnia usually include negative thoughts about the effects of sleep loss, and the use of worry and self-blame as an attempted strategy for controlling thoughts[10])
- Sleep hygiene education (advice on the effects of tea, coffee, exercise, etc on sleep)

Such treatment packages need not be dependent on sources of specialist expertise: Sivertsen and Nordhus[2] discuss the feasibility of treating insomnia psychologically within primary care.

(ii) Treatment of sleep problems in dementia

When cognitive impairment makes psychological treatment difficult, pharmacological treatment may be necessary. Benzodiazepines (probably temazepam for preference) must be used very cautiously; they are dangerous in ambulant patients, though less so for a patient who is no longer mobile. Sedating antidepressants (e.g. trazodone) can be used instead. An atypical antipsychotic may be appropriate if there is severe anxiety and suspiciousness (sometimes of delusional intensity) of the carer at night.

The sleep problems of the carer of a patient with dementia also need to be taken very seriously. The carer may benefit from the psychological measures outlined above, and can institute some of the measures (such as sleep hygiene) on behalf of the patient. Insomnia in a caregiver, caused by the wakefulness of the person cared for, can lead to rapid breakdown of the support system. If the patient's sleep problem cannot be resolved then it is essential to give the carer the opportunity for uninterrupted sleep at times, through arranging residential respite care, a night-sitter, or some other form of relief.

The use of medication in old age psychiatry

Specific uses of medication for the various psychiatric disorders occurring in old age are dealt with in the relevant chapters. Here, three general principles will be discussed:

- medication as an experimental trial
- stopping medication
- compliance and concordance.

(a) Medication as an experimental trial

Starting medication for any condition ought to be treated as the test of a hypothesis. There should be a plan, shared with the patient, setting out the following:

1 how long the trial will last before a decision is made that the treatment is unsuccessful and should be ended;

2 which target symptoms will be monitored, and what records should be kept (by the patient or caregiver);

3 when progress will be reviewed;

4 what side-effects might be developing and which of these should alert the patient to stopping the drug and contacting the doctor;

5 what will follow if the trial succeeds;

6 what will be tried instead if the trial fails.

This watchful approach to medication is particularly important for people with dementia. Often the patient's confusion, lack of insight, and communication difficulties mean that a tentative diagnosis has to be based on scanty information. For example, disturbed behaviour in a patient in a nursing home may be due to depression, but the patient cannot describe the depressive symptoms. Clues to the diagnosis may come from the care staff (e.g. 'She never jokes with us now'), and an empirical trial of an antidepressant may result in a resolution of the symptoms. However, the trial must be set up carefully and medication not thoughtlessly continued for months without review, merely because no one has asked whether it is helpful or not.

(b) Stopping medication

The decision to withdraw medication can be as valuable and informative as the decision to start it, and documentation of the reasons for the decision is equally important (though often neglected). In delirium, medication may be contributing to the problem, particularly where a cocktail of medications has been built up over time—improvement after withdrawal of a drug gives valuable guidance for a future episode. Also, patients reaching the terminal stages of dementia should have their medication gradually decreased, to test whether it is still needed: drugs prescribed at an earlier stage to control behavioural syndromes are rarely, if ever, required for the entire course of the disease.

However, there are also reasons for being cautious about stopping medication. Some drugs have important withdrawal effects (e.g. paroxetine and benzodiazepines). Medication that has been used prophylactically may seem to be unnecessary while the patient is well, but when the drug is withdrawn the need for it is revealed. For example, a patient can remain well for years on an antidepressant following a severe depressive episode, or on a low dose of antipsychotic after a schizophrenic illness—until well-intentioned withdrawal of the drug, however carefully monitored, precipitates the return of the illness.

(c) Compliance and concordance

The word 'compliance' appears to imply that patients must be obedient in following instructions; but nowadays patients are seen more as partners in their own treatment, for which the word 'concordance' (when the partnership is successful) is more apt. The same principle of partnership in treatment decisions applies to older people, wherever possible. However, in addition to willingness to participate there needs to be the ability to do so, which in older people is often impaired by physical causes (such as arthritic hands which cannot open child-proof packaging, or poor vision which misreads instructions), and by psychological causes, especially memory loss and temporal disorientation. Therefore patients will often need help in maintaining their concordance with treatment, through suitable packaging and memory aids (such as calendar boxes), or through supervision or administration of the tablets by others. Families may take a long time to notice that their parent or grandparent, who has taken medication reliably for years, has started to miss or duplicate doses.

It is important to understand the feelings of a caregiver who takes responsibility for the medication prescribed for a patient with dementia, especially where it is being used to control behaviour. Anything prescribed on an 'as-needed' basis should be very carefully explained. The caregiver may be afraid of provoking the difficult behaviour by offering the medication, or conversely of overdosing her relative into stupor, or she may feel guilty about meeting her *own* needs by giving drugs to another. Such feelings make it hard for a caregiver to judge objectively when discretionary medication should be used.

Psychological treatments in old age

Many older people prefer a 'talking treatment' to medication, and there is increasing evidence of the success of psychological interventions with cognitively intact older people.[11] Brief focused interventions are particularly suitable.

However, even mild cognitive impairment, too slight to amount to dementia but enough to interfere with grasp and retention, may hamper psychological treatment. For example, such a patient being treated psychologically for anxiety may try hard to cooperate with her therapist, only to be made more anxious as she fails to remember the instructions, or to put them into practice, and so the sense of failure she feels elsewhere in her life is reinforced.

The principles of supportive psychotherapy and problem-solving are always relevant, both on their own or in conjunction with medication: providing 'unconditional positive regard' for the patient; accepting a degree of dependence by the patient, while limiting its consequences; setting appropriate expectations so that the patient is not unnecessarily exposed to a sense of failure; openly facing realities that cannot be altered (such as loss, disability, and death); helping to think through practical problems as they arise.

Family and systems therapy[12] has a particular role in old age psychiatry, because it explicitly recognizes the patient in his or her social context. Although family therapy requires special training, the principles of a family approach can be adopted by all those working with older people.

When patients are cognitively impaired, psychological approaches generally focus more on the interaction between the patient and the caregivers, who can be helped to understand the patient and the reasons for her behaviour, as well as their own actions and the reciprocal effects that these have on her. For example, reminiscence therapy helps professionals to respond to patients as 'whole people' with individual lives, as well as helping patients to reconnect with their former identities, and to recover some of the confidence they took for granted in the past.

Therapies based on music, dance, drama, art, and sensory stimulation are also important in old age.[13] They can help patients, individually or in groups, to express feelings and thoughts, bypassing the impairments of verbal skills which come with dementia.

The blurred boundaries of 'normality'

We consider two examples of the way in which 'normality' is not clearly defined in old age.

First, social expectations allow for increasing dependency in the old, and many people have personalities better suited for protective relationships than for solitude and independence. People who have struggled with anxiety and loneliness in middle age can experience the onset of physical dependence as a kind of relief: now they can allow themselves to be looked after. What was a dysfunctional personality structure in earlier life becomes adaptive in old age. In contrast, the person who has always jealously guarded his or her personal boundaries will find the adjustment to disability very hard. Determined refusal to accept necessary help converts a 'normal' preference for independence into a problem for others.

The second example concerns the idea of death. For a younger person to say that they were waiting for death would prompt a psychiatrist to look for evidence of a depressive illness. In old age this way of thinking may be found in someone with no disorder of mood, with the ability to enjoy life, and with a rational appreciation of life's inevitable limits. But even in old age, to see nothing valuable in one's future might be a sign of depression.

The role of specialist services and cooperation between agencies

This topic is dealt with more fully in the succeeding chapter (see Chapter 8.7).

The difficulty in separating the normal from the pathological, and in deciding when specialist intervention is required, is reflected at a structural level in shaping the responsibilities of the different bodies involved in the welfare of older people. Help required by older people can range from the simplest of neighbourly assistance to the full resources of specialist hospital care. As people live longer, despite some morbidity, the demand for care increases. The response to this demand is shaped by many different factors: market forces, public concern, private enterprise, voluntary or charitable groups which spring up to fill perceived gaps, and governments acting to remedy abuse or to limit demand on state-funded services. The divisions of responsibility are arbitrary, and people fall between services as often as within them. Systems change. For example, in the United Kingdom older people with permanent disability (especially dementia) used to be cared for in state-funded hospitals, but this care has now become the responsibility of independent-sector nursing homes, with means-tested financial support from the social security system.

The scope of specialist services for older people extends beyond the treatment of established illness. They have a role in the prevention and early detection of illness, in supporting and educating other service providers and in collaborating with other agencies in strategic planning. This span of interest can be represented as a pyramid (Fig. 8.6.1). The peak of the pyramid represents those requiring the scarcest and most intensive specialist services, and the base represents the population at large. In the middle are the people who need day care, support at home, social support, and help in primary care.

Between a population base of, say 20 000 older people and an inpatient provision of, say 10 beds lies the range of people to whom psychiatric services can offer significant help without assuming total responsibility for care. It is essential that the different agencies interact effectively and overlap their care, rather than leaving patients to negotiate their way from one island of provision to another.

Key components of such a 'mixed economy of care' should include:

1 A system for prompt and accurate assessment of people's needs which leads to offers of appropriate help, including reassessment. Assessment for specialist psychiatric care should be both 'generic' (using the skills that all disciplines share) and specific (using the particular skills of medicine, nursing, occupational therapy, etc.).

2 A clear understanding by staff working in the various agencies of what their tasks will be, and good training that will allow them to carry these out with confidence and satisfaction. When non-specialist care breaks down, it is usually because the carer do not expect to have to deal with the problem which faces them.

3 Systems ensuring that people are not obliged to make frequent and abrupt transfers between one setting and another as their disabilities increase. Assessment must therefore include prediction of future need; and each provider should offer a degree of

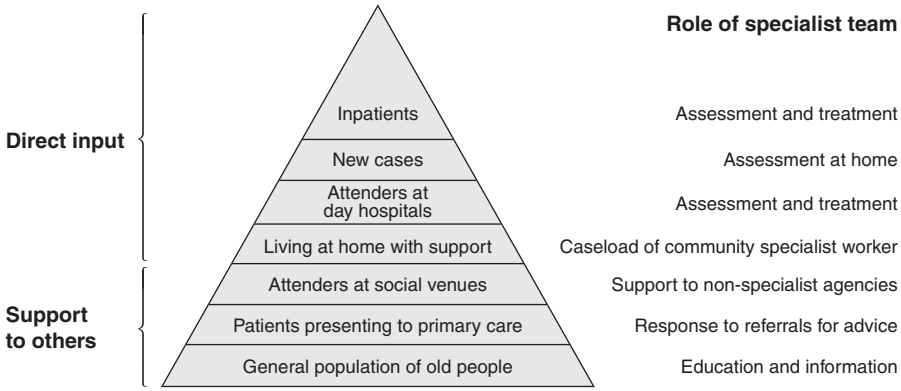

Fig. 8.6.1 The span of old age psychiatry services.

flexibility, to encompass people who will soon need what they provide, and others whose needs are greater than the norm. For some people, a change of setting as their condition progresses—for example, from a sheltered flat to care in a nursing home—will be inevitable, so care systems must have ways of making transitions as smooth as possible.

Loss of capacity and dependence on help

Mutual helpfulness is an accepted part of marital and family relationships. Family members often adapt unquestioningly to an older person's increasing dependence until the emotional pressures, or the adjustments they have to make in their day to day lives, become severe.

The 'needs of caregivers' are part of the currency of discussion amongst service providers. But people in everyday life do not necessarily perceive themselves that way: they think of themselves as simply participating in a normal aspect of family life. Younger people who have duties to their work, partner, and children—duties which conflict with the needs of their parents—may be readier to seek professional help than are the ageing spouses of a failing partner. Spouses often view caring as an intrinsic part of their lifelong relationship, and may resent offers from outside as an intrusion upon their privacy.

On the other hand, relationships can have malign as well as protective aspects, and sometimes the dependent partner in a caring relationship suffers more disadvantages than benefits. In extreme cases, abuse can occur.

Abuse of older people

This is much better understood now than a few decades ago, although systematic study is difficult because of the varieties of abuse or exploitation that arise, the ambivalent relationships that surround them, and the concealment often practised by abusers and victims alike.[14] Abuse can occur in any circumstances, any class, and any relationship, from blood ties and friendships to professional and commercial relations. Nevertheless pointers to risk have been identified (Table 8.6.1).

Typically, the victim is disabled, often but not always with a dementing illness, perhaps with impaired communication, and is unrewarding to look after. Typically, the abuser is also impaired in some way, isolated from support (often by refusal rather than the absence of offers of help), and may abuse alcohol. There may have been a long history of ambivalence or of mutual aggression between abuser and victim, or the victim's illness may have reversed a power relationship that previously operated (damagingly) in the opposite direction. The intention of the abuser is sometimes clearly to do harm. In other cases the abusive behaviour seems to be an impulsive response to emotional pressures which the abusers are poorly equipped to deal with because they do not understand them properly, do not know how to share the burden with others, or are overwhelmed by the patient's need for care. The abuse may range from a single assault by a caregiver, who is immediately horrified by what he or she has done, to systematic cold-blooded persecution. Abuse may be physical, emotional, psychological, sexual, or financial.[15]

Where harm to a vulnerable person is suspected, there is an obligation to report it to the appropriate authority—in the United Kingdom, the local social services. Sometimes they in turn will involve the police and a criminal prosecution may follow. But frequently the problem can be dealt with in other ways, such as arranging a different form of care for the vulnerable person. The emphasis will be on reducing and managing risk, and on enhancing

Table 8.6.1 Some risk factors for abuse in old age

Victim	Abuser	Relationship
Female	Family member in caregiver role	Previous relationship – not close, ambivalent, mutually abusive
Aged over 75	Psychiatric history	Role reversal
Physically dependent	History of abuse as a child	Power reversal
Cognitively impaired	Substance misuse	Lack of problem-solving skills in the relationship
Socially isolated (lacking external support)	Financial dependence on victim	Forced proximity
Sensory impairment	Dependence for housing on victim	Mutual dependence
Incontinence	Overburdened by caregiver role	
Abusive or unrewarding to caregiver	Unsupported, or rejecting support	
Ready to adopt sick role		

Reproduced from Fisk, J. Abuse of the elderly. In *Psychiatry in the elderly* (2nd edn.) (eds. R. Jacoby and C. Oppenheimer), copyright 1997, with permission from Oxford University Press.

the quality of life of the person concerned. Difficult ethical issues arise when an elderly couple both suffer from dementia and one of them (often the husband) insists on continuing to care alone for his wife, unaware of the loss of his competence to do so. For a time, professional staff will try to support both partners in their wish to remain together at home, and to mitigate the effects of the inadequate care by the husband. But at some point the professional duty may need to shift from supporting, to taking over legal responsibility for the neglected person—though the displacement of the caregiving spouse in this way is distressing for everyone concerned.

Ethical problems in old age psychiatry

The same classical principles of bioethics (beneficence, non-maleficence, autonomy, and confidentiality)[16] apply in old age psychiatry as in other age groups, but with differences in emphasis.[17] Since older people can rarely be considered in isolation, ethical principles have to be applied with the whole system in mind, and professionals often have ethical obligations to more than one person at a time.

(a) Values

In old age, as in youth, we should seek to produce benefit and not harm—but identifying which of these is which may be more difficult in old age. Death is usually thought of as harm, but sometimes may be regarded as a blessing. Therefore it is essential to understand what the patient, rather than the professional, sees as beneficent, and the patient's right to name the value that they set on something must be respected. Some older people would prefer their assets to go to their descendants rather than being spent on their own care at the end of their lives—but the state sets limits to such self-denial.

(b) Ambivalence

Impairments of thinking and communication can complicate ethically observant professional practice. People of all ages may have ambivalent feelings, or say things which seem belied by their actions, but this is particularly common among older people with cognitive impairment. Perhaps memory failure causes decisions to be swayed by feelings of the moment, so that decisions are not consistently maintained; and cognitive difficulty in marshalling complex information means that weighing alternative possible outcomes to a decision is much harder.

An illustration may make this clearer.

Case study: A woman with early cognitive impairment lives alone in the house where she brought up her children. She loves her house and garden, and tells her daughters that she never wants to live anywhere else, and she is convinced that she needs no help from them. At night, however, she becomes anxious and confused, and telephones her daughters, asking them why they 'haven't come home yet' and begging them not to abandon her.

Does the night or the day reveal her 'true wishes' better? Which should guide the decisions of her family, and of the professionals whose help they seek? Possibly, her considered daytime thoughts and her anxious actions at night are tapping different areas of her experience—the daytime communication reflects her aspirations and her lifetime self-perception, while her telephoning at night reflects the immediacy of her feelings and needs. Our duty is to give weight to both kinds of communication, helping the patient herself to understand what they mean, and to offer her the real-life opportunity (rather than theoretical discussion) of testing out the options she needs to consider.

(c) Giving information and safeguarding information

Ethical obligations here include truth telling, giving information to patients about themselves, and protecting patients' information from others.

'Loss of insight' is a feature of dementia, but good insight is also dependent on sound information. A person who is making significant errors, or beginning to fail in self-care, should not be left in ignorance of what is happening to them. In fact, public knowledge nowadays about Alzheimer's disease is so much greater that sufferers often recognize the early signs themselves. They are entitled to an open discussion with their doctors, in which full information is put before them. (Relatives often shy away from such open disclosure, although when asked in surveys what they would wish for themselves, they tend to say that they would rather be told).

When it comes to disclosing information about a patient to the people involved in his or her care, the arguments are different. It is generally accepted that better care must depend on the best information, and it is normal for information to be shared between members of a clinical team, where the members share also in the duty of confidentiality to their patient. But in old age it is harder to know where to set these boundaries—both as regards information that should be protected, and as regards recognition of who is a team member. For example, senior staff in some residential homes, strictly preserving confidentiality, may not share information about the residents with the untrained care staff. However, if they know nothing about a resident's former life, caregivers will tend to respond to her as 'a bundle of needs' rather than as a real person; and if she cannot tell her own story, others must do it for her. This is much better recognized now than it used to be, and homes may ask families to construct a diary or album of a resident's life, with photographs, mementos, and recollections by different relatives. The diary also acts as a memory prompt and trigger for enjoyable conversations between resident and caregiver, and it creates a domain of shared information about individuals within the institution.

(d) Autonomy

Healthy people strongly value the freedom to make their own decisions, to pursue their own aims, and to determine the course of their own lives. The onset of a physical illness may constrain this freedom, but people should still have as much influence as possible over decisions about their illness and its consequences.

Psychiatric disorder is different, because it may affect the powers by which that freedom is exercised, and may lead to decisions which would never have been made in health. From this comes the need, universally recognized although taking different legal forms in different countries, to set external controls over the decisions of people when they are mentally ill. Such legal controls are typically based on acute functional illness as the paradigm case. This gives them an 'all or nothing' character, envisaging hospital treatment of an illness capable of being relieved, so allowing patients to resume their autonomy when they recover their health.

This legal model is not really suited to cognitive impairment and its effect on decision-making in old age. The illness will not get better,

and patients are unlikely to give (as they might with an acute illness) later 'retrospective informed consent' to the treatment. The emphasis therefore has to be much more on **minimal necessary interference**—on setting up protective frameworks, in which as much autonomy as possible can be exercised; on supporting patients at home or in homely settings, rather than bringing them into a hospital where no effective treatment can be given; and on gathering the information (both from patients and from those who know them best) which will enable professional decisions to reflect the wishes that the patient would have expressed, had they been able to do so.

Looking after cognitively impaired patients requires us to try constantly to maximize the opportunity for autonomous decisions, while also being very clear when a patient lacks the capacity to engage with a more complex issue. At such times, the responsibility for the decision must be openly and seriously taken by others. Trying to circumvent the problem by concealment and persuasion is a greater affront to autonomy than is an honest explanation to the patient of the reasons why a decision has been taken out of his hands.

Medico-legal issues

'Doctors and lawyers have common responsibilities to ensure the protection of people who are incapable of deciding matters for themselves, and to promote the choice of those who can and should regulate their own lives. The careful assessment of whether individuals have or lack capacity is essential to protect their rights'.[18]

The legal framework in England and Wales for both financial and welfare decision-making where capacity is in doubt has been transformed by the Mental Capacity Act 2005, which came into force in April 2007. Some general principles of the Act are worth discussing here: more detail can be found in Lush.[19]

'Capacity' is a legal rather than a medical concept (see Chapter 11.1). Every adult is presumed to have full capacity, and a loss of capacity must be proved in relation to the particular decision being made: capacity is 'decision-specific'. For example, a person may *have* the capacity to choose another person to act for her in the management of her affairs, while *lacking* the capacity to manage those affairs herself. Although the final decision on capacity is made by the courts, they look to doctors to advise on whether a mental disorder has affected the individual's ability to make a particular decision or to carry out a specific task.

(a) Assessing capacity

The Act states that a person lacks capacity if ' . . . he is unable to make a decision for himself . . . because of an impairment of, or disturbance in, the functioning of the mind or brain'. Such a person lacks capacity in relation to a particular decision if he cannot:

- understand the information about the decision
- retain the information
- use or weigh the information in the process of making the decision
- communicate the decision

The information that he must understand should include the consequences of deciding one way or another, and of making no decision. The information must be retained only for long enough to allow a decision to be made, therefore memory loss does not automatically remove capacity. The focus of this test of capacity is on the *process* of decision-making: the Act explicitly states that 'a person is not to be treated as unable to make a decision merely because he makes an unwise decision'.

(b) Acting on behalf of a person who lacks capacity

Anyone acting on behalf of an incapacitated person must do so in the 'best interests' of that person. The action or decision in question should be delayed if there is a chance that the person may regain capacity; every effort should be made to enable them to participate in the decision-making process; the decision-maker should take into account the person's past and present wishes, beliefs, values, and feelings, especially any that had been written down when they had the capacity; the views of families, carers and anyone else interested in their welfare should be sought; and the least restrictive method for achieving the intended purpose should be chosen. The obligation to act in a person's best interests, and the protection from legal liability if they do so without negligence, extends to anyone carrying out acts of treatment and care, such as physical assistance, doing shopping, giving medical treatment, or nursing care.

(c) Lasting power of attorney

A person with capacity can choose to appoint someone to act on their behalf (their 'attorney'). The power to act continues to be valid even after the 'donor' loses capacity, provided that the document giving the power has been registered with the Public Guardian (a newly created statutory office, replacing the former Court of Protection). The donor can give authority to his attorney to take both financial decisions *and* decisions relating to his health and welfare, provided that he is shown to lack the capacity to make those decisions himself at the relevant time. The attorney has the duty to act in accordance with the principles of the Mental Capacity Act 2005 and the guidance of its Code of Practice, and must act in the best interests of the donor.

(d) Advance decisions to refuse treatment

The Mental Capacity Act 2005 also provides a framework and safeguards for a person who wishes to decide in advance what treatments should be withheld if they lose capacity in future. Advance decisions to refuse life-sustaining treatment have to be explicit, in writing, signed, and witnessed.

(e) Driving

A common problem concerns the ability of older patients with early cognitive impairment to drive safely.[20] Scores on simple cognitive tests (e.g. the Mini-Mental State Examination) are very poorly correlated with driving ability, except where there is severe and obvious impairment. No quick objective test of driving skills has yet been devised. A worried but competent patient can often be reassured by booking an hour with a driving instructor. Loss of the freedom to drive represents such a loss of independence and enjoyment, and such a blow to self-esteem, that advice to give up driving may be strenuously resisted. On the other hand, families and professionals are conscious of potential risks to the public even if the patient denies these risks. Legally, the position in England and Wales is straightforward: individuals have a duty to notify the Driving and Vehicle Licensing Authority (**DVLA**) if they have an illness which might impair their ability to drive. A doctor must tell the patient who has such an illness that they are under an

obligation to inform the DVLA; if it seems that this advice has been ignored, the doctor has an obligation to inform the DVLA himself—the duty of confidentiality is overruled by that requirement. Thereafter, the DVLA will arrange for the patient to receive an independent medical examination, and it is the DVLA which decides whether the driving licence should be withdrawn. If the patient fails to attend the medical examination (whether through forgetfulness or lack of insight), the licence is automatically withdrawn.

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8.7

The planning and organization of services for older adults

Pamela S. Melding

Introduction

When does an individual become an older adult? When they show signs of ageing? When they retire from work? When their health becomes frail? When they feel old? When society says they are old? Any of these indicators could define an 'older person' anywhere between 40 and 90 plus years! However, it was for statistical simplicity that many jurisdictions chose the chronological age of 65 to mark the change in status from mature adulthood to 'older person', mainly to establish an age for expected retirement and entitlement to certain benefits, including access to geriatric health services. When this arbitrary discriminator was instituted in the mid-twentieth century, 70 years was a good lifespan for most people. However, over the past 50 years, life expectancy steadily increased and is currently advancing at 6 weeks per annum, boosting the overall number of adults over 65 years and, particularly the over 80 years cohort.[1] Increasing life expectancy, due to improved health care and lowering birth rates, is causing worldwide 'population ageing'. This phenomenon will affect all health and mental health services in future years.

Already, health care resource and cost implications of population ageing for health services are enormous. Older adults occupy about two-thirds of general hospital medical, surgical, and orthopaedic beds; they are the greatest users of primary care and prescription medicines. Internationally, late-life illness takes up a considerable proportion of government or insurance funded health care budgets. As an example of the enormous costs involved, in 2003/2004, the United Kingdom's NHS spent around 43 per cent (£16.471 billion) of its hospital and community health services budget on people over the age of 65, and the cost of community and residential care for older people was 44 per cent (£7.38 billion) of all social welfare budgets.[2] These figures will rise dramatically over the next decades.

Mental Health Services have been slow to anticipate that population ageing will also increase the need for psychiatric services for older people. In many areas of the world, services are scarce, sporadic, or sub-standard. Even in developed nations, there is considerable variation in availability from one area to another. In the past decade, practically all OECD countries have promoted policies of de-institutionalization and community-based care for the elderly, in response to rising cost pressures associated with population ageing, plus a requirement to improve satisfaction for increasingly knowledgeable and assertive consumers, by providing better quality in all health services for older adults, including mental health.[3]

The need for services for older adults

Epidemiology

Whilst most people aspire to longevity, it can be a mixed blessing. Living longer increases risk of developing chronic degenerative diseases of body and brain, which can precipitate mental illness, possibly for the first time in life. Psychiatry services for older adults see a full range of new and chronic psychiatric disorders. However, the commonest new threats to mental health in late life are affective disorders and dementia.[4] Healthy, community-dwelling older adults are generally resilient, with a prevalence of major depressive illness of about 3 per cent, but studies of people in residential care find significant depressive symptoms in 14–42 per cent,[5] and in populations over 80 years, about 40 per cent.[6] Depression occurs in 15 to 40 per cent of medical inpatients[7] and in approximately 35.9 per cent of patients in geriatric rehabilitation units.[8] Among the many aetiological contributors to mental health problems in late life, physical illness and poor health are major risk factors, particularly for depression,[9] the risk increasing with disability.[10] Co-morbid physical disorders can mask depression or impede management with psychotropic medications. Although, pure anxiety disorders reduce in late life to about 1 per cent in community-dwelling older adults, anxiety co-morbid with other psychiatric disorders, particularly depression, is more common at approximately 4 per cent. Older people with anxiety disorders are high users of health care resources and may initially present with physical symptoms.[11] Affective disorders are often multi-dimensional, their treatment complicated, and frequently, they need joint management with geriatric colleagues.

Dementia affects 5–7 per cent of people at 65 and 20 per cent of those over age 80 years. By 2040, the number of people with the disorder will double in the developed countries of Europe and North America.[12] Older adults with dementia are an exemplar of consumers who require multi-disciplinary management. While geriatricians manage the majority of patients, those who exhibit behavioural and psychological symptoms of dementia (BPSD), approximately one-third, do best with additional specialist psychiatric

expertise and management.[13] People with dementia can live for many years as their disorder progresses, requiring increasing levels of support from family, social workers, nurses, residential care providers, and other community health care workers in addition to psychiatry services.

Why are specialist services required?

As indicated above, mentally or physically frail older adults have complex needs and frequently require a broad, multi-disciplinary approach in several domains, (a domain being a broad area of specialist services i.e. mental health, geriatrics, primary care, social services, etc.). This can be difficult to achieve if provision is by disjointed services. In many places in the world, lack of specialist services requires generic mental health services to treat older adults with dementia, depression, or other mental illnesses but this practice risks medical needs being unnoticed, or unappreciated. Frail older adults can find the experience of inpatient care in units with younger psychotic patients frightening and unacceptable. There are also different perspectives for working-age and geriatric psychiatry. Many working-age services promote a recovery model whereas care of older adults focuses on maintaining function, improving quality of life and paying more attention to the spiritual, environmental, and social influences on mental health. In addition, there is greater need to involve the social and family network of mentally frail elderly people than there is for working-age adults. For older adults, specialist services, with close collaboration with geriatricians and other geriatric providers, are preferred for optimal management.

Principles of good service delivery for older adults

Optimal service delivery starts by establishing the principles that services wish to adopt. These should govern the ethos of service delivery. For the World Health Organization (WHO), the mnemonic CARITAS (Latin for Compassion) summarizes a global consensus on specific values required for good service delivery for psychiatry of old age.[14] These principles, championed over many years by many international pioneers of mental health services for older adults, assert that optimal services are:

- **Comprehensive**

  They take all aspects of the patient's physical, psychological, and social needs and wishes into account i.e. are *patient-centred*.

- **Accessible**

  They minimize the geographical, cultural, financial, political, and linguistic obstacles to obtaining care.

- **Responsive**

  They act promptly and appropriately to a wide variety of patient needs.

- **Individualized**

  They focus on each person in her/his family and community context aiming, wherever possible, to maintain and support the person within her/his home environment.

- **Transdisciplinary**

  They optimize the contributions of people with a range of personal and professional skills and facilitate collaboration with voluntary and other agencies.

- **Accountable**

  They accept responsibility for assuring the quality of the service delivered, monitoring this in partnership with patients and their families. They are ethically and culturally sensitive.

- **Systemic**

  They work flexibly with all available services to ensure continuity of care.

Summarizing, good services provide *patient-centred* care with *easy access to a comprehensive range of services* delivered by *multi-disciplinary personnel* working in a *collaborative, responsive, respectful, and accountable* way.

Patient-centred care

The UK National Frameworks for Older People[15] and most OECD health administrations promote patient-centered care as a major means of improving quality of services and consumer satisfaction. Whilst most health professionals believe that they already practice patient-centred care, many patients would not agree. Predominantly, health systems for older people, particularly hospital-based, are far from patient-centered, being mostly organized around clinician or administrative requirements rather than patient needs. Patients encounter rigid appointment times, lack of evening or night services, inflexible boundaries between departments, inconvenient visiting hours, limited or expensive parking, and silo'd funding streams. Access or contact processes are obscure or difficult, information is inadequate, multiple assessments take place, and poor coordination between providers leads to treatment omissions or errors. Notably, older adults and their caregivers consider having multiple referrals to different specialists or providers and frequently repeating the same history to be a waste of time and resources.[16]

Consumer appraisals of their experience of services often result in common themes. Many experience poor communication—provider to patient, provider to caregiver, and provider to provider. Another common topic is lack of flexibility in developing management plans capable of involving several domains and dimensions of care (a dimension is a subset of a specialist domain, i.e. depression, dementia, continence, or mobility). As many elderly people have difficulties with mobility, or live far from services, transportation is another major issue.

In contrast, patient-centered services emphasize smoothing the progress of the patient 'journey' through the health system, eliminating duplication, matching care plans to patient needs, and generally making a demanding experience easier for the patient and their family. Unsurprisingly, the concept is appealing to patients and families. Increasingly, consumers, and their advocates, want to contribute to service planning, delivery, and evaluation.[17] So, what do older people want from their health care providers? Older people value their independence and being involved in the decision-making for their own care plans. Most want to remain in their own homes for as long as possible, but if that is detrimental for them, they want the right to relinquish decision-making, in various degrees, to other parties such as family members or their clinicians. They expect providers to treat them as an individual, to preserve their dignity, and to elicit and respect their preferences. Above all, they wish to be appropriately informed.[18] These desires are appropriate to all health care delivery for older adults, not just mental health. As most of us hope to grow old, we can empathize

with these wishes. Perhaps we need to remember that in planning and organizing services for older adults, we are potentially designing them for ourselves. The quality of care required is what we would be happy with, if we ever become clients.

Comprehensive and integrated services

Patient-centered care is more achievable with comprehensive or integrated services. The terms comprehensive and integrated are not interchangeable. A comprehensive service is one with a full range of inpatient and community services available within the same domain. An integrated service is one with a single point of entry capable of providing care plans that incorporate interventions and support in multiple domains and specific dimensions of care. Integrated services are characterized by a single point of entry, case management, geriatric, psychogeriatric and social assessments, and have multi-disciplinary teams.[3] They should have a seamless joining together of the various components of service, encompassing 'systemness' without diminishing component part identities.[19] Integration of different organizations is much easier if there are common administrative processes and financial systems. However, many mental health organizations have reporting and funding structures separate from other older adults' services (e.g. The Mental Health Trusts in the United Kingdom). Quasi-integration can be achieved by building effective *functional* links with a wide variety of health care professionals outside mental health e.g. primary care, geriatrics, acute medical and surgical care, social care, community health care, and non-clinical resources. For these collaborations to work, it is essential that bureaucratic processes enable easy transfer of funding and information across different entities and do not thwart clinicians' efforts to implement care plans for patients.

Integrated services are potentially more efficient as they should reduce duplication of assessments or investigations and service gaps. Currently, the majority of well-established services for older adults provide comprehensive rather than integrated services,[20] the latter being more ideology than practice, although this might be slowly changing. Research indicates that integrated care can delay institutionalization, reduce costs, and has benefits in consumer satisfaction[3] but is insufficient, as yet, to demonstrate that integrated services are more effective in achieving better health outcomes.

(a) The place of the common geriatric assessment (CGA)

An important tool for assisting integration is the common geriatric assessment (CGA). Different disciplines all have their own styles and foci for assessment but, despite individual differences, it is useful to have some common information for all teams and multidisciplinary groups, regardless of who takes the main responsibility for the patient.[21] Advocacy for the comprehensive geriatric assessment (CGA) covering all the main domains and dimensions of physical illness, mental health, disability, and social assessment, is increasing internationally, notwithstanding a lack of research on their effectiveness in improving health outcomes. They aim to save a patient from multiple repetitions of the same information.[16] To be useful, CGAs require personnel to work across professional and agency barriers, which can have benefits in creating relationships with allied colleagues, essential for developing integrated services. There needs to be agreement amongst the providers as to the applicability of the information required, agreed processes by which the CGA generates onward referral to the appropriate domains of care and procedures for updating and review. In some jurisdictions, (e.g. United Kingdom) CGAs or single assessment processes (SAPs) are mandatory for all older adults' services, in others, i.e. New Zealand and Australia, they are being trialed with a view to future obligatory use. SAPs and CGAs vary in their comprehensiveness and can aim at different levels, e.g. screening, proactive assessment, primary care, or secondary care services. They provide useful background information common to a range of providers but are not a substitute for specialized clinical assessment.

(b) The 'core business' of mental health services for older adults

Irrespective of whether a mental health service for older adults is part of a comprehensive or integrated system, their 'core *business*' is the:

- Diagnosis and management of new cases of mental illness arising in late life, often associated with the ageing process.
- Treatment of mental illness complicating physical illness and disability.
- Management of older adults with long-term mental illness complicated by ageing.
- Education and support for caregivers of older adults with mental illness.

Most psychiatry for older adults is about the management of chronic illness and care, rather than cure, is usually the main priority. An adaptation of the 5As model for patient-centred chronic illness management[22] is useful to describe the 'core *tasks*' of patient-centered psychiatry of old age. They are:

- Assessment of multiple care needs
- Advice on diagnosis and options for management
- Agreement with patient and caregiver on a care plan
- Assistance with implementation of care plan
- Assertive follow-up when needed

The 'core areas of *expertise*' for specialist services for older adults are the:

- Treatment of affective and psychotic disorders in late life
- Assessment of neurocognitive disorders and the management of the behavioural and psychological symptoms of dementia (BPSD)
- Rehabilitation of long-standing, chronic psychiatric disorders in patients whose disorder is complicated by physical illness or ageing
- Management of delirium in medically ill or complicating dementia
- Liaison with families, caregivers, and community providers

Core components of psychogeriatric service delivery

The evidence base for the 'core components'

Working with mentally ill older adults involves a variety of locations i.e. the patient's own residence, medical and surgical wards in a general hospital, psychiatry inpatient facilities, residential care facilities, outpatient clinics, outreach clinics, geriatric rehabilitation

units, or day hospitals. Several models of services for older adults have evolved over the past 30 or 40 years, shaping a degree of accord amongst clinicians on what are 'core components'. The evidence for these has been systematically reviewed by Draper and Low.[23] (See Table 8.7.1.)

Community old age psychiatry services

The lynchpin of geriatric psychiatry services is the community-based assessment and case management team. The community team model originated from the closure of the mental hospitals and the move into community-care in the 1980–90s. Their focus is domiciliary assessment and management. This offers the clinician opportunities to observe patients in their own environment, and promotes optimal cognitive functioning by decreasing stress for the individual. Home assessment avoids the sometimes-perceived stigma of attending a psychiatric clinic and eliminates transportation difficulties, as well as allowing ready access to family members or other caregivers. Treatment for most patients can take place at their residence (own home or nursing home), reducing reliance on inpatient or residential care. The model has proven to be efficient and highly acceptable to consumers and caregivers.

A typical multi-disciplinary team consists of at least one psychiatrist, psychiatric nurses, clinical psychologists, social workers, occupational, and other therapists plus support and administration staff. A psychiatrist traditionally leads a multi-disciplinary team but not necessarily so. As a whole, the team should be able to address the biopsychosocial, therapeutic, and psychoeducational requirements of a wide range of disorders and intervention settings. Team members case-manage depending upon their own special skills and expertise. Working collaboratively, with appropriate training, good supervision, and well-designed protocols and communication systems enhances the multi-disciplinary team. Some of the most effective teams are 'interdisciplinary' who develop flexible working patterns characterized by a non-hierarchical structure, and shared decision-making. They facilitate lateral communication between team members, and free exchange of ideas to develop optimal treatment and support management plans as a group. The evidence for community-delivered specialized multi- or interdisciplinary psychogeriatric assessment and management teams is strong and indicates consistently better outcomes than 'usual care' of primary care or generic mental health management.

Inpatient units

Inpatient units vary from the specialized older person's assessment, treatment, and rehabilitation (ATR) unit, similar to their geriatric counterparts, to dedicated beds in geriatric wards, or donated beds in working-age mental health units. Preferably, a specialized inpatient unit is purpose built or has the functionality to separate patients with functional and organic disorders, as each group has different clinical features, nursing needs, disabilities, and requirements for care plans. The evidence for specialized inpatient psychogeriatric units is positive but as there have been few random controlled trials (RCTs), it is less robust than the evidence for

Table 8.7.1 Level of evidence and study qualities for areas of service delivery

Area	No. of studies reviewed	No. of controlled trials	Quality range,* range (low) 0–1 (high)	Mean rating of quality	Level of evidence of effectiveness
Psychogeriatric day hospitals	10	0	0.43–0.82	0.57	Level IV (particularly depression)
Community old age psychiatry services	24	7	0.79–0.94	0.87	Level I for multi-disciplinary psychogeriatric teams, level IV for adult psychiatry teams
Integrated hospital and community-care	4	2	0.71–0.82	0.76	Level II for psychogeriatric services post-discharge care, no evidence for geriatric medical services (level I)
Primary care collaborations	3	2	0.89–0.94	0.92	Level II
Older people in general adult psychiatric wards	6	0	0.51–0.67	0.59	Level IV
Acute psychogeriatric wards	23	0	0.43–0.78	0.61	Level III-2
Hospital medical services	6	2	0.82–0.90	0.89	Level II for prevention of delirium without dementia, no evidence for other mental health outcomes (level 1)
Combined psychogeriatric and medical wards	3	0	0.52–0.53	0.52	Level IV
Hospital-based CL psychogeriatric service delivery	7	3	0.62–0.90	0.79	Level II effectiveness for reducing costs and length of stay
Long-term psychogeriatric care	11	0	0.58–0.71	0.66	Level III-2
Psychogeriatric outreach to long-term care	8	6	0.73–0.95	0.84	Level II for liaison style outreach services, Level III-2 for consultation style
Overall	108	25	0.62–0.95**	0.85**	

*If ≥2 controlled trials in service area, quality range reported for RCTs only, otherwise reported for all studies. **overall mean and range reported for controlled trials only.
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community teams. Nevertheless, what evidence exists points to specialized psychogeriatric units having better outcomes for psychogeriatric patients than working-age mental health, or geriatric medical units.[23] Scientific evaluation of combined psychogeriatric and geriatric medical wards is inadequate but expert clinical opinion considers them useful in the management of co-morbid medical and psychiatric illness.

Consultation and liaison

Hospital-based consultation and liaison (CL) services are important components because of the high number of mental disorders in the physically ill, general hospital populations. However, while the evidence is relatively good for CL outcomes such as hospital stays and costs, it is only modestly positive for mental health outcomes.[24] Notwithstanding, in recent years, older adults' CL services have outreached from the general hospital to provider organizations in the community. The evidence for these liaison services, usually provided by community teams to psychogeriatric long-term care facilities and voluntary organizations, is relatively good.[23]

Day Hospitals

Day Hospitals, originally attached to the old psychiatric institutions, have mostly devolved into community facilities and many United Kingdom psychiatrists of old age consider them indispensable.[25] Specialist day hospitals provide care for people with moderate and severe needs, including people with functional mental illnesses such as depression, anxiety, and schizophrenia, who may need specific support with daily activities and people with moderate to severe dementia. The Day Hospital allows hospital level treatment while allowing patients to remain living in their own residence. The evidence for their effectiveness, while positive, is sparse. As they are less common, they are not as revered in other parts of the world as much as they are in the United Kingdom. Day Hospitals need to be differentiated from Day Centres, managed by the voluntary or welfare sectors, which usefully provide social activities to keep the patient involved with their community and much needed respite for caregivers.

Residential care

An elderly person unable to support themselves in their own home needing daytime or 24 h supervision requires a resident caregiver or residential care facility. A community may have a range of residential care facilities managed by a variety of agencies such as local authorities, voluntary organizations, for-profit or not-for-profit religious and welfare organizations in the independent sector. These non-government organizations (NGOs) provide care homes for older adults who need support because of physical or mental frailty but not to the extent of requiring hospital level care. Many of the residents have some cognitive impairment and, unsurprisingly, a relatively high number of patients have complicating depression and/or psychosis. Residential care homes vary markedly in their ability to support older adults with mental health problems. Evidence from The Netherlands[26] and Australia[27] indicates that mental health enhanced care in the form of regular psychogeriatric team liaison to these residential facilities has beneficial outcomes for patients for example less inpatient stays, less psychotropic medication, and improvement in depression and psychosis.

Long-term care

Progressive de-institutionalization since the 1980s saw the transfer of many long-stay, public sector beds, for patients with dementia or intractable mental illness, to community-based nursing homes or hospitals. These facilities were generally smaller, more home-like and provided additional activities for the residents than traditional long-term care did. The change required many old age psychiatry services to collaborate with independent sector stakeholders and evolve outreach and liaison models of care with the new long-term residential care facilities. This paradigm shift for service delivery was beneficial for patient outcomes.[23]

Disturbed behaviour resulting from dementia (BPSD) can be severe enough for patients to need Specialized Care Units (SCUs). Examples are in Italy, France (CANTOUs), Tasmania (ADARDS unit), and London (Domus units). Such units have a small number of beds, well-trained staff with high staff to patient ratios and access to ongoing specialized psychogeriatric team care. When compared with traditional psychogeriatric ward patients, unsurprisingly, those in a specialized care unit, show improvements in cognition, self-cares, activity participation, and behaviour. The key elements to success appear to be training staff to anticipate and recognize mental health problems and close liaison with specialist mental health teams.

Respite care

The community-care model relies on family, rather than professionals to attend to most of the needs of a mentally frail, elderly person. This is demanding, difficult work for caregivers who usually have other responsibilities of family, home, and jobs. The main focus of respite care is to give lay caregivers a break from caring every few weeks, so they may continue to provide care and thus delay the need for permanent nursing home care as long as possible. Respite can be in the patient's own home, by providing a 'live-in' professional caregiver, or in hospital or a residential care facility for a week or two, every couple of months, allowing the family caregiver to take a holiday. Whilst seeming an admirable concept, the effectiveness of respite care is doubtful and there is little evidence that it has a significant effect on caregivers' burden, psychiatric status or physical health, or on patients' cognition, function, physical health, or rate of institutionalization.[28] Respite care, even when available, may be poorly utilized. Caregiver barriers to using respite care include guilt, financial reasons, cultural attitudes, and fear of stigma. Access barriers include unavailability, lack of publicity about services, long waiting lists, and poor identification of at-risk caregivers. Patient barriers include severe problem behaviours, immobility, incontinence, wandering, and inability to communicate.[29]

Primary care collaborations

Despite older adults forming the majority of patients seen by general practitioners (GPs), they detect and treat less than half the number of older adults with mental disorders.[30] Short consultation times, with a concentration on physical symptoms, with few patients presenting explicitly with mental health complaints, plus a reluctance of older adults, especially men, to express psychological distress to their GP, leads to under-recognition of mental disorders in older adults. Depression may be erroneously attributed to loneliness or ageing and early dementia overlooked. Primary care practitioners also make fewer decisions to treat or refer patients

to specialist services and often preferred to monitor, or defer decisions.[31]

Mere cooperation between mental health services and primary care seems insufficient to improve matters. Collaborations such as mental health enhanced primary care looks more promising. Education of primary care nurses in recognition of mental disorders and use of screening instruments might be useful to improve identification of mental disorders.[31] Even better is the idea of 'embedding' nurses, who have the skills necessary to identify health problems and coordinate care for older adults, into primary care practices. One experimental scheme in New Zealand, 'The Coordinators of Services for the Elderly' (COSE project) works within a primary care small group of practices to coordinate care of the practices' elderly patients across health, mental health, community, and accident services. An RCT of the COSE project over usual care significantly demonstrated that patients in the COSE arm were less likely to be hospitalized, their residential care was delayed and morbidity reduced.[32]

The primary care physician is crucial to patient care from start to finish, not only in identifying people who are at risk but also for any post hospital discharge care as the majority of patients seen by specialist services eventually return to primary care for ongoing management, in conjunction with their family caregivers. Even if patients or their caregivers can self-refer to secondary services, it is important not to bypass the general practitioner, who has awareness of the patient's overall health care and context.

Special components of services

Memory clinics

The substantial growth in numbers of memory clinics, over the past 10–15 years, was stimulated by the licensing of cholinesterase inhibitor drugs for Alzheimer's disease.[33] Memory clinics offer a range of services from assessment of cognition to specialized treatment of memory problems. Dedicated memory services can improve diagnostic expertise and lessen stigma for the patient. Usefully, they often focus on education of patients and caregivers as well as monitoring medications. However, the concept of a 'memory clinic' flies in the face of the trend towards community-based services and integration with local services. Furthermore, the intervention base is often very narrow. Very few studies provide any evidence of increased mental health gain over other psychogeriatric services, but there is some evidence that the attention, communication, and counselling offered increases consumer satisfaction.[34]

Older adults with intellectual handicap

People with intellectual handicap are also living longer and they are at particular risk for developing dementia. Over 55 per cent of people with Down's syndrome between the ages of 60–70 have Alzheimer type dementia, which often begins to emerge in midlife rather than old age. Consequently, the intellectually handicapped older person has complex care needs that require dementia, mental health, and learning disability services to work together assiduously.[2]

Older prisoners

An often forgotten group is ageing prisoners, who are also increasing in number. Older prisoners have increased risk for depression and other mental health problems. Long-serving older prisoners may develop dementia and require the special challenge of care delivery whilst incarcerated. Specialist services for older prisoners are scarce and, if available, usually provided by visiting community assessment and treatment services in conjunction with local forensic psychiatry services.

Younger people with dementia

Early onset dementia is fortunately rare but when it occurs, it is devastating. The patients usually have family responsibilities with young children, jobs, and financial commitments. Early onset dementia usually has a more accelerated course and genetically related family members may be concerned for themselves or their offspring. Diagnosis is often delayed for younger people so considerable distress and problems have usually built up before they reach services. Their management needs may also be different as they are usually physically fitter and they may require more structured activities than their older counterparts do, but need similar levels of supervision. Appropriate services for younger people who develop dementia are often scarce and the patients may fall into an under-resourced gap between working-age and older adults' services.

Academic units

Although there has been a growth in the number of geriatric psychiatry academic units and positions worldwide, they are still under-represented in universities worldwide. They are important providers of under and postgraduate teaching and research.

Planning to commissioning

Commissioning is 'the process of specifying, securing, and monitoring services to meet the needs of a population at a strategic level'.[2] Proposed services require a 'business case' with the place of an intended service clearly demonstrated in the overall schemata of health provision for a population. Demonstration of the need for services and projections of likely demand is necessary and the chief scientific tools available to identify these are epidemiology, demography, and utilization studies.

Epidemiology predicts the likely problems in a population and demography the characteristics of the population that could increase risk. The older the population, the more likely it will have a high prevalence of dementia requiring services. An important demographic to consider when planning services is the socio-economic status of a population. Low socio-economic status increases the likelihood of poor physical health, poor functionality and deprivation causes stress, leading to poverty of control over one's life, low self-esteem, anxiety, insecurity, and depression.[35] Also important is the number of immigrant residents. Ethnic elders are more likely to have earlier social disadvantage compounding in later life into a multiple jeopardy of social disadvantage, poorer physical health, and mental health problems.[36]

Whilst epidemiological studies may predict potential need, and demographics highlight areas of possible risk, not all people with problems will demand services. Demand or utilization is considerably less than the potential need as predicted from the demographical and epidemiological data. International research consistently shows similar demand patterns. About half of identified patients obtain treatment from a health care provider. Only 10–16 per cent reaches specialist mental health services, and

primary care treats about 30–40 per cent. For older adults utilization is even less than for working-age adults.[37] Stigma can have a detrimental effect on willingness to access services[38] as may cultural barriers.[36] For individuals, utilization of mental health services is more likely if the disorder is severe, is adversely affecting the family or social network, or the patient is female.

Services are usually commissioned based on expected demand rather than predicted need. Demand often increases, outstripping supply once services are available and information about them permeates into the community. Resource review on a regular basis as demand rises is necessary.

Commissioning new or revised services involves stocktaking of available resources for older adults. Often these are inequitable, with urban areas enjoying a range of services that rural areas lack. Despite unique individualities of different countries of the world, some characteristics tend to be true of all rural communities. They tend to be older and poorer than urban populations with a higher percentage of females.[39] Stoical older adults living in rural areas have a high tolerance of distress and are often reluctant to seek help from mental health services.[40] Consequently, rural populations are even more vulnerable for late-life mental health disorders, yet outreach services are usually infrequent, and primary care limited. Some countries with very large rural areas, for example United States, Canada, Africa, Australia, and New Zealand commission novel methods of outreach, such as flying doctor services.

Ideally, service planning should be in conjunction with key stakeholders, that is consumers, other geriatric services and providers, including NGOs. For optimal patient-centred mental health care for older adults to be effective, individual specialist hospital services need to work with general practitioners and other community providers. Attention to administrative pathways and funding structures that promote collaboration between providers is important. Commissioning strategies that propose integration of existing services need to recognize that, whilst the end result *may* be beneficial to patients, the process *will* cause enormous upheaval and distress among the workforce involved, particularly if there is decommissioning or amalgamation of duplicate services.

Service development is contingent on there being a skilled, educated workforce. A development plan needs to consider the specialist disciplines required, based on expected case mix and work loads, staff availability, recruitment and retention, morale, job satisfaction, and very importantly, training issues. Clinical services have important roles in teaching and training ongoing professional development and in clinical evaluation, and systems research. These tasks can also be important means of cross-fertilization, dissemination of ideas between related services, teams, and disciplines. Involvement of different disciplines in undergraduate or postgraduate teaching programmes including psychiatry trainees can be useful for disseminating a broader perspective to prospective health professionals intending to work with older adults.

A vital aspect of commissioning is monitoring, that is, the methods of service review and evaluation. This is important for continuing quality initiatives and research but also to provide evidence for review and future commissioning of even better services.

Conclusion

Worldwide, population ageing is driving the development of mental health services for older adults. Finite resources, burgeoning costs, expanding therapeutic repertoires, and increasing consumer interest in involvement in health care is challenging health organizations to develop effective, efficient, and economic patient-centred services for older adults. Fundamentally, the quality of services is dependent on health personnel working with their patients and other providers, towards shared aims of improving health outcomes and quality of life. Clinicians' adaptability, flexibility, responsiveness, availability for patients, and willingness to collaborate are the keys to success in developing future services for older adults.

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SECTION 9

Child and Adolescent Psychiatry

9.1

General issues

Contents

9.1.1 Developmental psychopathology and classification in childhood and adolescence

Stephen Scott

Introduction

Classification schemes of psychiatric disorders in childhood and adolescence have to take into account three particular features. Firstly, the individual is continually changing and growing. Sound knowledge is therefore required of the normal range of development and its limits. For example, some fears may be normal in a 5-year-old but abnormal in an 8-year-old. Once identified, it is helpful to decide if abnormalities are due to *delay in* or *deviance from* the usual pattern of development. The implications of each differ, and should be classified differently. Secondly, the majority of childhood mental health problems arise from an excess of behaviours exhibited by many young people, such as aggression or dieting. They are seldom due to qualitatively distinct phenomena of the kind more often seen in adult conditions, such as hearing voices or hanging oneself. Consequently choosing a cut-off point to make a categorical entity from a dimensional construct is more often used in child psychiatry. This is inevitably an arbitrary process (albeit informed by empirical criteria), which may lead to loss of information, and may be held to be labelling the child unnecessarily. That dimensions can be interchanged with categories does not necessarily mean they are unhelpful—after all, day and night are useful terms yet the boundary between them is continuous and arbitrary. Psychiatrists in particular may be criticized for 'medicalizing' a child's difficulties by talking about disorders or diagnoses, whereas other professionals and parents may prefer to see them as understandable variations in child development, and prefer to call them 'emotional and behavioural difficulties'. However, diagnoses are a quick way to convey a lot of information that dimensions may not. Thus to only say a child is at an extreme of an antisocial behaviour dimension does not necessarily convey the association with specific reading retardation and ADHD, which could be (and often are) consequently missed.

Thirdly, children's difficulties nearly always arise in the context of relationships within the family. More often than in adulthood, some or all, of the problem may appear to be the result of the functioning of the family, rather than in the individual child who may merely be reacting to the situation. For example, a child who is disobedient and shouts in class may simply be behaving the way his parents do at home. A classification system will be stronger if it can take family functioning into account—it will have a greater chance of capturing clinically important causal and therapeutic considerations.

A valid and useful classification system will need to take into account these features and be based on a thorough understanding of normal development and how it can go wrong, rather than merely include static descriptions of presumed pathological states. The term of *developmental psychopathology* was coined in the early 1980s to denote the scientific study of how abnormalities can be understood in terms of processes underpinning human development.[1,2] There are now journals and books incorporating the term into their titles.[3,4] Many disciplines are relevant, from embryology and genetics to social learning theory and criminology. Developmental psychopathology, besides studying the impact of pathogenic influences on pathways through life, such as the way the

monoamine oxidase-A (MAOA) genotype interacts with an abusive upbringing to cause conduct disorder,[5] or how specific reading retardation leads to low self-esteem, also investigates protective mechanisms, such as the ameliorating effect of high IQ on the propensity to juvenile offending, or the beneficial effect of a trusting and caring relationship with an adult on the impact of childhood abuse. This chapter aims to show how findings from developmental psychopathology have informed current classification systems, and what challenges remain.

General issues

Change over time

Because mental processes and behaviour change as a child develops, it is not always clear whether the same diagnoses should be applied across the age range. Thus a highly aggressive toddler may throw himself screaming onto the floor in daily tantrums, whereas a highly aggressive teenager may assault old ladies and rob them. Do they suffer from the same disorder? ICD 10 holds that they do—both meet criteria for conduct disorder, which is defined in terms of antisocial behaviour that is excessive for the individual's age, and that violates societal norms and the rights of others. DSM IV-R on the other hand has two separate diagnoses, oppositional-defiant disorder for the younger case, and conduct disorder for the older. However, as both diagnoses have similar correlates and there is a strong continuity from one to the other, the validity of the division is questionable. Yet current adult psychiatric schemes have no diagnosis at all to apply to antisocial behaviour, unless it is part of a personality disorder.

The extent to which adult criteria should be applied to children requires good empirical data. In the case of obsessive–compulsive disorder, the phenomenology is remarkably similar in childhood, so there is no problem. However, for depression the picture is rather different. Currently, ICD 10 and DSM IV-R have few emotional disorder categories specific to childhood, and they are mostly subtypes of anxiety. Mood disorders are diagnosed according to adult criteria, with the consequence that surveys of depression find prevalence rates close to zero under 8 years of age. Yet there are miserable children who cry frequently, say they are unhappy, look sad, and are withdrawn.[6] However, they usually sleep and eat reasonably well, and their mood fluctuates during the day, with spells when they sometimes appear more cheerful. Should they not be allowed a diagnosis? ICD 9 had a category for 'disturbance of emotions specific to childhood and adolescence, with misery and unhappiness', and such children suffer impairment.[7] Follow-up studies of prepubertal children referred with this picture showed a moderately increased risk of adult type depression later on, whereas adolescents with depressive symptoms had a higher risk of adult depression.[8] Genetic studies show that symptoms of depression in prepubertal children are predominantly due to environmental influences, whereas after puberty genetic influences become more important.[9] Finally, tricyclics are not effective in childhood but are effective in adults. This example shows that misery in younger children has some phenomenological features and external correlates in common with adult depression but also several differences, so the current approach which makes a comprehensive yet parsimonious classification system for all ages loses validity.

In contrast, there is continuing reluctance to diagnose personality disorders in childhood. This may be because they are often seen as a life sentence of a noxious, untreatable condition, in distinction to the general hope that there is opportunity for 'growing out of' conditions in childhood, or treatment for them. However, with perhaps the most destructive personality type, dissocial, there is growing evidence that the combination of antisocial behaviour and callous-unemotional traits is well established by the age of seven. Moreover, this combination of childhood characteristics has a far higher heritability than antisocial behaviour without callous-unemotional traits.[10]

Validity

Categories need to be distinct not only in terms of the phenomena used to define them, but, crucially, also in terms of external criteria. Even if categories can be reliably distinguished, if external criteria are the same, then one is likely to be dealing with two variants of the same condition. An analogy would be the difference between black and white cats.

Typical validating criteria in child psychiatry derived from developmental psychopathology are:

1 *Epidemiological data*, such as age of onset and sex ratio. Forty years ago 'childhood psychosis' was a unitary classification, but work showing the clear difference in age of onset helped validate the distinction between autism and schizophrenia, which seldom co-occur. Disruptive disorders occur four times more commonly in boys, whereas emotional disorders are commoner in girls.

2 *Long-term course*. Most childhood disorders show reasonable *homotypic continuity*, that is they stay the same. Some show *heterotypic continuity*, so that for example, some cases of childhood hyperactivity end up as antisocial adults. This does not necessarily invalidate the category, but requires explanation.

3 *Genetic findings*. If individuals with distinct categorical diagnoses have relatives with different disorders, this helps validate the distinction. This has confirmed the validity of several diagnostic categories, but not all. For example, it has not held for the many specific subtypes of anxiety disorder in ICD 10, whose validity is questionable. Genetic studies can also clarify the scope of symptom clusters. For example, family studies of autism have revealed a broader phenotype in relatives of probands,[11] so that new disorders may need to be considered, which encompass only one of the original three constituent domains of classical autism, namely social relatedness, communication problems, and repetitive and stereotyped behaviours.

   The hunt is now on for specific genes associated with particular psychiatric disorders. Thus dopamine receptor and transporter genes are reliably associated with Attention Deficit Hyperactivity Disorder,[12] but unless (i) the gene always leads to the disorder and (ii) all cases of the disorder are caused by the gene, particular genotypes are unlikely to be used to validate diagnostic categories.

4 *Psychosocial risk factors*. The association between institutional upbringing with many changes of carer and reactive attachment disorder is so strong that it has been made a requirement for diagnosis in ICD 10. Conduct disorders are strongly associated with discords at home, whereas autistic disorders are not. However, most psychosocial risk factors are less specific in their associations, and so are only modestly helpful as validating criteria.

5 *Neuropsychological tests*. The hyperkinetic syndrome is clearly distinguishable from conduct disorder on tests of attention such

as the continuous performance task. Recently, there has been considerable progress in showing that one of the core deficits in autism is failure on 'theory of mind' tests of ability, to see another person's point of view, which non-autistic children, with comparable levels of intellectual disability, can do.

6 *Medical investigations*. There have been many failed attempts in this field, including biochemical markers of adolescent depression and endocrine markers of aggression. However, the advent of functional neuroimaging is allowing exciting relatively non-invasive pictures of children's brains to be built up, and reliable findings are beginning to emerge, for example in ADHD.[13] In future these may well be helpful validators for classification.

Reliability

This is a prerequisite for validity, and most categories have reasonable inter-rater and test–retest values, once investigators are trained up. Where there are many overlapping categories, as in current definitions of the many varieties of anxiety disorders, or personality disorders, inter-rater reliability falls.[14]

Effect of informant and instrument

Traditionally information is obtained from parents and the child, and is then combined by the clinician on a case-by-case basis. However, the need for consistent diagnostic rules that is imposed by a 'menu-driven' approach can prove difficult, since the weight given to a particular informant may best vary according to condition. Thus, if a parent says a child has symptoms of conduct disorder but the child denies it, the parent is more likely to be right and the child may be covering up or ashamed. However, if the parent says the child is not depressed but the mental state examination of the child reveals otherwise, it is the parent who may be ignorant of their child's true state. Such difficulties reduce the validity of interviews which use invariant combination rules. Further, in genetic studies, the heritability of a condition may vary greatly according to which informant is believed. Thus in the Virginia Twin study, conduct disorder was 69 per cent heritable according to the information derived from the mother interview, 36 per cent using information from the child, and only 27 per cent using information from the father.[15] Studies such as these underline the need for clinically sensitive ways of combining information, and the use of multi-informant, multi-method ascertainment of information. Statistical techniques such as latent variable analysis may help reduce measurement error, but may build in unwarranted assumptions which distort the raw data.

Structured interviews, which accept the respondent's reply, do not require lengthy training or clinically informed investigators, and so are popular in epidemiological surveys. However, the quality of information differs little from that obtained by questionnaire,[16] and often has a high false-positive and false-negative rate in comparison to semi-structured interviews. Direct observation, although expensive, often provides the most reliable and valid information for assessment of disruptive disorders.

Comorbidity

There are many artefactual reasons for comorbidity appearing high, such as Berkson's bias[17] in clinical samples (where not all cases get referred, the chance of referral will be related to the *combined* likelihood of referral for each condition separately), or overlapping criteria, or artificial subdivision of syndromes. However, even after taking these possible sources of error into account, comorbidity is marked for child psychiatric disorders. In a meta-analysis of community samples,[18] the odds ratio for anxiety with either Attention Deficit Hyperactivity Disorder (ADHD) or conduct disorder is 3, for anxiety and depression 8, and ADHD and conduct disorder 10. Rates are even higher in clinical samples. True comorbidity may arise through several mechanisms:[19] (i) shared risk factors (e.g. early deprivation may lead to oppositional-defiant disorder and an attachment disorder), (ii) overlap between risk factors (thus a depressed mother may pass on a genetic liability to depression in her son and provide inconsistent discipline which predisposes him to conduct disorder), (iii) one disorder creating an increased risk for the other (e.g. conduct disorder leading on to drug dependency), or (iv) the comorbid pattern constitutes a meaningful syndrome (e.g. depressive conduct disorder, described below under combined categories).

Some classification schemes

A simple scheme with three main groups of disorders

A simple but well researched, valid way of grouping child disorders 'lumps' them into three groups, which are helpful to hold in mind when considering specific diagnoses:

Emotional disorders including anxiety, depression, phobias, somatization, and obsessive–compulsive disorder; **disruptive disorders** including conduct disorder and hyperactivity; and **developmental disorders** including intellectual disability, the autistic spectrum, language and reading delays, and enuresis and encopresis.

Comorbidity within each grouping is very common, but only occurs across groups in a minority of cases. External criteria validating the differences between these groups are given in Table 9.1.1.1.

Table 9.1.1.1 Validating criteria for main diagnostic groupings

	Emotional disorders	Disruptive disorders	Developmental disorders
Age of onset	over 8	under 8	under 3
Sex ratio	commoner in girls after puberty	commoner in boys	commoner in boys
Family size	normal	large	normal
Family history	anxiety and depression increased	criminality increased	related disorders may be increased
Socio-economic status	normal	lower	normal
IQ	normal	lower range of normal	normal or low, sometimes very low
Specific delays	absent	present in a third	common
Neurological signs	absent	uncommon	common
Cause	mixed, sometimes mainly genetic	mixed, sometimes mainly environmental	often mainly genetic

Current schemes: ICD 10 and DSM IV-R

The DSM IV-R and ICD 10 committees worked closely together and strove to have names and criteria that are as close as possible. However, there are some general differences.

(a) 'Picture-fitting' versus 'menu-driven' approaches

Firstly, as in adulthood, ICD 10 has one set of 'clinical descriptions and diagnostic guidelines' and a separate set of 'diagnostic criteria for research'. The former comprises general descriptions of disorders requiring a qualitative matching of case characteristics with the scheme, a 'picture-fitting' approach which is similar to the way clinicians practise. The latter comprises lists of symptoms with explicit criteria detailing the number and permutation required for diagnosis, a 'menu-driven' approach. DSM IV-R has only the latter. It has advantages in increased reliability, but is relatively cumbersome so that many clinicians do not bother to apply the criteria rigorously. Even for the simpler DSM III criteria, a study found that whilst trained researchers achieved kappa values of 0.83, 0.80, and 0.74 for attention deficit disorder, conduct disorder, and emotional disorder, the comparable figures for United States clinicians in regular practice were 0.30, 0.27, and 0.27, which are seriously low.[20]

A further disadvantage of the 'menu-driven' approach arises in cases where although the clinician believes a diagnosis is present because of the severity of symptoms, their number is insufficient to meet criteria. For example, consider the following youth: he repeatedly mugs old ladies, sets fires frequently, often argues, is often spiteful or vindictive, has unusually severe tantrums, and has no friends or job because of his behaviour. According to ICD 10 research diagnostic criteria (or DSM IV-R criteria) he has no diagnosis, as he has two but not three symptoms of conduct disorder, and three but not four symptoms of oppositional-defiant disorder. However, according to ICD 10 'diagnostic guidelines' he easily meets the requirements for conduct disorder since 'any category, if marked, is sufficient'.

(b) Multiple diagnoses

A second difference between ICD 10 and DSM IV-R is in multiple diagnoses. ICD 10 encourages the selection of one diagnosis that closest fits the picture, assuming that differences are due to a variation upon the typical theme. DSM IV-R (and the closely linked ICD 10 research criteria) encourage selection of as many diagnoses as criteria are met. Problems arise with this approach when symptoms are common to two disorders, for example irritability contributes to affective disorders and to conduct disorders, so double coding is more likely. Since comorbidity is very common in clinical practice, multiple coding is frequent using a 'menu-driven' approach so that it begins to approach a dimensional system and to lose the advantages of categorization.

The pros and cons of each approach will vary according to whether extra information is conveyed by the second diagnosis. Where there is good evidence of the validity of common comorbid conditions, ICD 10 has combined categories. Thus the external validating characteristics of 'depressive conduct disorder' are similar to those of pure conduct disorder, with no increase of affective disorders in individuals, nor in their relatives, followed up to adulthood. Double coding would convey erroneous information about the depressive aspect. 'Hyperkinetic conduct disorder', on the other hand, is characterized by more severe neuropsychological deficits than occur in either condition alone, and by worse psychosocial outcome in adulthood. Double coding would not convey the poor prognosis.

(c) Multiaxial framework

The ICD 10 has a multiaxial framework for psychiatric disorders in childhood and adolescence[21] which will be described here. DSM IV-R uses a somewhat different multiaxial framework, which is applicable for disorders arising at all ages. It will not be described here except as a contrast to ICD 10. Each axis except the last (psychosocial impairment) is coded independently of the apparent causal contribution to the psychiatric syndrome. This avoids tricky decisions about causality and allows conditions to be recognized and clinical needs addressed.

(i) Axis one: clinical psychiatric syndromes

Criteria for particular diagnoses are described in the relevant chapters of this text.

(ii) Axis two: specific disorders of development

These include speech and language, reading, spelling, and motor development. In DSM IV-R they are included in Axis one. However, having a separate axis helps to ensure that they are not overlooked. This can easily happen, for example, in children with conduct disorder, where the antisocial behaviour tends to command attention, while in fact one-third of the children also have specific reading retardation (dyslexia), which if untreated worsens the prognosis.[22] It very desirable to administer standardized psychometric tests in order to characterize specific disorders of development.

(iii) Axis three: intellectual level

The categories are no intellectual disability (IQ 70 or over), mild intellectual disability (50–69), moderate intellectual disability (IQ 35–49), severe intellectual disability (IQ 20–34), and profound intellectual disability (IQ under 20). In DSM IV-R personality disorders are also included on the axis.

Subtyping intellectual disability gives a good example of substantial differences which arise when categories are imposed on top of a dimensional construct. If all children with an IQ below 50 are taken together (often together also called severe), and compared with those having an IQ of 50–70 (mild), major differences emerge on independent validating criteria, as shown in Table 9.1.1.2.

From the table it will be seen that there are major differences between the categories on fronts as varied as brain pathology and life expectancy. There is no particular psychiatric pattern arising in children with intellectual disability, rather the incidence of all disorders is raised, so that in those with IQ under 50, fully one half have a psychiatric disorder.[23]

(iv) Axis four: associated medical conditions

All medical conditions should be coded. A few have specific associations with psychiatric disorders, for example tuberous sclerosis predisposes to autism, Cornelia de Lange syndrome to self-injury; Down syndrome on the other hand protects against autism but often leads to presenile dementia. Even where there is no specific disorder, congenital syndromes are often characterized by a particular pattern of behaviour. The study of these *behavioural phenotypes* is a discipline in its own right.

(v) Axis five: associated abnormal psychosocial conditions

These include a range of pyschosocial hazards, from abnormal intrafamilial relationships such as physical or sexual abuse, to mental disorders in other family members, distorted intrafamilial communication patterns, abnormal upbringing, e.g. in an institution,

Table 9.1.1.2 Characteristics of children with severe versus mild intellectual disability

	Severe retardation	Mild retardation
Definition	IQ under 50	IQ 50–70
Social functioning	Invariably marked impairment	Many have minor or no impairment
Cause	Organic pathology in majority	Usually no organic cause evident
Family history	Parents and siblings usually of normal intelligence	Parents and siblings often at lower levels of intelligence
Background	Fairly equal distribution across SES levels Neglect at home unlikely	Much commoner at lower SES levels Neglect at home more likely
Appearance	Dysmorphic features often evident	Normal appearance
Medical complications	Physical handicap common (e.g. cerebral palsy) Major health problems frequent Life expectancy shortened Fertility low	Physical handicap uncommon Health in normal range Life expectancy normal Fertility little impaired
Psychiatric complications	Severe and pervasive disorders such as hyperactivity, autism, and self-injury especially common Presentation of disorders often altered, mental state may be difficult to determine	Disorders similar in type to those found in children without MR, but occur more frequently Form of disorders and mental state examination similar to children without retardation

acute life events, and chronic interpersonal stress arising from difficulties at school. Each is coded dimensionally on a three point scale. As the number of psychosocial adversities goes up, the rate of psychiatric disorders increases.[24] Conduct disorder is particularly associated with poor immediate psychosocial environments. As with other axes, abnormalities are coded irrespective of apparent cause. This is particularly relevant since while perhaps 20 years ago the mechanism was thought to be directly environmental, in the last 10 years good evidence has been collected to show that some environmental characteristics of the home are genetically mediated.[25] For example, the association between lack of books in the home and poor child reading is partly mediated through parents with lower IQ buying fewer books.

(vi) Axis six: global social functioning

Here a judgement is made on a nine point dimensional scale ranging from superior social functioning to profound and pervasive social disability. Unlike other axes, ratings of disability are not independent, but have to be judged as due to a psychiatric or developmental disorder on axes one to three. Thus impairment arising from adverse circumstances cannot be coded—it must arise from intraindividual factors. This rule therefore excludes recognition of psychosocial interventions which aid functioning, from reduction of parental Expressed Emotion to changing schools. DSM IV-R studies often use the Children's Global Assessment scale,[26] an adaptation of the Global Assessment of Functioning (GAF) used in adults. An advantage of the CGAS is that it is rated without impairment having to be caused by psychiatric disorder. A disadvantage is that psychiatric symptoms, rather than impairment alone, contribute to the rating.

(d) Should impairment of social function be part of psychiatric diagnosis?

In general, ICD 10 and DSM IV-R do not require impairment of social functioning to be present in order to make a diagnosis. There are exceptions, thus in DSM IV-R, oppositional-defiant disorder *does* require impairment. With many qualitatively distinct adult disorders, having no impairment criterion makes sense, so that a person experiencing the delusions and hallucinations characteristic of schizophrenia, but able to go to work and form relationships while on neuroleptics still has schizophrenia. But should a child who says he is afraid of dogs and crosses to the other side of the pavement on seeing one, but otherwise functions well, be deemed to suffer from a phobia? If impairment criteria are not applied, very high rates of disorder are obtained in epidemiological surveys. This lacks credibility with the general public, who may then dismiss all psychiatric problems in children, and is unrealistic for clinicians and health planners, who would not see most of the identified individuals as cases needing treatment. For example, a large epidemiological survey[27] found that using DSM III criteria, 50 per cent of children and adolescents had a diagnosis. However, when an impairment criterion was added, the figure came down to 18 per cent. This would appear to be a much more realistic figure. However, it could be argued that social impairment is too constraining, and for example would exclude an adolescent who is fairly depressed but able to function. The term *impact* can be used to include subjective distress as well as impairment, and is gaining in popularity among many child psychiatrists.[28]

Falling through the cracks: children with social impairment but no diagnosis

Diagnostic systems have to be practically useful above all. If they are overinclusive, the risk is that there are too many categories, which have poor reliability and high overlap. If on the other hand they are too exclusive, the risk is that there will be many individuals suffering from problems which are not encompassed by the scheme. In one thorough survey, 9.4 per cent had no diagnosis but significant impairment.[29] Across a variety of 'caseness' measures, the individuals were as disturbed as those with a diagnosis. Many of the difficulties were around relationships with parents and siblings, and arguably, such children who have symptoms associated with psychosocial impairment should be regarded as suffering from a psychiatric disorder.

Conclusion

Classification of child psychiatric conditions has advanced enormously in the last 20 years. There is a much stronger empirical basis to support current schemes, which are grounded in the many scientific disciplines that contribute to developmental psychopathology. Nonetheless there are considerable obstacles to overcome if DSM V and ICD 11 are to be major steps forward.

Further information

To access the journal *Development and Psychopathology*, visit http://journals.cambridge.org/action/displayJournal?jid=DPP

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To access the official website discussing issues around DSM IV criteria, visit http://www.dsmivtr.org/index.cfm and regarding ICD 10 visit http://www.who.int/classifications/icd/en/

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9.1.2 Epidemiology of psychiatric disorder in childhood and adolescence

E. Jane Costello and Adrian Angold

Epidemiology is the study of patterns of disease in human populations.[1] Patterns are non-random distributions, and patterns of disease distribution occur in both time and space. Whenever we observe a non-random distribution, we have the opportunity to identify causal factors that influence who gets a disease and who does not. For example, we observe that depression rises rapidly after puberty in girls, but not to the same extent in boys.[2] This non-random distribution in time suggests that there may be something about puberty in girls that is causally related to depression.[3] An example of disease distribution in space can be seen in the Methods for the Epidemiology of Child and Adolescent Mental Disorders (MECA) study of five sites in the United States and Puerto Rico.[4] Although the prevalence of psychiatric disorders was fairly similar across sites, the likelihood that a psychiatric diagnosis was accompanied by significant functional impairment was much higher in children at the mainland sites than in Puerto Rico. This offers the opportunity to study between-site differences that might result in differences in the level of impairment caused by psychiatric disorders. The task of epidemiology is to understand these observed patterns in time and space, and to use this understanding as a basis for the prevention and control of disease.

Epidemiological medicine has both similarities to and differences from clinical medicine. Like clinical medicine, epidemiology is an action-oriented discipline, whose goal is intervention to prevent and control disease. Scientific knowledge about the cause and course of disease is another common goal. Epidemiology also reflects clinical medicine in using two methods of attack on disease: *tactical* methods, concerned with the practical and administrative problems of disease control at the day-to-day level, and *strategic* methods, concerned with finding out what causes disease so that new weapons of prevention and control can be engineered.[5,6] Thus, for example, in their tactical or public health role epidemiologists can be found reporting on the prevalence of adolescent drug abuse, the social burden (including cost) that drug abuse creates, and the best ways to control its spread, while others working at the strategic level might be exploring the science underlying environmental constraints on gene expression.

Epidemiology diverges from clinical medicine to the extent that it concentrates on understanding and controlling disease processes in the context of the *population at risk*, whereas the primary focus of clinical medicine is the *individual* patient. This does not mean that epidemiology is not concerned with the individual; on the contrary, it is very much concerned with understanding the individual's illness and the causes of that illness. The difference lies in the frame of reference. Put crudely, clinical medicine asks: 'What is wrong with this person *and how should I treat him or her*?' Epidemiology asks: 'What is wrong with this person and *what is it about him or her that has resulted in this illness*?' Why is this child depressed, but not her brother? If her mother is also depressed, is the child's depression a cause, a consequence, or an unrelated, chance co-occurrence? Such questions immediately set the individual child within a frame of reference of other children, or other family members, or other people of the same sex or race or social class.

Sampling, or selecting the population within which to count cases, is of central importance in epidemiology. Counting cases is an important first step towards measuring the social burden caused by a disease, and the effectiveness of prevention. For most diseases, however, simply counting the number of individuals presenting for treatment will produce estimates that are seriously biased by referral practices, ability to pay, and other factors. This is a big problem in child psychiatry because parents, teachers, and pediatricians all serve as 'gatekeepers' to treatment.[7] Community-based data are needed to measure the extent of need, and the unmet need, for prevention or treatment. Methods for assessing psychiatric disorders in the general population are discussed in another chapter. However, it is worth noting that methods for assessing disorder, whether they take the form of interviews, questionnaires, or neuropsychiatric tests, can only be as good as the taxonomy they are designed to operationalize. Current instruments mainly use scoring algorithms that turn the responses into diagnoses based on the DSM-IV or ICD-10 taxonomies. If these taxonomies do not mirror the 'reality' of psychiatric disorder then the results of using interviews or questionnaires based on them will in turn be faulty.

Estimating the burden of child and adolescent psychiatric disorders

In a world of scarce health care resources, it is important to understand the size of the burden to the community caused by these disorders. Burden, in terms of numbers affected, impact on the individual, and cost to the community, is a crucial factor in the battle for resources for treatment and prevention.

Attempts to reduce the burden of mental illness must, of necessity, pay attention to the early years. It is becoming increasingly clear that most psychiatric disorders have their onset before adulthood, and that many should be regarded as chronic or relapsing disorders. For example, the National Comorbidity Survey Replication, a representative population sample of over 9000 adults aged 18 and over in the United States,[8] found that, of the 46.4 per cent of all participants reporting one or more psychiatric disorders during there lifetime, half reported onset by age 12, and three-quarters by age 24.[9] Since we can expect a lot of forgetting of early episodes by older participants,[10] it is likely that onset in childhood is even more common than this.

If the burden of mental illness begins to be felt in childhood, it is important to know the extent of the problem so that we can begin to plan for treatment and prevention. Unfortunately, the data on which to build such estimates are very sparse. We have to rely on a national prevalence study of psychiatric disorders in the United Kingdom, and another of a large area of Brazil, together with a few national or large community surveys using symptoms scales, and a handful of diagnosis-based studies in smaller community samples, some of them longitudinal. Questionnaire-based surveys are not very useful for measuring prevalence, because they tend to define 'caseness' in terms of a certain percentage of the sample with high scores; a method that predefines prevalence.

In the past decade the United Kingdom has carried out a national prevalence study,[11,12] conducted by the Office for National Statistics, with funding from the Department of Education and other agencies. The primary purpose was to produce prevalence estimates of conduct, emotional, and hyperkinetic disorders, as well as pervasive developmental disorder, eating disorders, and tic disorders, using both ICD-10 and DSM-IV criteria. The second aim was 'to determine the *impact* or *burden* of children's mental health. *Impact* covers the consequences for the child; *burden* reflects the consequences for others'.[13] (p. 185). Third, the study measured service use. A stratified random sampling plan for England, Scotland, and Wales produced a sample of 10 438 children aged 5 to 15. Parent and child were interviewed using the Development and Well-Being Assessment (DAWBA),[14] a computer-assisted lay interview that uses a 'best-estimate' approach to diagnosis, in which responses recorded by lay interviewers are evaluated by clinicians. The first interview wave, conducted in 1999,[13] was followed by a questionnaire mailed 18 months later to all 'cases' with a diagnosis at Time 1, and a one-in-three random sample of non-cases. A second interview of all those completing questionnaires at Time 2, and all others who were cases at Time 1, was completed in 2002.[15] By weighting the responses to account for the various selection factors and for non-response, Meltzer and colleagues developed estimates of prevalence (i.e. the presence of a disorder at the Time 1 interview), of incidence (new cases between the two interviews), and of persistence.

The UK study found that almost one child in 10 (9.5 per cent) aged 5 to 15 had a psychiatric disorder based on the ICD-10 classification system. Prevalence was higher in adolescents (11.2 per cent at 11 to 15) than in children (8.2 per cent at 5 to 15), and in boys (11.4 per cent than girls 7.6 per cent). Conduct disorders were the most common (5.3 per cent), followed by anxiety disorders (3.8 per cent). Depression was rare in both sexes and all age groups

(0.9 per cent over all), as were hyperkinetic disorders (1.4 per cent). Seven per cent of previously unaffected children developed a psychiatric disorder in the 3 years between the interviews. Four per cent developed a new emotional disorder (anxiety and/or depression), and 5 per cent a behavioural and/or hyperkinetic disorder. More girls developed emotional disorders, and more boys developed behavioural disorders. Persistence, measured as the presence of the same diagnosis the years apart, was higher for behavioural disorders (43 per cent) than for emotional disorders (about one in four).

Factors affecting prevalence estimates

It is not a simple matter to compare the British prevalence rates with those from other countries, because there are few large studies, and the age ranges do not overlap. A study of youth age 7 to 14 in south-eastern Brazil, which used the same diagnostic interview but the DSM-IV taxonomy, found an overall prevalence of 12.7 per cent. Although prevalence estimates were slightly different from those reported by the UK study, the relative ordering was the same. Behavioural disorders were again the most common (7 per cent), followed by anxiety disorders (5.2 per cent) and ADHD (1.8 per cent). Once again, depression was rare (1.0 per cent). Other studies from around the world[16] usually generate prevalence rates of around 20 per cent. This puts the British and Brazilian studies at the low end of the range. However, there are many factors other than the 'true' rate of psychiatric disorder (if there is any such thing) that affect a published prevalence rate. The most important of these are:

1 *The time frame of the diagnostic measure*. Questions can be asked about symptoms occurring 'now', 'in the past month', 'in the past 3, 6, or 12 months', or 'ever'. Clearly, if recall is accurate the latter questions will elicit more symptoms than the former. Unfortunately, recall is not always accurate. Prevalence rates are higher from interviews with longer time frames, but not as much higher as would be consistent with accurate recall. For example, The National Comorbidity Study Replication, based on a nationally representative sample of adults in the United States, found that the lifetime prevalence of any disorder was 46.4 per cent, while the 12-month prevalence was 26.2 per cent. This means that 26.2 per cent /46.4 per cent = 56.5 per cent of all cases across the lifespan were present in the past 12 months. This could be explained in several ways: (i) there was an epidemic of psychiatric disorders in the 12 months before the survey; (ii) over half of all psychiatric diseases are chronic; once they occur they remain active for the rest of life; (iii) many early episodes are forgotten, and people report the onset of the most recent episode as the first occurrence of the disorder. In the absence of any evidence for (i), some combination of (ii) and (iii) seems the most likely explanation. We have evidence that the reliability with which children and adults recall the first occurrence of a symptom falls dramatically after 3 months,[17] and recommend concentrating on symptoms occurring in the past 3 months if a fairly reliable estimate is sought.

   In general, when comparing prevalence rates from different reports it is important to bear in mind the time frame. In a comparison of reported rates of child and adolescent depression published since the 1970s, we found that the time frame of the interview accounted for most of the variance, compared with taxonomy (DSM-III, DSM-IIIR, DSM-IV, ICD-9, ICD-10), diagnostic interview, or birth cohort.[2]

2 *The number and nature of the informants*. For several decades now clinicians and epidemiologists alike have recommended collecting information about a child from a range of informants: the parents, siblings, teachers, and peers, as well as the child. Most diagnostic instruments, whether questionnaires or interviews, exist in forms for diverse informants, with scoring algorithms that allow a diagnosis to be made on the basis of one informant or more. In the latter case, most follow the rule that clinicians generally observe, of counting a symptom as present if reported by any informant, rather than expecting agreement among informants, which rarely occurs.[18] Rates of psychiatric disorder will vary with the number of informants, and also depending on which informants report on which diagnoses. For example, across repeated assessments of 1420 children and adolescents, only 26 per cent of those with a diagnosis from the child interview also had one from the parent interview, and only 22 per cent of those with a diagnosis based on the parent interview had one from the child interview. This was statistically a highly significant level of agreement (OR 5.4, 95 per cent; CI 3.6, 7.9; $p < .0001$), but nevertheless only 13.5 per cent of cases were reported by both informants. Readers of epidemiological studies need to decide for themselves how much the number and type of informant matters in judging the accuracy of a prevalence estimate of a specific disorder. For example, parents often do not know much about their children's drug use, while young children themselves generally have little insight into their own hyperactivity, and teachers seldom notice children's depression. Prevalence rates based solely on these informants would be likely to be quite low.

3 *The age and sex of the subjects*. The prevalence rates of different disorders vary markedly by age, sex, and age-by-sex across childhood and adolescence. For example, a meta-analysis of 26 studies of child and adolescent depression[2] estimated the prevalence of adolescent depression (5.6 per cent) as twice that of childhood depression (2.9 per cent), and that of adolescent girls (5.9 per cent) as significantly higher than that of adolescent boys (4.6 per cent). Figure 9.1.2.1 shows prevalence rates of any psychiatric disorder (dotted lines) from a representative population sample of 1420 youth assessed regularly between ages 9 and 21. It is clear that prevalence, even when measured over time in the same subjects, varies markedly with age. This is because some of the common disorders of childhood, such as functional enuresis and encopresis, ADHD, and separation anxiety, diminish as children grow up, but then later on the problems of adolescence and young adulthood, such as drug abuse and depression, take their place. Between about 11 and 14, when the disorders of childhood have faded and those of adulthood not yet appeared, relatively few children have disorders. Prevalence rates will also differ depending on the distribution of males and females in the sample. Boys are significantly more likely to have developmental disorders, enuresis and encopresis, and ADHD in the early years, and drug abuse in the later years. Although girls are more vulnerable to depression after puberty,[19] this does not have a large effect on the overall prevalence of psychiatric disorder.

4 *The inclusion of measures of functional impairment*. Most DSM-IV and ICD-10 diagnoses require that to be clinically significant, symptoms must have a harmful effect on patients' ability to

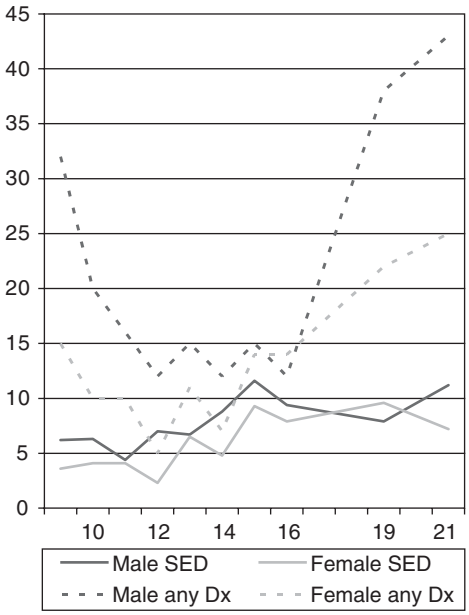

Fig. 9.1.2.1 (solid lines) shows the effect of applying a functioning criterion to diagnoses. It has the effect of flattening the U-shaped curve and revealing a doubling of psychiatric disorder with age, from around 5 per cent at age 9–10 to 10 per cent at age 21. This gradual increase is seen in both boys and girls.

function in their normal environments.[20] There is a wide range of measures of functional impairment,[20] and many diagnostic interviews employ measures of impairment as part of their diagnostic algorithms.

The inclusion or exclusion of a measure of impairment can make a dramatic difference to prevalence estimates. For example, version 2.3 of the Diagnostic Schedule for Children, a widely used interview for youth and parents, uses two measures of impairment. First, if a symptom is reported further questions are asked about whether it affects the child's functioning. Second, the interviewer scores the child on the Global Assessment Scale,[21] which rates the child's overall level of function from 0 to 100. Table 9.1.2.1 shows the impact on the prevalence of any anxiety disorder of including or excluding either or both of these measures of impairment, using data from a multi-site epidemiological study.[21] When no measure of impairment was used almost 40 per cent of subjects received an anxiety diagnosis. With both criteria applied at their most rigourous level, the prevalence of anxiety was cut to 3.2 per cent.

Future directions in the epidemiology of child and adolescent psychiatric disorders

Up to this point, the role of epidemiology has been mainly a descriptive one, addressing the basic questions: how many? who? where? when? However, child psychiatry is changing, and epidemiology will change as well. The goal is now to understand how risk exposure and vulnerability change over the life course, and how the requirements of 'normal' development shape the types of psychopathology that emerge if these requirements are not met. The term 'developmental epidemiology', first coined by Kellam in the 1970s,[22] is useful to describe what epidemiology is doing these days.

In this section we describe some rapidly growing research areas that will contribute to the next generation of studies, and will contribute to the shift from 'child psychiatric epidemiology' to 'developmental epidemiology'. We discuss the future under five headings: longitudinal research, genetic epidemiology, life course epidemiology, intergenerational epidemiology, and prevention science.

Longitudinal research

Although there have been many longitudinal developmental studies, some of them beginning at birth (or even before), longitudinal studies of psychiatric disorders had to await the development of appropriate technology; specifically, data collection methods that validly and reliably translated the psychiatric taxonomy into instruments that could be used repeatedly with the same subjects. Several of these have become available in the past 20 years.[23]

There are now several research groups that have used their longitudinal data to look at continuities and discontinuities in mental illness from childhood into adolescence and beyond. Some of the longitudinal studies have followed their subjects into adulthood.[16] These are beginning to show indications of continuity of disorder across childhood and adolescence,[24] and between temperamental characteristics in early childhood and the onset of psychiatric disorders in late adolescence and young adulthood.[25]

Genetic epidemiology

There have been two revolutions in genetic epidemiology in the past two decades that will have a tremendous impact on psychiatry in the next decade.

(a) Psychiatric–behavioural genetics

The first revolution occurred when the methods of psychiatric epidemiology were applied to behavioural genetics. Psychiatric interviews

Table 9.1.2.1 Effect of different rules for defining impairment on the per cent prevalence of any anxiety disorder (parent or child interview) using the DISC 2.3

	Diagnosis without diagnosis-specific impairment criteria				Diagnosis with diagnosis-specific impairment criteria			
	Criteria only	CGAS <= 70 (mild)	CGAS <= 60 (moderate)	CGAS <= 50 (severe)	Criteria only	CGAS <= 70 (mild)	CGAS <= 60 (moderate)	CGAS <= 50 (severe)
Any anxiety diagnosis	39.5	18.5	9.6	4.3	20.5	13.0	7.2	3.2

(Reproduced from D. Shaffer *et al.* The NIMH diagnostic interview schedule for children version 2.3 (DISC 2.3): description, acceptability, prevalence rates, and performance in the MECA study, *Journal of the American Academy of Child and Adolescent Psychiatry*, **35**, 865–77, copyright 1996, American Acadamy of Child and Adolescent Psychiatry, Lippincott Willams & Wilkins.)

DISC = Diagnostic Interview Schedule for Children. CGAS = Children's Global Assessment Scale.

like those described earlier were used in studies with genetically informative designs, such as twin, adoption, family, and migrant studies. For the first time, researchers examined categorical disorders such as depression, in ways that approximate clinical diagnosis. Furthermore, behavioural geneticists began to take seriously, issues of sampling, so that they could talk about the contribution of genes to disease in the population as a whole, rather than in highly selected families or groups. There have also been some longitudinal studies looking at how genes can have different effects at different developmental stages.[26]

(b) Molecular genetics

The second genetic revolution occurred when it became feasible to apply the methods of molecular genetics to epidemiologic samples. This development opens up the opportunity to use not only twin or adoption studies but a wide range of singleton samples to test theories about candidate genes for specific symptoms. Even more exciting is the new opportunity to use the treasure house of data from longitudinal studies to test for gene–environment interactions. Such studies can answer questions about which genes interact with which environmental factors, and at what developmental stage.[27,28]

Life course epidemiology

Life course epidemiology is the study of long-term effects on chronic disease risk, of physical and social exposures, during gestation, childhood, adolescence, young adulthood, and later adult life. It includes studies of the biological, behavioural, and psychosocial pathways that operate across an individual's life course, as well as across generations, to influence the development of chronic diseases.[29]

Life course epidemiology has developed a special concern with 'the "embodiment" of social phenomena into the biological'[30] encapsulated in the concept of 'health inequalities'. This concern arose historically from work showing that mortality from many diseases is spread unequally across the population and that these differences in risk can be linked to social inequalities that often go back to infancy or even to the parental generation. This body of work has had enormous significance for international thinking about social policy and is having a direct effect on the allocation of public resources in the United Kingdom and elsewhere.

Intergenerational epidemiology

A life course approach to epidemiology intertwines biological and social transmission of risk across generations, recognizing that geographical and secular characteristics may be unique to one cohort of individuals.[31,32]

Experiences of the previous generation can operate at many different levels of generality. They may be specific to the mother–child dyad (e.g. the effect of drug use during pregnancy), or may affect everyone living in a certain neighborhood (e.g. poverty, or exposure to an environmental toxin). All mothers and children maybe affected by a particular event, such as a period of famine or disease, or children may be affected by their mother's developmental stage (e.g. children of teen mothers or elderly mothers). Models for intergenerational research have recently appeared[33] and statistical methods have become more tractable.

Prevention science

Prevention science uses theory about the causes of disease to generate interventions, which when tested provide information not only about the effectiveness of the intervention, but also about the aetiology of the disease. Epidemiology traditionally divides prevention into thee categories, depending on the mean level of risk in the population of concern. Programmes available to all, like clean water, car seat belts, and parental leave programmes, are examples of *primary* or *universal* prevention. For example, the 'Just Say No' drug abstinence programme was introduced as a primary prevention for all children in school, designed to stop drug use before it began. Unfortunately, the results were neutral if not negative.[34] On the other hand, primary prevention with both children[35] and *families*[36] can be both effective, and suggest aetiologic pathways that could be explored in further research.

Secondary intervention *programmes* are based on high-risk children, schools, or communities. Many of them are both theory-driven and scientifically sound. A good example of a secondary intervention that yields insights for epidemiology is the 'Fast Track' programme for aggressive children in grade school. This was based on clearly articulated theory about cognitive difficulties that could interact with environmental risk to produce aggressive behaviour in socially ambiguous situations.[37] Hostile attributional bias was indeed found to be a partial mediator of the effect of the intervention on reductions in aggressive behaviour.

Once children have developed clinically defined psychiatric disorders, interventions tend at present to focus on clinical treatment rather than tertiary *prevention*. Tertiary prevention programmes are rare. One example of proven effectiveness is Multisystemic Therapy.[38] Given the early onset of most psychiatric disorders, this is clearly a vitally important area for future work.

Conclusions

This chapter has covered a lot of ground; from the first stirrings of understanding about childhood psychiatric disorders to the possibility of using molecular genetics to identify gene–environment interactions that can generate psychiatric disorder. There are fuzzy boundaries between epidemiology and developmental psychopathology, life course epidemiology, genetic epidemiology, services research, and clinical psychiatry. It will be important to keep these boundaries pervious, to share a common language where possible, and to learn and use one another's methods.

Further information

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9.1.3 Assessment in child and adolescent psychiatry

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The goals of assessment of a child/adolescent are to (1) detect psychopathology and its impacts on the child's functioning in family, school, and peer domains, (2) allow appropriate intervention targets to be identified and prioritized; and (3) identify relevant variables, including family or school factors that may influence treatment adherence.

Distinctive aspects of the psychiatric assessment in children

1 Parents (or other adults) ordinarily initiate and pursue the evaluation of the child for diverse reasons. Adult expectations for the child sometimes exceed the child's abilities, or the adult's own parenting or teaching style may be a poor fit with this child. Some adults may seek treatment to alter the child to remedy this poor fit.

2 Children may not be receptive to changing their behaviour. Children may attribute problems to others and be unable to accept their contribution to an identified problem. The psychiatric assessment of children requires attention to what the child wishes would change.

3 Young children may not trust unfamiliar adults (including clinicians), and adolescents may perceive the clinician as another adult imposing expectations or judgements. Multiple informants[1] are often needed to identify the child's functioning in school, home, and peer domains, to identify the child's areas of strength on which the clinician can build, and to identify others (peers or adults) able to introduce or reinforce more adaptive skills or behaviours.

4 Most DSM-IV-TR diagnoses were defined amongst adult samples.[2] Efforts to consider where a particular child fits on the depressed mood, anxiety, and aggression axes, for example, requires attention to developmental differences in symptom expression.

5 The ability of the clinician to forge alliances with the child, the parent, and outside entities is essential. A breach in any of these relationships can impede treatment. Parental permission should be obtained to contact and collaborate with relevant parties.

Content of the clinical interview

Reason for referral

Who initiated this referral, their motivations, and what changes they seek is vital. Expectations of various parties may collide and must be reconciled for effective treatments to be implemented. For example, the school may seek changes in parental discipline, while parents may expect the evaluation to yield additional school services.

History of problem(s)

Parents often experience intense pain while recounting the deterioration or anguish of their child. Clinicians should provide parents an opportunity to describe the evolution of the problem, attending to the context in which symptoms emerged and occur, changes in frequency and intensity of symptoms, and their current progression. The clinician should inquire directly about the *functions* of problem behaviours, including secondary gains (e.g. tantrums diminish chore requirements, etc.). The clinician should clarify whether symptoms are specific to one functional domain or whether they pervade multiple areas of the child's functioning at home, school, and with peers.

Past problems

Significant past symptoms impairing the child should be identified. It is especially important to understand whether symptoms have been persistent since early childhood, are intermittent, or represent deterioration from a previously better level of functioning.

Comorbid problems

Clinicians should inquire about disorders often seen in tandem. For example, bipolar disorder in children is often associated with previous attention deficit hyperactivity disorder.[3] Screening instruments (such as those selectively available free of cost at websites such as www.schoolpsychiatry.org) can be useful to provide comprehensive information about less conspicuous symptoms.

Substance use history

Clinicians should inquire about the child's exposure to and use of tobacco, alcohol, and illicit substances. Children may perceive that substances alleviate their distress (e.g. anxiety, depression) and 'self-medicate.' Clarifying impacts of substances on symptoms may yield intervention points attractive to the child.

Previous treatment(s)

Chronological assessment of past treatments may reveal strategies adaptable to the current problem. Past treatment history may suggest treatment modalities (in)tolerable to this patient (and family). Medication trials, counselling, hospitalizations, or alternative treatments should be explored.

Developmental history

Parents may vary in their recollection of their child's attainment of developmental milestones. Review of earlier videotapes of the child may improve the reliability and completeness of reports regarding the sequence of the child's growth.

The child's development regulating *sleep, eating,* and *toileting* should be investigated. Attained skills may suddenly be lost, sometimes signalling the importance of emotional events at particular times. Eating behaviour has become complicated as both hunger and obesity increase risks of psychopathology.[4,5]

Psychomotor development includes standing, walking, running, throwing, hopping, and playing sports or musical instruments. How the child fares at sports may clarify psychomotor skills. Fine motor and gross motor skills may not be congruent.

Cognitive development refers to the child's acquisition of thinking skills. Specific inquiry concerning speech development, reading, writing, and math skill progression may reveal global or specific difficulties.

Interpersonal development refers to how the child interacts with others, particularly family members and other children and adults.

Stability of relationships, numbers of friends, types of activities shared, and expectations of peers often reveal sources of difficulty or maladaptive patterns.

Emotional development and *temperament* reveal the child's capacity to recognize his or her own mood state and to self-soothe or regulate negative affect. Prevailing moods can be described by parents, who may also detail past suicidality, irritability, specific fears and anxieties, and conditions associated with the child's happiness and pleasure.

The child's *moral development* indicates whether conscience or moral values are too lax, too harsh, overly focused on particular areas, or uneven and out of proportion to daily events. The child's ability to recognize impacts of decisions on others, and to acknowledge and correct mistakes provides clarity about the child's strengths and limitations. The child's religious and cultural/ethical views and practices also shape this area, and may guide treatment interventions.

Trauma may impact or even arrest development. Investigation of actual events (such as documented abuse), but also of events perceived traumatic by the child and family may shed important light on the child's behaviours and patterns of relating to others. Events surrounding the trauma, disclosures to others, and reactions of adults are also important for the clinician to recognize and address.

Harmful behaviour, towards self or others, may reveal important developmental progressions that warrant intervention. Head-banging may reveal sensory disturbances, thoughts or comments about death may reveal suicidality, and self-harmful acts such as self-mutilation or cutting may reveal primitive coping mechanisms.[6] Harmful acts towards animals or people may indicate needs for monitoring while other diagnostic or treatment interventions occur.

Family history

Few psychiatric disorders appear transmitted exclusively genetically. Many parents fear that their other child may be destined to suffer psychopathology when a family member manifests a disorder, so clarification of contributions to expression of disorders can reduce unwarranted fear, guilt, and distress. Please refer to Chapter 6.3.8 for more information on assessment of family functioning.

Divorce, separation, and single-parent family circumstances may stress all family members. Even when parents part amicably, children may attempt to reunite family members. Children may exhibit symptoms even years after separations as they enter different developmental phases.[7]

Adoption may be a positive event for the child, and adoption warrant tactful attention by the clinician, including age at adoption of the child and biological parents, the involvement with biological parents, the child's understanding of the adoption, and how the adoption is discussed at home.

Medical history

Pregnancy complications, birth difficulties, hospital stays, and medical illnesses requiring treatments (e.g. asthma, diabetes) should be investigated, as they increase the child's risk for psychopathology.[8] Inquiry into emergency room visits or surgeries can shed light on the child's fears, or parental over/underprotectiveness. Allergies should be ascertained, as well as responses or side effects to medications, including naturopathic or homeopathic agents.

Child strengths/weaknesses

Interests, hobbies, and talents of the child should be obtained from the child and parents. Parents may have aspirations the child does not share, or the child may have fantasies beyond apparent abilities. In most cases, though, the child will have some identifiable interests or abilities that serve as potential points of connection with peers and adults (including clinicians).

The child's media diet

Children are exposed to television, music, videos, electronic games, cell phones, e-mail and instant messaging, personal digital assistants, etc. It is important to clarify which media the child uses, how much time each day is spent with these various media, and what consequences these media have on the child (e.g. in response to watching action TV show the child becomes more violent versus has developed interest in Asian food through watching cooking programmes).[9] The degree of parental awareness and appropriate limit-setting regarding TV, video games, and instant messaging may warrant intervention.

Mental status examination (MSE)

The MSE must be adjusted for children (see Fig. 9.1.3.1). The MSE includes a clinical description of the child's appearance, mood, sensorium, intelligence, and thought content and process. Much of the MSE takes place implicitly as the clinician interacts and observes the child during the child and family interviews.

Structure of the clinical interview

Preparatory phase of the child interview

Unlike regular pediatric check-ups, the psychiatric evaluation usually occurs because of prominent symptoms often perceived as embarrassing by the parents or the child. A phone call before the interview by the clinician or staff can clarify the structure of the interview, the collaboration anticipated to devise solutions, and the opportunity for parents to provide any confidential information to the clinician.

The parent interview

The parent interview can be complicated by parental ambivalence about having a child evaluated by a psychiatrist, fears of loss of control or criticism, or parental shame or embarrassment about perceived parenting faults. The clinician should remain sensitive throughout the interview to parent vulnerabilities. Techniques to help parents overcome such obstacles during the interview are summarized in Fig. 9.1.3.2.

The developmentally sensitive clinical interview of the child

The interview process and wording of questions must be tailored to fit with the child's understanding.[10] The child may not understand terms necessary to answer questions accurately. The child may also provide misleading answers to shield other family members, to protect against acknowledging some perceived failing, or to address circumstances if the child fears it might entail placement out of the home. Please refer to Chapter 9.1.1 for more specific information for obtaining reliable information during the child interview.

Category	Components	What to Assess
Appearance	Physical Appearance	Gender; ethnicity; age (actual and apparent); cleanliness and grooming, hair/clothing style, presence of physical anomalies, indicators of self-care and parental attentiveness
	Manner of Relating to Clinician and Parents	Ease of separation from parent, guardedness, defiance, eagerness to please, flirtatiousness
	Activity Level	Psychomotor retarded to agitated, sustained or episodic, goal-oriented or erratic; coordination, unusual postures or motor patterns (e.g., tics, stereotypies, compulsions, catatonia, akathisia, dystonia, tremors)
	Speech	Fluency (including stuttering, cluttering, speech impediments), rate, volume, prosody
Mood	Current Affect	Predominant emotion and range (constricted to labile) during the interview, and appropriateness to content (e.g., giggles while talks about sibling's illness); intensity; lability
	Persisting Mood	Predominant emotion over days/weeks; whether current affect unusual or consistent with mood; whether mood reactive to situations or same across range of situations
	Coping Mechanisms and Regulation of Affect	How child manages conflict or distress, age-appropriateness of responses to and dependency on parents; sexual interests, impulses, aggression; control or modulation of urges (finding alternative or socially appropriate means of satisfying urges); how deals with frustration or when anxious
Sensorium	Orientation	Self (name), place (town, State), time (awareness of morning, day of week, month, year varies by age), situation (why at this appointment)
Intellect/ Cognition	Attention	Need for repeating, how long sustained on activity, degree to which child shifts from activity to activity, distractibility (to outside noises, etc.)
	Memory	Immediate (repeat numbers, names back), short-term (recall 3 objects at 2 and 5 minutes), long-term (recall events of past week)
	Intelligence; Fund of Knowledge	Age-appropriate recognition of letters, vocabulary, reading, counting, computational skills; age-appropriate knowledge of geography, history, culture (celebrities, sports, movies, etc.); concrete to abstract thinking, ability to classify and categorize
	Judgment	Best assessed after rapport established, as initially minimization or denial more common); what would do if found stamped envelope next to mailbox, fire started in theater, say if saw man with big feet
	Insight	Ability to see alternative explanations, others' points of view; locus of control (internal v. external); defense mechanisms
Thought	Process: *Coherence*	Logical, goal-directed, circumstantial or tangential (consider age-appropriateness), looseness of associations, word salad (incoherent, clanging, neologisms)
	Process: *Speed*	Mutism, poverty of thought (long latency, thought blocking), poverty of content (perseveration), racing thoughts, flight of ideas
	Perceptions	Altered bodily experiences (depersonalization, derealization), misperception of stimulus (illusion), no stimulus (hallucination: auditory [psychosis > PTSD > organic causes], visual [substance use, delirium], olfactory (neurological, seizure disorder] gustatory [from medicine side effects])
	Content	Obsessions (ego-dystonic), delusions (ego-syntonic), thoughts of harm to self or others (magical thinking, or fears at night often age appropriate)

Fig. 9.1.3.1 The mental status examination in children.

1 Forming a clinical alliance with parents
- (a) Facilitating Narrative History
  Open-ended questions allow parents control, and can be followed with narrow questions to fill in needed details. Using the parent's own words can help parents feel heard.
- (b) Finding Common Themes/Patterns
  Inquiry into problems or conflicts the child has with other adults, peers, or unfamiliar others may illuminate patterns of the child's behavior that play out in a variety of settings, decreasing parents' anxiety that they alone provoke the child's problem.
- (c) Finding Good Intentions Gone Awry
  Parents may feel ashamed of past parenting efforts done in desperation.
  Acknowledging the parent's good intention leading to a misguided effort can diminish self-reproach. For example, a parent's harsh response often belies a fear about the child's future behavior, so identifying the fear and then examining alternative responses can be productive).
- (d) Partnering with Parents (Clinician as "partner" in decision-making process)
  Clinicians increasingly serve as partners, outlining several appropriate treatments, risks, and side effects, and helping parents to choose and invest in preferred treatments. If parents propose treatments the clinician regards as unhealthy or unproven, the clinician can identify potential risks of such treatments to minimize risks to the child.
- (e) Clarifying Expectations of the Evaluation
  Parents sometimes have unrealistic fantasies about what the evaluation will accomplish. Inquiring early about what the parent hopes will be accomplished by this evaluation can reveal such expectations and fantasies, which the clinician can realistically address. For example, parents may believe the evaluation can definitively prove the child had been abused by someone. At the other extreme, parents may fear that the clinician will tell them that their child will never be normal, will require institutionalization, or ultimately harm others.

2 Eliciting Sensitive Information
- (a) Providing the Parent Opportunities to Convey Sensitive Information
  Apprising parents of times and methods to convey information can provide appropriate mechanisms for sharing of information.
- (b) Revisiting Sensitive Information at Safer Points
  If parents resist disclosing information, the clinician should not force answers (as they are more likely to be inaccurate or incomplete), but rather proceed to less distressing information.
- (c) Explaining the Purpose of Sensitive Information
  Some parents may need to understand the underlying reasons for inquiring about personal information. For example, the clinician may need to explain the need to inquire about relatives to clarify genetic contributions to the child's difficulties.
- (d) Describing How Sensitive Information Will Be Reported
  Parents are sometimes fearful that details of embarrassing past parental personal problems may be included in reports to be seen by others. Parents may fear that marital conflict information might be used to alter custody arrangements, or symptoms in a report that could jeopardize their child's future educational or occupational pursuits. Clarifying that general information will be provided ("history of substance abuse on maternal side") rather than specifics and that parents will be able to review reports whose release they authorize can diminish resistance to sharing sensitive information.

3 Handling Discrepant Reports
- (a) Contextualizing Points of View
  Differences between observers' descriptions of a child's behavior have several potential sources. For example, teachers sometimes report very different presentations than parents. Examining what precipitates the child's problem, and how it expresses itself in different environments may allow clinicians to borrow effective strategies across environments without "blaming" adults.
- (b) Aligning Different Perspectives
  When parents or adults exhibit conflict during the psychiatric evaluation, the clinician may continue to refocus adults to the child's needs. For example, the clinician may encourage "middle ground" approaches to increase consistency between environments.

Fig. 9.1.3.2 Parent interview techniques.

The child's understanding of the psychiatric interview

The child and parent are usually seen together at the beginning of the child psychiatric interview to put the child at ease. Once comfortable, the child usually can tolerate the parents leaving the room. Transitional objects (books, electronic devices from home) may ease these transitions. Inquiring about what the child believes parents, teachers, or other adults want to be different as a result of this interview often elucidates what the child recognizes about others' perspectives, and also facilitates the child projecting thoughts or fantasies about this evaluation.

Adolescents sometimes fear parents will skew the interview by telling their 'version' first to get the clinician to side against the adolescent.[11] Meeting briefly with the parent and adolescent to clarify objectives, and then meeting with the adolescent alone at length may enhance an alliance with an adolescent. During this initial segment the clinician can clarify the plan to meet alone with parents after meeting with the adolescent to review birth history, developmental milestones, and family.

Adolescents may resist answering questions or participating. Clinicians can identify the adolescent's priorities and side with those that are reasonable, or identify what the adolescent needs to do to satisfy parents so that the adolescent no longer needs to see a psychiatrist. Clinicians may also decrease resistance by inquiring first about the adolescent's interests, strengths, musical preferences,

rather than focusing on their 'problems,' as adolescents are developmentally struggling with their identity, and may resist fitting into the 'psychiatric patient' category.

Developmentally sensitive techniques for the psychiatric interview

Four categories of techniques are commonly employed in these interviews. *Engagement* techniques are often required to put the child at ease so that the child will provide accurate clinical information. *Projective* techniques allow the child to reveal underlying themes or issues which cannot be verbalized directly. *Direct questioning* techniques clarify particular points needed to distinguish disorders, contributions to the child's problems, and intervention options. *Interactive* techniques clarify how the child relates to, as well as accepts or integrates input from, others.

Techniques to engage the child

Child psychiatrists often provide toys or objects for patients in the waiting room and office. Toy figures, puppets, and 'relationship-oriented' toys may ease the child into the interview. Generic toy figures are usually preferable, since they are more likely to evoke the child's specific themes and concerns rather than 'scripts' based on TV shows or movies. Tasks framed as 'games' or active (e.g. drawing a house or family) often help the child transition into the psychiatric interview. By allowing the child to direct the content, the interviewer can follow the sequence of the child's concerns, note themes that emerge, and observe the points at which a child avoids or shifts to a new topic.

With *adolescents*, efforts to indicate familiarity with contemporary adolescent tastes (music, movies, terms, etc.) can be perceived ingenuine by the adolescent. Instead, clinicians may inquire about current interests, musical preferences, and current adolescent values from a curious, 'help me understand it' perspective, rather than from one of 'trying to be hip.' Manipulable items (squeeze balls, modelling clay, finger cuffs, cards, etc.) may allow adolescents a socially acceptable option for keeping their hands busy so that the interview feels less like an interrogation.

Projective techniques

Projective techniques may help the child express concerns indirectly, so that anxiety about significant fears, telling family secrets, or betraying loyalties is minimized. Common projective techniques include having the child draw a picture of him- or herself or family doing something. For pictures of the child, body details including sizes of appendages or body parts and articulation (fingers, toes), relative size of the figure to the page, and frequent erasures can all reveal underlying issues of anxiety, perceived agency to address difficulties, or needs to control the environment. Depictions of the self as non-human, grotesque, imbued with super powers, or of the opposite gender may provide clues about the child's self-image and underlying wishes. The relative size and placement or omission of family members in a family drawing may illuminate the child's feelings about family relationships. Aggressive or sexual themes may be revealed in drawings.

Verbal projective techniques can similarly yield important information. Asking what animal or character (TV/movie star, cartoon, superhero) the child would most like to be, or whom the child would take along to a deserted island, or asking what the child would do with three magic wishes often allow underlying issues to emerge. Wishes may reveal basic needs, such as food or a safe place to live, or longings for parents to reunite or for the return of a departed friend. Wishes sometimes reveal specific desires, such as 'not to have tics anymore,' or 'never to get teased.' Very general or altruistic wishes, such as 'world peace' or 'to live in a big house with lots of money' warrant further exploration, such as 'Are there particular fights you would especially like to stop?' or 'Who else would live there?' and 'What would you do first with lots of money?'

Projective techniques may help *adolescents* to reveal and share emotionally significant concerns with the clinician. Inquiries into favourite, or most disliked, movies, television characters, political or historical figures, musicians or artists, or sports figures, all allow elaboration of the teenager's ideas in displacement. Adolescents less distrustful of the clinician may readily speak about their own social longings or anxieties regarding friends at school. Adolescent resistances are often revealed by reluctance to divulge names of friends, or even questions about why the clinician needs to know this information. If resistance is detected, questions about what the adolescent most admires about a character, or what the adolescent imagines this character would do in given situations may reveal the adolescent's perceptions. Asking the adolescent about the different cliques or groups at school and his or her relationship to them provides useful information about the teen's self-image. Similarly, questions about what the adolescent sees as fair or would most like to change about school or the world often reveals underlying concerns and issues.

Direct questioning

Direct questioning can specify symptoms or events, clarify how the child sees the world and functions within it, and follow-up on material from other parts of the evaluation. Asking the child to describe friends ('Tell me about your best friend.'), siblings, or parents, is preferable to 'Do you get along with your brother?' Open-ended questions such as 'What sorts of things make you mad/afraid/happy?' and 'What do you daydream about?' are similarly preferable to 'Do you get mad?' or 'Do you ever daydream?'

Anchoring direct questions to major events may help children provide more accurate answers. For example, 'Did that happen before or after your birthday?' or 'How has that (problem) been since school ended?' improve respondent accuracy.

Substance abuse, sexuality, and risky behaviours are often assessed through direct questions. The clinician can use simple questions, such as 'Substance use?' that allow significant latitude, and then focus in further, contingent on the child's responses. For example, the clinician may hear 'No, I don't do any of that anymore,' which could then be followed by 'What led to that decision?' and then proceed back to when and what substances were used. Similarly, sexuality can be assessed by gentle direct questions that do not prematurely close off response options, such as 'Have you had romantic feelings towards another? How did that go?' (rather than 'Have you had a boyfriend yet?'). Adolescents may fear the interviewer will be disapproving, so questions like 'romantic feelings towards anyone' are preferable to 'are there any girls you like?'[12] Finally, direct inquiries into risky behaviours (stealing, vandalism, assaults, gambling, etc.) often require general questions such as 'Have you done anything that you now look back on and think was dangerous?' before proceeding to specific questions (e.g. 'Have you ever stolen anything? Have you ever been beaten up?

Beat up someone else?'). Suicidal risk behaviours may be minimized or trivialized, so additional questions to examine fantasies about impacts of the suicide on family and friends or value contradictions may be needed to clarify suicidality risks.[13]

Interactive techniques

Throughout, the clinician observes how the child relates to another person and what feelings or reactions this child elicits. How the child reacts to a new person, sustains interactions, and terminates the interview often reveal patterns important in the child's larger social life. The clinician can evaluate more complex social interactions during transitions ('It's time to put these toys up in the box.') and during games. Short games (tic tac toe) are useful since the clinician can quickly detect the child's response to winning, tying, and losing.

Adolescents employ more complex patterns, often specific to a subgroup to which they now belong, so clarifying what clothing symbols represent, meanings of confusing terms, and values espoused by subgroups can clarify how the adolescent relates to others. The clinician should observe provocative comments, often used to titrate space between the clinician and the adolescent, or to reject others first.

Concluding the interview

Ending collaboratively increases the likelihood that the child will feel positive about subsequent encounters with clinicians, including treatment. Questions such as 'Are there other things that would be important for me to know about what you're like or how things have been for you?' or 'What else have I not asked about that is important?' facilitate this process.

The child may be curious about what the clinician will say and to whom. The interview is one piece of a larger evaluation, so the clinician may need to clarify that other testing, conversations with others, or additional meetings may be needed. Discussing findings (including treatment recommendations) with parents is usually advisable since parents may disagree with the clinician's conclusions or resist suggested interventions (e.g. medication, school placement, etc.).

Confidentiality is one of the most challenging issues surrounding child psychiatric interviews, especially with adolescents. Describing to the adolescent what will be told to specific others is helpful, as well as what information will not be revealed (e.g. specific details about substance abuse or sexual behaviours). Parents and the child should be told explicitly that confidentiality does *not* extend to situations that pose a clear danger to the child or others. In cases where dangerous content emerges (e.g. the child describes obtaining bullets to frighten a peer), the clinician should clarify with the child *how* they will tell appropriate others, preferably together.

Neuropsychological testing

Patients may have subtle or complicated difficulties processing certain types of information. Consultation with a pediatric neuropsychologist may clarify appropriate tests to address persisting diagnostic questions. Clinicians should recognize that young patients may not be 'interested' in testing tasks, so scores should be interpreted cautiously, with input from the person who did the testing, when the clinician discusses findings with families.

Laboratory evaluation in the child psychiatric evaluation

Few definitive clinical tests identify specific child psychiatric disorders. Laboratory testing remains useful when symptoms and physical findings suggest a particular disorder. Collaboration with the primary pediatric care provider may guide decisions about possible further medical consultations (e.g. audiometric, genetic, neurological, speech, etc.) or diagnostic tests (e.g. blood tests, neuroimaging, sleep studies).

Testing in specific childhood disorders

Laboratory testing yields findings that alter the working diagnosis in approximately 1 per cent of cases, and the yield for laboratory abnormalities, without the presence of other supportive physical findings, remains less than 5 per cent.[14] Laboratory tests commonly considered are summarized in Table 9.1.3.1. Specialized technologies, such as positron emission tomography (PET), single photon emission computerized tomography (SPECT), functional

Table 9.1.3.1 Laboratory tests to consider in childhood psychiatric disorders

Lab test	Disorder					
	MR/PDD	Mood	Psychosis	OCD tics	Substance abuse	Eating disorders
Chromosomal testing	X		X			
Wood's (UV) lamp	X					
Monospot		X				
Thyroid		X	X			X
Lyme titre		X				
CBC	X	X	X		X	X
Serum chemistry	X	X	X		X	X
Lead level	X					
Throat culture antistreptolysin O antibody (ASO), antideoxyribonuclease B titres				X		
Urine drug screen		X	X		X	
Cerebrospinal fluid analysis		X	X			
Neuroimaging			X			
EEG	X					

MRI (fMRI), and brain electrical activity mapping (BEAM) remain attractive research tools at this time in child psychiatry.

Further information

Recommended Websites:

www.aacap.org: the home site for American child psychiatry; includes current practice parameters for various psychiatric disorders.

www.schoolpsychiatry.org: rating scales, school interventions for psychiatric symptoms.

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9.1.4 Prevention of mental disorder in childhood and other public health issues

Rhoshel Lenroot

Introduction

Over the last two decades advances in psychiatric classification systems and screening tools have allowed the global and national burden of mental disorder to be described with the first large-scale epidemiologic studies. The World Health Organization's *World Health Report 2001* estimated that over 450 million individuals suffer from mental disorders, and that psychiatric disorders ranked as 5 of the top 10 causes of disability in the global population.[1] Studies specifically of psychiatric disorders in children report that between 3 per cent and 18 per cent of children have a clinically significant psychiatric disorder, a number far exceeding those with access to treatment.[2] A recent study which included data on age of onset found that 50 per cent of psychiatric disorders had their onset by age 14, and 75 per cent by age 24.[3] Treatment on this scale is unlikely to ever be feasible, even if available methods were more effective and less risky than those currently available. Preventing mental health disorders from occurring is an alternative to decrease the extent of this public health problem. However, if a key characteristic of prevention is acting prior to onset of a disorder, the early age of onset for most mental disorders indicates intervention must occur during long before adulthood.

Neuroscience has contributed evidence that longitudinal trajectories of brain development are affected by a combination of genetic and environmental factors. Neuroimaging studies have shown dynamic changes in brain structure and function continuing through childhood and adolescence, and geneticists have found that gene expression is highly dependent on environmental conditions. These findings imply that the brain is still highly plastic during childhood and adolescence. This may confer greater vulnerability to long-term effects of insults from trauma, substance abuse, or other adverse influences than in adulthood, but also the potential for lifelong beneficial effects from early positive interventions.

Growing interest in the possibilities afforded by research into prevention in children's mental health stimulated a series of large-scale reports and initiatives beginning in the early 1990s.[4–7] Advances in epidemiology, developmental psychopathology, and prevention science have converged to provide a framework to guide and evaluate prevention programmes. This chapter will discuss basic principles of public health and preventive medicine with application to mental health disorders in children and adolescents.

Public health and prevention: history and basic concepts

The goal of public health is the prevention of disease and promotion of health in communities. The World Health Organization has defined *health* as 'a state of physical, mental and social well-being and not merely the absence of disease or infirmity',[8] and *mental health* as 'a state of well-being in which the individual realizes his or

her own abilities, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to his or her community'.[1] Public health differs from clinical medicine in that it addresses health-related matters on the level of populations rather than individuals. Public health activities include assessment of the health status and risk factors within a community through epidemiology, and population-focused interventions such as supporting the practice of preventive medicine, health education and behavioural modifications, creating and enforcing measures to maintain a healthy environment, and working to increase support for public health initiatives within the political sphere. In countries without universal access to health care public health offices may also act as providers of medical treatment for individuals without other means of access.[9]

Communities have acted to support the health of their members throughout history.[10] Common concerns for most societies have included control of epidemics, public sanitation, and promotion of personal hygiene, although the forms of public health interventions have varied depending on societal values, conceptions of the causes of ill health, and available resources. The health risks posed by the large-scale urban poverty and overcrowding associated with the industrial revolution helped to stimulate the growth of modern public health organizations, whose concerns eventually broadened to include issues such as workplace safety and regulation of the production of foods and medicines. Public health interventions changed to reflect advances in the understanding of disease processes, for example moving from general notions of the value of sanitation to focusing on specific infectious agents. Measures such as widespread vaccination and regulation of sanitary conditions have been so effective in developed countries that the focus of public health in these areas has shifted to chronic disorders such as heart disease and hypertension. Although emotional and behavioural issues have always been a concern of communities, systematic intervention to prevent mental disorders has lagged behind other disorders, in large part because of the lack of consensus regarding the nature of these problems or even how to classify them. A key factor in the advances in public mental health of the past several decades has been progress in epidemiology of mental health disorders.[9]

Epidemiology in public mental health

Incidence and prevalence

Epidemiology provides information about the *incidence* of a condition, meaning the number of new cases, which arise during a certain period of time, and its *prevalence*, meaning how many individuals have the condition during a certain period. The goal of prevention is to decrease a condition's incidence, i.e. prevent new cases from occurring, while successful treatment results in the decrease of the prevalence. Mental health disorders have presented challenges to epidemiology on several levels. In order to determine how many cases of a certain condition exist within the population, it is necessary to know how to define a case, but this is far from straightforward in the realm of mental health. Classification of medical disorders tends to evolve from symptom-based to mechanism-based as the links between a specific pathophysiology and the observed signs and symptoms are established. The lack of knowledge about the mechanisms producing cognitive and behavioural symptoms means that classification of mental disorders still relies upon descriptions of constellations of symptoms. *The International Classification of Disease version 10*(*ICD-10*),[11] and its United States counterpart *the Diagnostic and Statistical Manual TR-IV* (*DSM-TR-IV*),[12] are the results of iterative attempts by experts in the field to create meaningful classifications of psychiatric disorders based upon such observations in conjunction with applicable considerations of length and severity of illness, age of onset, and risk factors. This work has provided the standardized terminology that made possible the first large-scale epidemiologic descriptions of mental disorders. However, problematic issues pertinent to epidemiology remain, including questions regarding the relative merits of categorical versus dimensional classification systems; how to interpret the high rate of comorbidities for several disorders; and how best to account for individuals who have subthreshold symptoms, including how to determine the starting point of a disorder. It is not uncommon for individuals who have come to meet criteria for a mental health disorder such as schizophrenia or depression to have had a preceding period of subthreshold 'prodromal' symptoms, but healthy individuals also have occasional subthreshold symptoms that resolve without intervention. Unfortunately this differentiation often cannot be determined except retrospectively, despite the fact that there may be different implications for epidemiologic and preventive efforts.

The question of how symptoms change over time gains additional relevance when attempting to describe the epidemiology of mental health disorders in children and adolescents. As described in more detail elsewhere in this volume,[13] the science of developmental epidemiology has arisen as a response to the recognition that mental disorders may manifest in different ways over the lifespan, and that certain types of symptoms at one age may indicate that an individual is at high risk for developing a different disorder at a later stage of maturation. Risk factors may also have differing impact depending on an individual's developmental stage. Function may appear impaired if children are developing slowly in comparison with their peers, and it must be decided when this is normal variation and when it should be considered pathological. An additional layer of complexity in epidemiology in paediatric populations is the incorporation of information from additional informants such as parents and/or teachers, and determining how to evaluate the relationship of symptoms to particular contexts.

Risk factors

Epidemiology is also used to assess for the presence of risk factors. *Fixed risk factors* are those that cannot be altered, such as genotype. *Malleable risk factors* are susceptible to intervention, such as exposure to lead-based paint or domestic violence. *Causative risk factors* are those with known relationships to a particular outcome, and are of particular interest to prevention because they represent potential points of intervention. *Protective factors* instead decrease the risk of an adverse outcome. *Resilience* is a term used to describe an individual's ability to do well despite exposure to a typically high-risk situation.

Effective intervention to decrease risk factors or increase protective factors requires determining how these factors relate to each other and to the targeted health issues. The ultimate goal is a chain of causative steps leading from risk factor to outcome, but epidemiological data itself may provide sufficient guidance for action. One of the most famous examples of this was John Snow's identification of tainted drinking water from a particular well as the root

of a cholera epidemic in London, which he did based solely on epidemiological observations. Removal of the pump handle stopped the epidemic and proved that exposure was a causative risk, decades before the bacteria itself was identified. We are currently in a similar situation to Snow in regards to connecting risk factors to mechanisms for many mental health disorders, with the additional complication that mental health disorders are typically associated with combinations of a large number of individually modest potential risk factors.

Risk factors can be classified in terms of how they relate to each other and to the specified outcome,[14] and thus what type of intervention if any is appropriate. *Mediating* risk factors are those which explain how or why another factor affects the outcome; for example, the phenylketonuria enzyme *mediates* the effects of the phenylketonuria gene on IQ.[14] Although all causal factors are mediators, the reverse is not true, and experimental conditions are generally necessary to demonstrate that a particular mediator plays a causal role. A *moderating* risk factor instead specifies under what conditions or for whom another risk factor will affect outcome. Moderating risk factors describe populations that have differing responses to a given exposure, and may also represent potential sites of intervention to prevent an adverse outcome by reducing vulnerability or increasing resilience. A *proxy* risk factor, also called a *pseudocorrelation*, is one that itself does not strongly predict outcome but is highly correlated to a risk factor that does. *Overlapping* risk factors are those that arise from the same underlying construct and are observed to equally predict outcome, be highly correlated with each other and not stand in a specific temporal relationship; these can often be combined into a single factor. *Independent* risk factors conversely are unrelated to each other; they both predict outcome but without correlation or temporal precedence.

Theoretical models in prevention

The identification of risk factors and their interpretation evolves together with theoretical models for the causes and treatments of health problems. The fundamental model used throughout public health and epidemiology is that of *host-agent-environment*, in which the *host* is the person affected or at risk, the *agent* is the direct cause of disease, and the *environment* includes external factors which affects the host's vulnerability to the agent and the vector by which the agent reaches the host. While this model was first developed for infectious disease, it has been expanded to include other types of chronic non-infectious disorders.[10] Examples of pathogenic agents in the latter case include nutrition, chemicals, and genes; host factors include age, sex, and lifestyle; while social or economic issues are among those potentially affecting the environment. Another dimension that has gained increased attention in psychopathology is the actual transaction between the individual and environment—for example, the features of the way a child and parent interact. Intervening to remove risk factors from multiple domains simultaneously can potentially provide the most effective outcome.

Incorporating development into this model adds many challenges. The fields of developmental science and developmental psychopathology arose to create as a framework for the integration of information from developmental epidemiology, neuroscience, genetics, psychology, psychiatry, sociology, and other disciplines in order to better understand the complex interplay of factors affecting the health of an individual throughout their lifespan.[15] Major contributions from work in this area have been establishing the importance of interactions between genes and environment in determining the trajectory of development, rather than attributing mental health outcome to being due entirely to one factor or the other, and the dialectical nature of the relationship between the developing individual and their environment. The recognition of the importance of context in development has led to an elaboration of the different overlapping systems, or *ecologies*[16] that a child resides within and which present unique risks and opportunities for intervention.

Risk factors may be generalized, such as malnutrition and poverty, or more disease-specific, such as exposure to a particular toxin. Many risk factors will tend to occur together, and often risk factors have a non-linear relationship to outcomes; i.e. one or two may not significantly affect outcome, while as the number goes above a certain level risk increases sharply for a number of disorders. An additional complexity in developmental psychopathology is the presence of multicausality and multifinality. Multiple risks or disease processes may produce similar behavioural phenomena, while specific risk factors may be associated with a wide range of clinical presentations. Tracing causal paths and determining what are the factors that are mediating and moderating the relationships between risks and outcomes depends upon the ability to follow the impact of specific interventions over time.

The prevention research cycle and evidence-based prevention

Although direct experimentation on human subjects to establish causality among the risk factors affecting developmental trajectories is not in itself ethically feasible, suitably designed longitudinal controlled trials of preventive interventions can address the same goals.[17] Recognition of the value of considering prevention research as an iterative process led to the formulation of the *preventive research cycle*.[4] The steps in the cycle are: (i) identification of the problem or disorder and the size of its impact on a community; (ii) review of relevant information, particularly regarding relevant risk and protective factors available data from existing preventive research programmes; (iii) design, conduct, and analysis of pilot studies, including replication at multiple sites; (iv) implementation of larger-scale trials which will provide additional information about which populations may be more or less appropriate, and how the intervention does when scaled up in size; and (v) large-scale implementation and ongoing evaluation.

The randomized clinical trial, in which individuals or discrete communities are randomly assigned to receive either the intervention under investigation, a different intervention, or no intervention at all, continues to be a gold standard for determining whether a prevention programme itself is responsible for observed changes and thus to establish causality. Only a randomized clinical trial can determine if the intervention actually results in prevention, i.e. evidence that new cases did not develop that otherwise would have. Some trials, particularly those for populations in which some symptoms may already be present, result in decrease of those subsyndromal symptoms. While this is not without value, it is more strictly considered treatment than prevention.

The ability to make a convincing case for the value of a preventive intervention is particularly important because investing in prevention is asking an individual or community to devote resources towards a problem that has not yet occurred. Standards for evidence-based preventions have been explicitly identified to help with design and evaluation of studies, including criteria for when there is sufficient grounds to move along the research cycle from pilot studies to large-scale field trials and final dissemination.[18] The recommendations provide guidance for appropriate statistical methodology and design, and emphasize the need for replication in independent samples, adequate provision of training materials for non-research personnel as the scope of the project grows, and ongoing data collection after dissemination to inform communities and researchers about the impact of the intervention and direct the next iteration. They also differentiate between *effectiveness*, defined as showing a positive result in pilot studies under highly controlled circumstances, and *efficacy*, indicating a programme is also able to produce results in the less-optimal conditions associated with larger-scale trials.

Types of preventive interventions

Once it has been determined that a particular problem is present, and pertinent risk and protective factors have been identified, it is necessary to determine what type of intervention is most likely to be effective. Two broad distinctions are applicable to any intervention. The first, as implied by the host-agent-environment model, is whether to address the individual, their environment, or both. The second distinction concerns which portions of the population potentially at risk are to be addressed.[19]

Primary, secondary, and tertiary prevention

The first widely used public health prevention categories were proposed by the Chronic Disease Commission in 1957, who classified prevention as being primary, secondary, or tertiary.[20] *Primary prevention* is aimed at the normal population and defined as efforts aimed at decreasing the incidence of new cases, such as preventing access to contaminated water supplies as illustrated by the case of John Snow. *Secondary prevention* is targeted towards individuals who already show early signs of disease or disorder, with the aim of decreasing the prevalence of already established cases. *Tertiary prevention* attempts to minimize the degree of morbidity associated with an established illness, through decreasing its duration or associated disability. Such definitions were a crucial step in designing interventions to focus on a specific population and problem and take into account specific characteristics of that situation. However, classifying prevention by the disease stage may require a greater understanding of how risks related to disorders than is possible for many conditions.

Universal, selected, and indicated prevention

An alternative classification system based upon risk–benefit considerations for preventive interventions was introduced by Gordon in 1983[21] and disseminated through the seminal *Institute of Medicine* report in 1994.[4] Gordon proposed that the benefit of a prevention programme could be assessed by comparing an individual's risk of developing a disorder with the risk or cost of the associated intervention. In his system, prevention is classified as *universal*, *selected*, or *indicated*, depending on the degree of identified risk. *Universal prevention* is applied to everyone in a defined population, and the associated interventions are optimally low risk, low cost, and may be administered by individuals who possess relatively little specialized training. However, universal prevention spends resources on a large number of individuals who would not have become ill in any case. *Selective intervention* is aimed at individuals at above-average risk for a disorder, and anticipates a commensurately higher cost and intensity of intervention. Finally, *indicated prevention* is for individuals who are showing early signs of a disorder or exhibit biological markers indicating risk; the acceptable cost and risk here would again be higher to reflect the increased need of the individual.

There are areas of similarity between the two systems, which has led to some confusion. The populations and goals of primary and universal prevention are comparable, but selected and indicated groups indicate individuals at increasing levels of risk but who do not yet meet criteria for a disorder, whereas secondary and tertiary address issues related to different stages of having a disorder.

Comparison of prevention and health promotion

An alternate conceptualization of how to proactively intervene to improve outcomes is *health promotion*, defined as measures to increase likelihood of wellness as a positive quality rather than limiting efforts to decreasing risks for a negative outcome.[5] From a practical standpoint it overlaps largely with universal prevention, but the theoretical foundations and targeted outcomes differ. Although few would disagree with the potential benefits of promoting health in the community, health promotion has not always been included within the scope of prevention policy due to concerns that it may dilute efforts towards risk prevention that are characterized by more clearly definable and measurable outcomes. Others have argued that in view of the complex pathways of developmental psychopathology, a less-specific approach is more consonant with our existing knowledge, and may actually be more effective over the long run to create resilience against a broader range of disorders.[22]

Effective preventive interventions for children's mental health

The degree of implementation of preventive measures for mental health disorders in children and adolescents depends largely on how convincingly specific risk factors can be demonstrated which are malleable to politically and economically feasible actions. For example, realization of the adverse effects of prenatal alcohol exposure on neurodevelopment resulted in widespread public education efforts. Other toxins such as lead-based paint have also been the focus of education and regulations to decrease children's exposure. Vaccination programmes have significantly reduced mental disorders associated with infectious diseases such as rubella, and programmes have been put in place to decrease risks from accidents through measures such as use of car seats and bicycle helmets.

For conditions where the core risk factors are less clearly defined progress has been slower, but enough data has accrued over the past two decades of systematic prevention trials to be able to begin to assess the effectiveness of preventive interventions in this context. The scope of the current chapter does not allow a detailed description, and the reader is referred to relevant chapters for

specific disorders within this text, as well as general reviews and meta-analyses available elsewhere.[4,5,16,19]

Health promotion

A recent report by the World Health Organization summarized globally relevant general risk factors and the state of evidence for specific interventions to promote mental health and resilience.[6] Social, environmental, and economic factors have a major impact on mental health. Increasing attention is also being paid to *social capital*, a concept which broadly refers to aspects of social organization and community norms that facilitate the ability of individuals to work together for mutual benefit. General measures for health promotion include improving nutrition, housing, access to education and economic security, as well as strengthening community networks, reducing exposure to violence, decreasing substance abuse, and intervention to help with recovery from disasters. Risks for children's mental health from the proximal family environment include adverse maternal behaviour during pregnancy, such as substance abuse, child abuse, parental mental illness, and domestic violence, and may be addressed with measures such as home-visiting programmes for pregnant women and new mothers and pre-school programmes.

While the benefits of such general health promotion activities seems highly plausible, rigorous evidence of their effects on mental health allowing quantification for cost-benefit questions is difficult to come by and currently patchy, especially for larger-scale interventions. Reasons for this include the length of time necessary to see the results of these interventions, which may be much longer than the policy environment which fostered them; the large samples necessary, which may range from difficult to impossible to randomize appropriately, and the lack of funding for this type of information-gathering. Here in particular naturalistic 'experiments' may be of aid, in which populations exposed to changes in risk factors or social policies are closely monitored for the impact on health outcomes.

Prevention

The practical implementation of universal preventive measures overlaps largely with health promotion, despite the differences in their theoretical background and aims. Universal prevention programmes have the significant advantage of not conferring stigma upon participants, but their benefits are difficult to quantify due to the generally small effect sizes and consequent large samples necessary,[23] and they by definition devote significant resources to individuals who likely would not have had problems regardless. Universal prevention programmes with evidence of benefit have been developed for issues such as conduct disorder, anxiety, and depression. These have been primarily school-based, focusing on classroom behavioural management, social skills training, and cognitive strategies to help children learn prosocial behaviours and cope with stressful situations. Some programmes adopt a multimodal approach which includes parents. Universal approaches to decrease substance abuse have had mixed results. Educational techniques have shown clear success in increasing the knowledge base regarding risks of substance abuse, but impact on actual usage has been harder to demonstrate outside of more comprehensive programmes targeting multiple types of risk.[16]

Preventive measures for selected populations become more specific to individual disorders. Children at risk for conduct disorder often come from impoverished environments with high rates of exposure to violence, substance abuse, and weak family and community structures. Secondary preventions in these settings accordingly rely more strongly on multimodal interventions which include the family. Children with a depressed parent are at increased risk for depression, and secondary interventions in this case may include treatment of the depressed parent in addition to cognitive therapies for the child. Stress related to difficult transitions such as parental death, divorce, or unemployment are also significant risk factors for children, and have been effectively addressed with courses of cognitive group therapy.

Indicated prevention measures, for children with early symptoms or biological markers of a disorder, have the narrowest scope and generally the clearest evidence of effectiveness. A seminal study in the prevention of depression was performed by Clarke and colleagues,[24] who showed that cognitive therapy in adolescents with subsyndromal symptoms of depression and a depressed parent could reduce the incidence of new cases of depression compared to a control group. Schizophrenia has become a target of preventive medicine through studies showing that treatment of adolescents with early symptoms of psychosis may delay onset of a full psychotic break.[25] Multimodal interventions have also been shown to be effective for children and adolescents already showing signs of increased aggressive or antisocial behaviour.

Common themes in 'best-practice' mental health prevention programmes have included the need for multimodal approaches which simultaneously address both the child and components of the environment, and the increased durability of improved outcomes when interventions are maintained for significant lengths of time. Many programmes focus on reduction of proximal risk factors rather than mental health disorders themselves as a more feasible outcome measure, although when possible it is optimal to incorporate both. When preventive research began to stratify interventions into universal, selected, and indicated, it was originally predicted that the lowest-risk individuals would benefit the most from universal-level interventions. It was instead found that higher-risk children actually showed the greatest response, supporting the development of tiered systems in which children who did not benefit adequately from universal measures could also be referred for secondary and indicated levels.

In general, meta-analyses have found that preventive programmes in mental health for selected and indicated populations have small to moderate effect sizes, similar to those seen in other areas of medicine. Anxiety and depression have shown the most consistent responses. Universal programmes have not shown significant effectiveness in meta-analyses of controlled trials, which is understandable given the necessary sample sizes, but has led to debate regarding the justification of their claim on scarce resources. Another concern is that most research has been carried out at the level of pilot studies, with much less available from large-scale trials or fully disseminated programmes. What information is available shows a tendency for a fall off in effectiveness when moving to larger-scale implementation. This suggests a need to spend more attention from the earliest stages on issues relating to dissemination such as adapting programmes for existing community infrastructure. Early collaboration with the members of the targeted community also helps to ensure relevance of a programme and consequent participation; for example parenting classes, while potentially valuable, may not attract individuals preoccupied with issues,

such as safety or securing transportation. Finally, the issue of how to transfer preventive programmes into different settings requires much more extensive attention. Most prevention research has been done in a few of the more affluent nations, primarily the United States, United Kingdom, Canada, Australia, and countries in northern Europe. Little is known about how to transfer programmes or which programmes may be suitable for other less-affluent areas.

Conclusion

Enormous progress has been made in recognizing the scope of mental health problems for children around the world, and in developing the theoretical framework needed to address decreasing this burden in a systematic fashion. Technological advances in neuroimaging, genetics, and computational biology are providing the tools to start describing the biological processes underlying the complex course of development, and have renewed appreciation of the role of the environment in determining how a genetic heritage is expressed.

However, rapid technological change is also altering the environment of children and their families at an unprecedented rate, and what kinds of challenges to public health these changes may present is not yet fully understood. What is becoming clear is that as technological advances increase the range of available health care treatments, along with the potential cost, the choices for societies between spending limited resources on treatment or prevention will have to become increasingly deliberate.

A substantial body of work has demonstrated that prevention in mental health can be effective, but those who would benefit the most from preventive interventions are often not those with the political or economic resources to make them a priority. While the potential interventions to prevent mental health disorders in children are constrained by the knowledge and resources available, what is actually done depends upon the social and political values of individual communities and nations.[9] It is to be hoped that as our understanding of these disorders grows, public policies to prevent the development of mental health disorders in children will become as commonplace a responsibility for modern societies as the provision of clean drinking water.

Further information

UK. National Health Service Guide for Child and Adolescent Mental Health: http://www.bma.org.uk/ap.nsf/Content/Childadolescentmentalhealth

World Health Organization webpage for mental disorders: http://www.who.int/topics/mental_disorders

U.S. substance abuse and mental health services administration: clinical preventive services in substance abuse and mental health update: from Science to services http://www.samhsa.gov/publications/allpubs/SMA04-3906/i.asp

Society for prevention research: http://www.preventionresearch.org

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9.2

Clinical syndromes

Contents

Note *Substance abuse is considered in Part 4, Section 4.12. Aspects relevant to young people are considered within the chapters of this section.*

9.2.1 Neuropsychiatric disorders

James C. Harris

The developmental perspective

Developmental neuropsychiatry addresses the neurobiological basis of behaviour in infants, children, and adolescents with neurodevelopmental disorders and in those with brain damage occurring during the developmental period. As a field, it includes the aetiology, diagnosis, and treatment of behavioural, emotional, interpersonal, and psychiatric disorders.[1, 2] The parent's response, adjustment to, and involvement in treatment is a critical element in outcome.

The developmental neuropsychiatrist utilizes a developmental perspective that focuses on the developing person who is active, socially oriented, and emerging rather than passively responding to the environment. The adaptive plasticity of the developing nervous system to change is emphasized, and the essential role of environmental experience in brain development is acknowledged. When working with the affected child, an effort is made to provide the supports needed to facilitate the mastery of age-appropriate developmental tasks always keeping in mind the child's individual capacities and strengths.

Scope of developmental neuropsychiatry

The scope of developmental neuropsychiatry is broad[2] and includes the following.

1 Neurodevelopmental disorders that are described in other chapters of this book, including attention-deficit and hyperactivity disorders (Chapter 9.2.4), pervasive developmental disorders and childhood-onset schizophrenia (Chapter 9.2.3), obsessive–compulsive disorder and Tourette's syndrome (Chapter 9.2.8), and specific developmental disorders (Chapter 9.2.2).

2 Neurogenetic disorders, both cytogenetic and metabolic, with behavioural phenotypes, several of which are also reviewed in Chapter 10.4.

3 Teratogenic exposure from both organic and inorganic toxins. In these instances, behavioural dysfunction may result from gestational substance abuse with alcohol and other substances or exposure to inorganic metals.

4 Endocrinopathies.

5 Traumatic brain injury.

6 Other neurological disorders (e.g. epilepsy).

Clinical features

Neurodevelopmental disorders

Developmental psychopathology applies developmental concepts to the study of neurodevelopmental disorders. The relationship of disordered to non-disordered behaviour is considered, as are the early origins of maladaptive behaviours that may not appear in clinical form until adolescence or adulthood. Knowledge of normal development is utilized to study children whose development is atypical, in order to understand the natural history of their disorder and establish the developmental trajectory of that particular condition. Conversely, the investigation of such deviant behaviour associated with a particular disorder is considered in regard to our understanding of normal development. For example, attention-deficit hyperactivity disorder has been investigated as a disorder of executive functions of the prefrontal cortex, and autistic disorder as a disorder of social cognition and communication. In both instances, new knowledge about brain functions has been derived from these formulations. Among the neurodevelopmental disorders, the age of recognition varies, multiple causes are involved, and many transformations in behaviour may occur in determining their complex course. The goal is to understand the mechanisms and processes through which risk factors lead to the emergence of a disorder. Disordered behaviour is not viewed as a static condition, but is considered as part of a dynamic transactional engagement. Behaviour and development are viewed within a social context, and the transactional nature of interactions is considered from infancy through adulthood to understand these processes.

Attention-deficit hyperactivity disorder, pervasive developmental disorders, obsessive–compulsive disorder, Tourette's syndrome, and childhood-onset schizophrenia are developmental neuropsychiatric disorders under active investigation and each is reviewed in the respective chapters. Their developmental psychopathology is investigated by addressing the origins and course of individual patterns of behavioural maladaptation in each of these disorders and determining their genetic bases, thought to be complex, and involving more than one gene. Information derived from genetics, developmental psychology, clinical psychology, psychiatry, sociology, physiological sciences, neurosciences, and epidemiology is included in the description of each of these disorders.

The interrelationship of the various child neuropsychiatric disorders is an important consideration. Disorders may be risk factors for other conditions, so that attention-deficit disorder may be a risk factor for conduct disorder. In this instance, the child's behaviour affects the adult and the transactional interactions between child and adult may result in further disruptive behaviours. Moreover, there may be a developmental basis for disorders whose full presentation is not evident until later in life, as is the case with schizophrenia—generally considered to be a disorder of late adolescence or early adult life, but with origins in the developmental period.[3] Some disorders may have co-occurring diagnoses that influence their outcome, as in Tourette's syndrome, where co-occurring conditions may determine the behavioural presentation. In Tourette's syndrome, obsessive–compulsive symptoms may be an aspect of 'pure' Tourette's syndrome, while co-occurring disruptive behaviour may be secondary to co-occurring attention-deficit disorder. Social and behavioural dysfunction in children with Tourette's syndrome is largely ADHD-specific. Children with TS alone have a different social-emotional profile.[4–6] Compulsive behaviours may not only interfere with the normal routines for the affected child but also become particularly problematic for their impact on other family members.

Neurogenetic syndromes with behavioural phenotypes

Particular patterns of behaviour, temperament, and psychopathology may be associated with specific chromosomal and genetic disorders.[2,5,7–9] The term 'behavioural phenotype' was introduced by Nyhan in 1972[7,10] to describe patterns of unusual behaviour that are so characteristic that they suggest a specific neurogenetic disorder. Nyhan described stereotypical patterns of behaviour occurring in syndromic fashion in sizeable numbers of affected individuals with a given syndrome, and observed that these patterns seemed self-programmed. In these children, he proposed that it is reasonable to hypothesize that their behaviours are associated with an abnormal neuroanatomy and that such stereotypical patterns of unusual behaviour could reflect the presence of structural deficits in the central nervous system. Recent developments in the neurosciences provide a means to investigate the biological bases of behavioural phenotypes. Behavioural assessments, neuropsychological testing, and neuroimaging procedures, carried out in well-characterized genetic syndromes, are being utilized to understand pathways from genes to cognition and complex behaviours in these conditions.

Comprehensive study of children with different neurogenetic disorders may increase our appreciation for the relative contribution of genetic variables in the pathogenesis of specific, affective, and behavioural disorders. Behavioural phenotypes have been studied most extensively in Down syndrome (language),[11] fragile X syndrome (gaze aversion, hyperkinesia, autistic-like behaviour),[12] Williams syndrome (sociability, hyperverbal behaviour, and visuospatial deficits),[13,14] Lesch–Nyhan syndrome (compulsive self-injury and aggression),[15–17] and Prader–Willi syndrome (hyperphagia, obsessive–compulsive behaviour).[13,18,19] The number of identifiable behavioural phenotypes is growing with careful observations of behaviours in neurogenetic disorders.[8,9] Besides behaviours, particular temperamental features have also been considered in these disorders. However, when studying temperament, the appropriate measures must be chosen. For example, when Down syndrome, proposed to be linked to a particular temperament, was studied using temperamental clusters of easy temperament, slow to warm-up, and difficult temperament, Gunn *et al.*[20] demonstrated both easy and difficult temperament in children with Down syndrome; therefore, a typical temperamental pattern among these three categories was not demonstrated. However, when a more comprehensive assessment was carried out in other syndromes[21] (that included the personality factors of extraversion,

agreeableness, conscientiousness, emotional stability, and openness, along with motor activity and irritability), specific personality phenotypes were identified. These were differentially related to parental behaviours and family context in Prader–Willi, fragile X, and Williams syndromes. Moreover, isolated special abilities, as in calculation and in music,[22] are recognizable that might be considered as phenotypes and linked to the proposed modular organization of the central nervous system. Finally, physical and behavioural phenotypes are not only identified in neurogenetic syndromes but also in those caused by environmental events, such as intrauterine exposure to alcohol: namely, the foetal alcohol syndrome. Because alcoholism is a familial disorder, there may vulnerability to its effects resulting in a severe presentation in some individuals and less severe presentation in others.[23]

Both traditional Mendelian laws of inheritance (Lesch–Nyhan syndrome) and non-traditional inheritance have been identified in conditions with behavioural phenotypes. Among the non-traditional forms of inheritance are triplet repeat amplification (fragile X syndrome), microdeletion or contiguous gene deletion (Williams syndrome), imprinting (Prader–Willi syndrome), transcriptional derepression (Rett's syndrome), and excessive gene dosage (Down syndrome). A key finding is the recognition that mutations of single genes can lead to complex behavioural symptoms, especially if the affected protein is essential for the expression or processing of multiple 'downstream' genes.

Behavioural phenotypes are also discussed in relation to intellectual disability in Chapter 10.5.1.

Neurobehavioural teratology

Neurobehavioural teratology investigates abnormal development of the nervous system and of cognition and complex behaviour that results from prenatal environmental insults. Neurobehavioural research addresses the prevalence of cognitive behavioural disorders in exposed individuals and the consequences of the brain insult on other developing brain systems, to identify risks for functional or behavioural deficits. Investigators focus on cognitive behavioural deficits and their underlying anatomy and embryology. Assessment emphasizes not only IQ but also neuropsychological profiles, because learning disability or difficulty in visuomotor integration may be evident in children who function in the low to average range of general mental ability.

The natural history of intrauterine drug exposure on motor, cognitive, emotional, and social behaviour is an area of growing concern. Multiple drug exposures during pregnancy are common among substance-abusing mothers. Of syndromes associated with intrauterine substance abuse, alcohol abuse has been studied the most extensively. Subsequently, retinoids, anticonvulsants (lithium, tegretol, and valproic acid), and the selective serotonin-reuptake inhibitors have also been studied. Other teratogens do not lead to major malformations of the nervous systems but they do compromise its integrity (for example, lead, heroin, methadone), and are associated with neurotoxic damage or effects on neurochemical systems.

The greatest period of vulnerability to drugs in a human pregnancy is during the period of embryogenesis (days 14 to 60). During embryogenesis, many neurobehavioural teratogens (for instance, retinoids and ethanol) produce syndromes with abnormalities that involve craniofacial, neural, and major organ systems. Behavioural abnormalities without detectable physical abnormality can occur when the insult occurs during the foetal period. The extent of malformation is stage-specific and dose-dependent, with outcomes ranging from death with malformation, malformation and survival, effects on growth, and cognitive–neuropsychological or behaviour disorder. The same exposure to alcohol needed to produce cognitive behavioural change in the foetal period would generally cause malformation if it occurred during embryogenesis. The term 'developmental toxicology' is sometimes used if the insult occurs in the postnatal period.

There may be a genetic vulnerability that influences the extent of expression of response to environmental toxins in an individual. A common family of regulatory genes is involved in the formation of structures of the face, head, hindbrain, parts of the heart, and thymus gland, all of which share a common origin from neural crest cells (anterior neural tube). These regulatory genes, known as HOX genes, provide rules for assembling various structures and for determining particular anatomical segments.[24] Homozygous HOXA1 mutations have been shown to disrupt human brainstem, inner ear, cardiovascular and cognitive development. Because the retinoid family is involved in controlling these HOX genes,[25] a similar pattern is produced by excessive retinoid administration, as in hypervitaminosis of vitamin A (retinol). Moreover, the enzyme alcohol dehydrogenase functions in the metabolism of both retinol and ethanol so that intoxicating levels of ethanol can competitively inhibit the metabolism of retinol and impact brain development. Thus, both genetic and teratogenetic agents may produce similar developmental abnormalities. Understanding these mechanisms helps to understand how an abnormal facial appearance may suggest an abnormal brain.

Foetal alcohol spectrum disorder syndrome

Foetal alcohol syndrome is one of the most commonly recognized causes of intellectual disability; one that is preventable if recommended guidelines regarding alcohol use are followed by mothers.[26]

Clinical features

Children with the full foetal alcohol syndrome demonstrate prenatal and postnatal growth deficiency, microcephaly, infantile irritability, mild to moderate intellectual disability, and a characteristic facial appearance.[23] The extent of the abnormality depends on the time of maximal exposure to alcohol and the dose. Approximately half of those affected have co-ordination problems, are hypotonic, and have attention deficits. Between 20 and 50 per cent have other birth defects, including eye and ear anomalies and cardiac anomalies. Those children who do not show growth retardation or congenital anomalies may show more subtle changes, such as attention problems, disruptive behaviour, reduced speed of information processing, motor clumsiness, speech disorders, fine motor impairment, and learning problems, especially in mathematics.[23,27] These findings have been documented in a prospective longitudinal study of the effects of prenatal alcohol exposure on a birth cohort of 500 offspring who were selected from 1529 consecutive pregnant women in prenatal care in community hospitals.[28] Dose-dependent effects are most clear from the neurobehavioural status of subjects when regular neurodevelopmental evaluations are carried out from birth to age 14 years. The more subtle abnormalities are referred to as 'foetal alcohol effects', or alcohol-related neurodevelopmental disorder. The full range of disabilities is described as foetal alcohol spectrum disorder.[27]

Subjects with average to above-average IQ may demonstrate neuropsychological deficits in complex attention, verbal learning, and executive functioning. Disruptive behaviour, attention-deficit disorder, anxiety disorder, and communication disorder have been described[29,31,33] in children with foetal alcohol syndrome and foetal alcohol spectrum disorder who test in the low normal range and in the moderate to severe range of intellectual disability.

Behavioural phenotype

The behavioural phenotype is characterized by problems in cognitive functioning, academic problems in arithmetic, difficulty with abstractions, understanding cause and effect, and generalizing from one situation to another. Thus, inattention, poor concentration, impaired judgement, memory deficits, and problems in abstract reasoning are characteristic. Behavioural problems related to impulsivity and hyperactivity makes them vulnerable to later diagnoses of oppositional defiant and conduct disorder.[27,30]

Natural history

Foetal alcohol spectrum disorder is not only a childhood disorder; the cognitive and behavioural effects and psychosocial problems may persist throughout adolescence into adulthood.[28,33] Although the facial features are not as distinctive after puberty and the growth deficiency is not as apparent as in the younger child, the central nervous system effects do persist throughout life. Approximately 50 per cent of those affected function as intellectually disabled persons. Moreover, adaptive behavioural problems in communication skills and in socialization are apparent in those with foetal alcohol spectrum disorder whose intelligence test scores are in the normal range.

Poor judgement, attention problems, distractibility, difficulty in recognizing common social cues, and problems in modulating mood continue as characteristic features. Family environmental problems often continue as risk factors for behavioural problems if there is a lack of stability in family life. In one follow-up study[34] that used structured interviews with non-intellectually disabled affected subjects, the most common diagnoses were alcohol or drug dependence, mood disorders, and personality disorders (especially passive aggressive or antisocial). Further follow-up is needed to investigate the mechanisms involved in these psychiatric presentations, and particularly in determining the pathways leading to alcoholism.

Epidemiology

Foetal alcohol syndrome is a common cause of neuropsychiatric disorders, with a worldwide incidence of approximately 1.9 in 1000 live births. When foetal alcohol syndrome and alcohol-related neurodevelopmental disorder are considered together, the combined rate in one study conducted in the United States was 9.1 in 1000.[35] Despite its frequency and severity, the syndrome may go unrecognized because physicians may not systematically enquire about alcohol use and may not recognize the spectrum of the effects of prenatal alcohol exposure on neurodevelopment.

Aetiology

The amount and pattern of alcohol consumption and the trimester of use during pregnancy, especially if during critical periods of brain development, are major factors in determining outcome. Binge drinking patterns with high blood concentrations are especially deleterious. Rapid changes in alcohol concentrations in the blood and central nervous system cause apoptotic damage (cell degeneration) in developing neurons and other cells in rat models.[36] Microcephaly is commonly reported in foetal alcohol syndrome and suggests an underdevelopment of the brain. Neuropathological studies demonstrate the underdevelopment or absence of the corpus callosum and enlarged lateral ventricles. Dendritic changes have been observed in animals with prenatal exposure to alcohol; these changes were correlated with decreased learning ability. Magnetic resonance imaging studies have documented brain abnormalities in foetal alcohol syndrome, particularly in midline frontal structures such as the corpus collosum.[37,38] Research to identify specific polymorphisms contributing to foetal alcohol spectrum disorder is at an early stage. Polymorphisms of only one of the genes for the alcohol dehydrogenase enzyme family, the ADH1B, have been demonstrated to contribute to vulnerability.[39]

Treatment

(a) Evidence

Mothers of children with foetal alcohol syndrome who drank more alcohol and drank excessively early in gestation have more severe clinical features. Alcohol use in late pregnancy is primarily associated with prematurity and infants who are small for gestational age, rather than with the full foetal alcohol syndrome. Because of these risks, treatment must begin with prevention.[32] There is no clearly agreed safe dose of alcohol for pregnant women. Because there is no known safe amount of alcohol consumption during pregnancy, it is recommended that women who are pregnant or who are planning a pregnancy abstain from drinking alcohol. Special efforts for educating women of child-bearing age are required that highlight the harmful effects of alcohol; identified children must be referred for early educational services.

(b) Management

A comprehensive treatment programme begins with parental acknowledgement of the aetiology of foetal alcohol syndrome or spectrum disorder and treatment for the parent, as indicated, for alcohol misuse and abuse. Parental counselling should include discussion of the physical and behavioural phenotype. The family should be advised about the need for special educational programmes and assisted in behavioural management. Family therapy is often required to help family members cope with the developmental disorder. Appropriate educational and behavioural treatment resources are needed to address the social deficits, particularly in those cases where disruptive behaviour, attention-deficit disorder and mood disorders are identified.

Foetal alcohol syndrome is also considered as a cause of intellectual disability in Chapter 10.4.

Gestational substance abuse

Opiates

Exposure to heroin and methadone ranges from a neonatal withdrawal syndrome to less predictable long-term outcomes.[40] An impoverished environment may have disproportionate adverse effects on methadone-exposed children when compared to unexposed children.[41] Methadone effects have been associated with

increased body tension, poor motor co-ordination, and delay in motor skills acquisition.[42] However, the effects on mental development are less clear, but they do affect the child-rearing environment. Since methadone exposure produces an increased vulnerability to the effects of poor parent–infant relationships, these relationships require careful monitoring.

Cocaine

Cocaine is a central nervous system stimulant that inhibits nerve conduction in the peripheral nervous system. Cocaine is metabolized primarily through the plasma cholinesterase system, with the primary metabolic product being benzoylecgonine. Since cocaine rapidly crosses the placenta by simple diffusion, foetal peak blood levels are reached as quickly as 3 min.[43] Having crossed the placenta in the foetus, cocaine has the same direct actions on the foetal cardiovascular system as seen in the maternal system. These cardiac changes involve the direct effects of cocaine, as well as indirect effects such as foetal hypoxia. Cocaine may lead to placental dysfunction (via vasocontracture effects), structural changes (via vascular compromise), and neurobehavioural abnormalities (via postsynaptic junction neurotoxicity).

Infant gestational age, birth weight, head circumference, and length have been found to be decreased in affected infants, and low birth weight is a frequent finding in studies of the offspring of cocaine-using women. In addition to abnormal growth patterns, congenital anomalies involving the genitourinary tract, heart, and central nervous system as well as limb-reduction abnormalities have been reported. A potential mechanism for all these anomalies appears to be interruption of the intrauterine blood supply, with subsequent disruption of embryonic development. Although approximately 25 to 30 per cent of infants exposed to cocaine *in utero* may have physical difficulties, overall neurobehavioural problems may be more common, and most apparent in early infancy and childhood.[40, 44] In one large study, at age 4 years, prenatal cocaine exposure was not associated with lower full-scale, verbal, or performance IQ scores but was associated with an increased risk for specific cognitive impairments. A better home environment was associated with IQ scores for cocaine-exposed children that are similar to scores in non-exposed children. Although irritability and problems in state regulation are reported in infants and impulsive behaviour in pre-school children, these behaviours diminish over time with behavioural and psychosocial interventions.[45] Child abuse is closely linked to substance abuse.

Treatment

These findings suggest that careful attention be paid to the postnatal home-rearing environment of children who are exposed to drugs *in utero*. Overall, the treatment programme must take into account physical and psychological change secondary to intrauterine drug use as well as the postnatal nurturing environment. Both substance use and psychiatric disorder in the parents must be considered, because parents with attention-deficit disorder and mood disorders may themselves self-medicate with cocaine. Without early intervention, special school programmes, behavioural management programmes, and a structured day programme will be necessary. Ongoing parent training is also required.[40, 42]

Endocrinopathies

Congenital hypothyroidism

Congenital hypothyroidism is associated with intellectual disablity and may be associated with decreased motor activity at birth, hoarse cry, and difficulty with feeding. It is rarely diagnosed at birth from clinical assessment alone, but it is recognized from newborn screening tests with confirmation by measurement in blood samples. Symptoms of hypothyroidism may not be clearly detected until the second month of life. The overall prevalence is 1 in 4000 live births. Neurological and learning disorders associated with untreated congenital hypothyroidism include attention-deficits, hearing loss, speech defects, ataxia, and abnormal muscle tone.[46] Rapid diagnosis in infancy is essential to prevent these complications. Without treatment, severe neurological dysfunction ensues. With initiation of oral thyroid hormone treatment (levothyroxine in a single daily dose of 8 to 10 μg/kg per day) in the first 6 weeks of life, IQ is in the normal range. If treatment is delayed until 3 to 6 months, IQ drops to an average of 75, and, if initiated after 6 weeks, to an IQ of 55 or less. Rearing environment is important in long-term outcome. Despite early treatment there still may be enduring cognitive and motor deficits in young adults.[47]

Traumatic brain injury

Traumatic brain injury is defined as physical damage or impairment in function of the brain as a consequence of the application of acute mechanical force. Other causes of brain injury result from birth trauma, poisoning, or asphyxia. Traumatic brain injury is a major cause of death and disability among children, adolescents, and young adults, and is one of the most common causes of chronic brain syndromes in childhood. Traumatic head injury is common and becoming increasingly more so.

Clinical features

(a) Cognitive and behavioural

The most common long-term outcomes of traumatic brain injury are cognitive and behavioural changes. Immediately after emerging from a coma, the child will be unable to form new memories. The time, from the accident to the time when new memories emerge, is referred to as post-traumatic amnesia. The length of coma and the duration of post-traumatic amnesia are especially important in regard to the extent of cognitive recovery. Moreover, there is a strong inverse relationship between subsequent IQ and duration of coma. The persistence of cognitive deficits is correlated with the duration of post-traumatic amnesia; the more persistent deficits follow more than 3 weeks of post-traumatic amnesia. Persistent verbal memory impairment is reported as long as 10 years after injury in up to one-quarter of those studied. Psychiatric symptoms in adults occurs more often following focal frontal-lobe traumatic brain injury than injury to other cerebral areas. In children, Rutter[48,49] reported behavioural disinhibition after severe closed traumatic brain injury characterized by over-talkativeness, ignoring social conventions, impulsiveness, and poor personal hygiene.

(b) Psychiatric

Psychiatric outcomes can be divided into those that occur during the early phases of recovery and those that occur later. The earliest

psychiatric sequelae are found before the termination of post-traumatic amnesia. During this time, behavioural and affective symptoms are linked to the neurological presentation. The most common psychiatric diagnosis is delirium. Symptoms include short attention span, agitation, hallucinations, and disturbances in the sleep–wake cycle.

Subsequent occurrence of post-traumatic psychiatric symptoms is linked to the severity of the injury, its location, the child's behavioural and emotional features prior to the accident, and the psychosocial interactions of the family members during the recovery phases. The more severe the traumatic brain injury, the greater the likelihood of psychiatric sequelae. All children in one prospective study of severely injured children who had premorbid psychiatric conditions showed post-traumatic psychiatric disorders.[50,51] Moreover, over half the children in this group who had no premorbid symptoms prior to the accident had developed psychiatric symptoms during a 28-month, follow-up period. The greatest premorbid risks for psychiatric disorder were previous difficulties with impulse control and disruptive behaviour. In addition, a prior history of family dysfunction increased the risk for later symptomatology. The range of disorders includes attention-deficit hyperactivity disorder,[52] disruptive behaviour[53] post-traumatic mood disorders (both depressive and manic symptoms), post-traumatic stress disorder,[54] and family dysfunction.[55, 56] Transient psychotic features may occur. Hallucinations tend to be less bizarre and more concrete than the typical hallucination in schizophrenia. Moreover, head injury in childhood may accelerate the expression of schizophrenia in families where there is strong genetic predisposition[57] Injuries involving focal frontal-lobe dysfunction are associated with impulsive aggression and behavioural dyscontrol,[58] most often following focal orbitofrontal injury. The rate of actual aggression is less than often assumed. When forensic issues are considered regarding violent behaviour each case should be evaluated individually taking into account the type of head injury and other risk factors especially a history of physical abuse.

Classification—types of traumatic brain injury

Neurological damage associated with head trauma can be produced in several ways. Traumatic brain injury is classified as open or closed; these types differ in the pattern of injury and neurobehavioural outcome. Open refers to penetration of the skull, as in a depressed skull fracture or bullet wound, the extent depending on the regions damaged by contusion or cerebral oedema. Closed head injury results from acceleration and deceleration of the brain within the hard skull; this often leads to contusion of the brain from a sudden impact and may result in subarachnoid haemorrhage. Different parts of the brain have different densities, and therefore shearing stresses that develop during rapid brain movement cause injury. Furthermore, compression of blood vessels against the falx cerebri or tentorium may result in infarction of the areas, which these blood vessels supply. Penetrating traumatic brain injury causes specific and direct loss of neural tissue.

Epidemiology

It is estimated that 185 children per 100 000 from infancy to 14 years of age and 295 per 100 000 adolescents and young adults aged between 15 and 24 are hospitalized each year for traumatic brain injury.[59] The risk is highest among the 15- to 19-year-olds where the rate is 550 per 100 000.[60] The incidence in paediatric populations is similar to that in adults. In the United Kingdom the rates for those under 16 years is approximately 45 000, with about 300 deaths each year.[61] A mortality rate of 10 per 100 000 makes head trauma a major cause of death in children, but the death rate is still less than that in adults. There is no difference in the death rate between boys and girls before the age of 5 years, but after this age males are four times more likely to die than females. Approximately 90 per cent of head injuries are mild.[61] Falls and transport injuries make-up the majority of cases. Inflicted traumatic brain injury from physical abuse is a growing concern especially of repeated injuries. A US statewide population based study found the incidence of inflicted traumatic brain injury to be 17 per 100 000 person-years in the first 2 years of life with the highest incidence in infants during their first year (30/100 00). The rate was higher in boys than girls.[62]

Aetiology

The causes of traumatic brain injury are different depending on the age of the child. The incidence is twice as high in males as in females, and children who live in poor psychosocial circumstances are at greater risk. Traumatic brain injury from child abuse occurs in infancy: in the pre-school years the most common cause is falls; in early elementary school, it is pedestrian accidents. From 10 to 14 years of age there is an increase in sports and bicycle accidents, but by 15 years motor vehicle accidents and violent assault are the most common. Risk factors include poverty, single-parent homes, congested living arrangements, and a parental history of psychiatry disorder.

A common complication of traumatic brain injury is cerebral oedema, but there are other complications such as infection and haematoma formation both inside and outside the brain. These complications result in neurological deficits that may be extensive. Furthermore, compensatory mechanisms that are involved in recovery from head trauma may alter brain function. A child who has suffered a traumatic brain injury is likely to experience both neurological and psychiatric difficulties depending on the brain regions involved. Multiple mechanisms lead to psychological symptom formation—both psychosocial and physiological factors are involved.

Course and prognosis

The level of consciousness, degree of somatic injury, extent and duration of post-traumatic amnesia, severity of head injury, and degree of neurocognitive dysfunction in the early post-trauma period are important in determining outcome. Children who experience severe traumatic brain injury usually follow a predictable postoperative course.[48] As previously noted, landmarks for recovery are associated with the time of emergence from coma and the time of emergence from post-traumatic amnesia. The emergence from coma is most often defined as the point at which the patient is able to follow simple verbal commands. Concurrently, visual tracking of objects in the environment may be observed.

Post-traumatic amnesia ends when the child is able to form new memories. The frequency of post-traumatic amnesia is probably related to concurrent injury to the temporal lobes associated with the head trauma. However, older memories may be recalled that do not involve the temporal lobe. The hippocampus has a central role

in the formation of new memories. Besides recovery from post-traumatic amnesia, another form of memory loss-retrograde amnesia-for events that took place before the accident, typically becomes shorter and shorter during the recovery process. It is important to remember that children with severe head trauma will rarely have specific memories of the accident itself. Overall, the most important milestones in recovery for future outcome are the length of coma and the duration of post-traumatic amnesia.

Treatment

(a) Evidence

Most mild head injury and post concussive problems will resolve without treatment. When there are ongoing symptoms, parents and teachers must adjust expectations depending on the extent of injury. Complete recovery of all brain functions following severe brain injury is rarely accomplished. Still, if recovery is defined as a reduction in impairments in behavioural and physiological functions over time then changes do occur so that, typically, there is recovery of function together with a fair amount of substitution of function. Mechanisms include resolution of brain swelling (oedema), resolution of damage to other brain regions damaged through shock (diaschisis), changes in the structure of the nervous system (plasticity), and regrowth of neural tissue (regeneration). The extent of recovery depends on the severity of the injury, the number of times injured, the age at the time of injury, premorbid cognitive status, extent to which loss functions can be subsumed under other systems, integrity of other parts of the brain, individual brain structures, motivation, emotional considerations, and the quality of rehabilitation programme.[63, 64]

Although children and adolescents tend to have a better outcome after severe traumatic brain injury than those over the age of 21, the adult brain has greater plasticity than previously considered. Despite this general rule, children who are younger than 7 years may have a worse outcome since they may be at increased risk of child abuse as a cause of injury which may be particularly traumatic. Furthermore, younger children may have a worse outcome based on the global effects of trauma on the developing brain. The duration of recovery of significant neuropsychological, behavioural, and emotional deficits may last several years following injury. These higher cognitive deficits lead to the major disability observed with traumatic brain injury.

(b) Management

Partial recovery of function can and does occur over time, not only in children but also in adults. Intervention through retraining and the use of cognitive memory aids is targeted to improve areas of cognitive functioning such as memory, attention, language, and perception.[63] Even though partial recovery does occur after various types of brain injury, there is variability in the extent of recovery.

Possibilities for prevention

The most important primary injury prevention activities focus on teaching safe behaviour, the use of seat belts in cars, and wearing helmets when riding horses, bicycles, or motorcycles. Once an injury has occurred both anticipatory guidance, which teaches the family and child what to expect, and preventive intervention strategies are necessary. Early and focused rehabilitation procedures coupled with medication for associated psychiatric disorder, behaviour management, supportive therapy for families, and appropriate school programmes are necessary to prevent behavioural and psychiatric complications.

Epilepsy

Epilepsy refers to recurrent seizures that are idiopathic (of unknown aetiology) or due to congenital or acquired brain lesions. Epilepsy is the symptomatic expression of brain pathology or disordered brain function and is not a disease in itself. The symptom complex is episodic and associated with an excessive self-limiting neuronal discharge. The seizure is a frightening experience for parents and they require support and guidance. Epilepsy impacts the whole family and can create problems for all family members.[65]

Clinical features and clinical course

Complex partial seizures involving the temporal and frontal lobe is the most common condition where complex neurological and psychiatric symptoms are seen in the same person. Complex symptoms include behavioural automatisms, perceptual alterations, changes in affect and memory, distorted thinking, and hallucinations[66] Forms occurring in infancy and childhood include temporal lobe epilepsy, frontal-lobe epilepsy, infantile spasms, Lennox–Gastaut syndrome, Landau–Kleffner syndrome, and benign focal epilepsy.[67]

Children with partial seizures and electroencephalographic evidence of frontal involvement have more severe formal thought disorders and deficits in communication discourse than those with temporal involvement. Because these seizures are rare in children, reports of symptoms are primarily found in case reports. For example, Saygi *et al.*[68] and Stores *et al.*[69] have described sexual disinhibition, pressured and tangential speech, screaming, aggression, disorganized behaviour, and nightmares in affected children.

Frontal-lobe epilepsy should be considered if there are episodes of brief sudden unresponsiveness without loss of consciousness. These episodes occur with continued understanding of spoken language and clonic or tonic motor phenomena involving the face and arms bilaterally. Laughing, crying, pedalling movements, and sexual automatisms may also suggest this diagnosis. A normal electroencephalograph does not rule out the diagnosis. Left frontal hypometabolism on positron-emission tomography scanning or reduced cerebral blood flow to the frontal area, although not diagnostic, support this diagnosis.

The Lennox–Gastaut syndrome is characterized by early onset of intractable seizures and bilateral slow spike-wave complexes on the EEG.[70] The onset is typically between the ages of 1 and 7 years. The seizure pattern includes tonic, generalized tonic–clonic, atypical absence, atonic, and myoclonic seizures. Approximately half of children with the Lennox–Gastaut syndrome test as intellectually disabled. Marked language delay, overactivity, and irritability are characteristic. However, these behavioural symptoms may improve with seizure control. Ultimately, the diagnosis is based on the characteristic EEG finding of interictal slow spike-wave discharges in children with the early onset of poorly controlled seizures and a developmental disorder. In some instances, there is prolonged minor status epilepticus. Such episodes may last for several weeks during which the child engages in a variety of everyday activities but is socially unresponsive, aggressive, less articulate, and has

minor twitching of the face and hands. This presentation must be differentiated from a psychiatric disorder.

Classification

Classification of epileptic seizures utilizes both clinical and electroencephalographic features.[71] The current classification divides seizures into two categories: partial and generalized. Partial seizures involve one cerebral hemisphere, in part or totally. They begin focally, although they may become generalized. Consciousness is preserved but cognitive functions may be transiently impaired: for example, speech may be impaired if the dominant hemisphere is affected. Partial seizures are further subdivided into those with simple or complex symptomatology. In children, simple complex seizures are most often simple motor or sensory phenomena. Complex partial seizures usually begin in temporal or frontal-lobe structures. It is this group that is particularly important to psychiatrists.

Diagnosis and differential diagnosis

Epilepsy is a clinical rather than a laboratory diagnosis, and diagnostic errors most commonly occur due to inadequate history and physical examination. The accuracy of diagnosis has improved with the establishment of a universally agreed upon classification. In some instances there may be confusion between sleep arousal disorders and epilepsy.[72]

The differential diagnosis includes complex partial seizures of temporal lobe origin and pseudoseizures. Frontal-lobe complex partial seizures differ from those of temporal lobe origin in that the amnesia of frontal-lobe seizures is more pronounced than the extent of loss of consciousness. Moreover, frontal-lobe involvement is associated with unilateral or bilateral tonic posturing and pedalling movements, partial and not complete loss of consciousness, and eye and head deviation to the contralateral side. In complex partial seizures of temporal lobe origin, oroalimentary and repetitive hand automatisms, and looking around are characteristic. Lastly, sensory, gustatory, or olfactory hallucinations in frontal-lobe epilepsy must be differentiated from psychotic disorders such as schizophrenia and manic psychosis.

The distinction between true seizures and pseudoseizures can be difficult. Children with pseudoseizures commonly also have true seizures. Emotional dysphoria can precipitate true seizures and many children with chronic seizures have psychiatric diagnoses. Frontal-lobe seizures may be confused with pseudoseizures. Frontal complex partial seizures differ from pseudoseizures in that pseudoseizures have a gradual onset and longer duration, while frontal-lobe seizures start slowly and last less than 1 min. Pseudoseizures include thrusting or rolling movements rather than the rhythmic flexion and extension clonic movements seen in frontal-lobe epilepsy. Still, it may be difficult to distinguish pseudoseizures[73] and video and electroencephalograph monitoring with depth electrodes may be necessary to definitively diagnose frontal-lobe epilepsy.

Other features differentiating pseudoseizures are as follows:

1 The seizure occurs when the child is observed, but not when alone.

2 The seizures are gradual rather than of sudden onset.

3 Uncontrolled flailing occurs, rather than true tonic–clonic movements.

4 The seizure is accompanied by histrionics, with screaming and shouting.

5 Painful stimuli are avoided during an attack;

6 There is a sudden cessation of the seizure, with immediate return to an alert and responsive state.

7 There is absence of paroxysmal discharge during an attack on electroencephalography.[73]

Epidemiology

The incidence of epilepsy ranges from 0.7 to 1.1 per cent of the general population. It is the most common of the neurological diseases diagnosed in children.[67] Approximately 50 per cent of all cases of epilepsy begin during the childhood years and about 5 per cent of children will experience repeated epileptic seizures without a known extracerebral cause. In addition, about 3 per cent of children will have febrile convulsions that are usually benign and accompany a febrile illness. The great majority of these children, approximately 98 per cent, do not go on to develop true epilepsy. Other causes include hypoglycaemic seizures in children with diabetes. In some instances, as in tuberous sclerosis complex, cognitive impairment and autistic regression with onset in the first year of life, are linked to epilepsy; and in others there is a late onset of language disorder as in the Laudau–Kleffner syndrome.

The British Child and Adolescent Mental Health Survey[74] canvassed over 10 000 children and adolescents and identified 0.7 per cent of 5–15 year olds with a diagnosis of epilepsy. Among them there was an increased prevalence of emotional, behavioural, and relationship problems within families and among peers.

Aetiology

Advances in understanding epilepsy in childhood have come from the newer medical technologies. Recognition of a typical spike-wave pattern has led to the identification of benign focal epilepsy. CT scanning and high-resolution magnetic resonance imaging (**MRI**) have led to the recognition of mesial temporal sclerosis, tuberous sclerosis, neuroblast migrational disorders, and small temporal lobe tumours. Positron-emission tomography scanning can demonstrate lesions undetected by MRI, such as focal lesions in patients with hypsarrthymia. Advances in surgical procedures have decreased the risks associated with callosotomies and hemispherectomies used for catastrophic seizures. New understanding about neurotransmitters involved in the production and inhibition of seizures has led to advances in seizure medications.

Epileptic seizures are the result of an imbalance between inhibitory [(γ-aminobutyric acid (**GABA**) and excitatory (glutamate)] neurotransmitter systems. Neuronal hyperexcitability leading to seizures may result from decreased inhibition or increased excitation.[75] Epilepsy has its highest incidence in childhood, suggesting that the immature brain is more vulnerable to seizures than the mature brain—a finding that is borne out by animal studies. Decreased inhibition or increased excitation may result in neuronal excitability and seizures.[75] The specific mechanisms responsible for this imbalance remain uncertain. However, it is known that the binding of GABA to $GABA_A$ receptors opens a chloride channel (ionophore) leading to a flux of chloride ions and consequent membrane hyperpolarization: it is also known that there are fewer $GABA_A$ high-affinity receptors in immature animals. Similarly, there are maturational differences in the development of major

ionotrophic receptors in excitatory systems and in the activation of *N*-methyl-d-aspartate receptors. In younger animals this results in larger excitatory postsynaptic potentials. It remains a puzzle why certain seizure types are age-specific in their onset.[76]

Epilepsy syndromes may have a genetic basis.[77] Gene localization for five epilepsy syndromes with Mendelian inheritance are recognized, and localization has been suggested in three epilepsies with complex inheritance. Those epilepsies with a single gene inheritance include symptomatic epilepsies with associated diffuse brain dysfunction and idiopathic epilepsies, where the seizures are the primary brain abnormality. Idiopathic single gene epilepsies include benign, familial neonatal convulsions. To date four autosomal-dominant forms of epilepsy have been described. Most genes discovered to be involved in human epilepsies encode subunits of ion channels, both voltage-gated and ligand-gated.[77] Molecular genetic studies are expected to lead to the discovery of other epilepsy genes. Investigation of animal models of epilepsy is continuing.

The aetiology of temporal lobe seizures includes mesial temporal sclerosis, tumours, and cortical dysplasia. The younger the child, the less frequent is mesial temporal sclerosis. Other factors linked to aetiology are proposed: temporal lobe hypoperfusion and hypometabolism in Landau–Kleffner syndrome, and diffuse cortical and subcortical hypoperfusion in Lennox–Gastaut syndrome.

Course and prognosis

Early-onset epilepsies are associated with cognitive, behavioural, and communication disorders. Moreover, there is evidence that both clinical and subclinical epilepsy may result in developmental deviance, which has led to earlier and more aggressive treatment to try to prevent these impairments. Psychosocial factors are important in impairment. One prospective study evaluated 220 adults with childhood-onset seizures[78] up to age 35. The majority of subjects were free of seizures as adults, but were at risk for social and educational problems. When compared with random control subjects, those with epilepsy demonstrated correlations between neurological and cognitive impairment and social deficits. Those with epilepsy only (100 subjects) had a fourfold risk of psychiatric disorder. The authors reported social adjustment problems, competence problems, and reduction in marriage rate and fertility. Psychotic disorders occur significantly more frequently in people with epilepsy than in the general population with prevalence rates ranging from 2 to 8 per cent; the prevalence varying with the type of seizure disorder.[79]

Management

Cognitive and behavioural findings suggest the importance of early intervention to prevent negative outcomes. The behavioural and psychiatric problems should be treated with the same approach used in children who are neurologically intact and include educational, family, and pharmacological approaches. The indications and choice of psychiatric drugs is similar; epilepsy is not a strong contraindication for the use of neuroleptic or antidepressants, even though some of these medications may increase the frequency of seizures. Dexedrine may be the treatment of choice for hyperkinetic behaviour because it may increase the seizure threshold. Although caution is needed in those with more severe neurological involvement, there is no strong evidence for an increased risk for neuroleptic-induced tardive dyskinesia. When there are behavioural problems one must consider the behavioural effects associated with anticonvulsant medications.[80] In some instances, reducing the dose or changing the medication may be helpful and this should be discussed with the referring physician.

The major drugs used for treatment include carbamazepine, valproic acid, gabapentin, vigabatrin, and topiramate. These medications are used for the various forms of epilepsy described above including temporal lobe seizures and Lennox–Gastaut syndrome. Lamotrigine is also used, but with caution because severe dermatological side-effects may occur. In some instances, temporal lobectomy has been successful in the control of behavioural dysfunction and illogical thinking when performed in children with intractable temporal lobe seizures. In tuberous sclerosis complex the seizure medication vigabatrin may be helpful (and more so than corticosteroids) for infantile spasms.[81]

Possibilities for prevention

A developmental perspective is indicated as there is increasing evidence of there being a developmental period during which a structure or function can be developed most completely.[82] For example, in tuberous sclerosis complex the cognitive impairment and autistic regression may be approached by way of early drug therapy and, in some instances, by the surgical removal of tubers.[83] Thus a developmental understanding of epilepsy is now crucial in treatment planning. Research is continuing to clarify why, in some instances, epilepsy may have a severe developmental impact and in other instances be more benign. With greater understanding of genetic mechanisms appropriate family counselling will be needed, and perhaps, new drug treatments may emerge. An important treatment goal is to prevent adverse psychosocial outcome by correct diagnosis, early intervention for seizures, continual assessment for cognitive and behavioural disorders, appropriate schooling, as well as effective family support, guidance, and therapy. Careful prospective follow-up studies are needed to demonstrate which interventions are most appropriate to specific types of epilepsy.

Further information

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9.2.2 Specific developmental disorders in childhood and adolescence

Helmut Remschmidt and Gerd Schulte-Körne

Introduction

The term 'specific developmental disorders' includes a variety of severe and persistent difficulties in spoken language, spelling, reading, arithmetic, and motor function. Skills are substantially below the expected level in terms of chronological age, measured intelligence, and age-appropriate education and cannot be explained by any obvious neurological disorder or any specific adverse psychosocial or family circumstances. As the deficits are quite substantial, analogies were initially made to neurological concepts and disorders such as word-blindness, alexia, aphasia, and apraxia, thus giving rise to the notion that neurological deficits are the aetiological basis of these disorders. Since this could not be demonstrated, the next step was to define the disorders in a more functional way,

Table 9.2.2.1 Specific developmental disorders: a comparison of ICD-10 and DSM-IV NOS, not otherwise specified

ICD-10	DSM-IV
Specific developmental disorders of speech and language (F80)	Communication disorders
Specific speech articulation disorder (F80.0)	Expressive language disorder (315.31)
Expressive language disorder (F80.1)	Mixed receptive–expressive language disorder (315.31)
Receptive language disorder (F80.2)	Phonological disorder (315.39)
Acquired aphasia with epilepsy (Landau–Kleffner syndrome) (F80.3)	Stuttering (307.0)
Other developmental disorders of speech and language (F80.8)	Communication disorder NOS (307.9)
Specific developmental disorders of scholastic skills (F81)	Learning disorders
Specific reading disorder (F81.0)	Reading disorder (315.00)
Specific spelling disorder (F81.1)	Mathematics disorder (315.1)
Specific disorder of arithmetical skills (F81.2)	Disorder of written expression (315.2)
Specific disorder of scholastic skills (F81.3)	Learning disorder NOS (315.9)
Other developmental disorders of scholastic skills (F81.8)	
Specific developmental disorder of motor function (F82)	Motor skills disorder
	Developmental co-ordination disorder (315.4)
Mixed specific developmental disorders (F83)	

taking into account not only psychometric testing but also psychosocial risk factors and the quality of schooling and education.

Today, numerous findings support the validity of the diagnostic concept of specific developmental disorders. These disorders and pervasive developmental disorders have the following features in common (ICD-10)[1]:

- An onset that invariably appears during infancy or childhood.
- An impairment or delay in the development of functions that are strongly related to biological maturation of the central nervous system.
- A steady course that does not involve the remissions and relapses that tend to be characteristic of many mental disorders.

Thus the term 'specific developmental disorders' reflects the fact that the deficits are circumscribed and relatively isolated against the background of an otherwise undisturbed psychological functioning.

Classification

In the multiaxial classification of child and adolescent psychiatric disorders,[2] specific developmental disorders are classified on the second axis named 'Specific disorders of psychological development', whereas pervasive developmental disorders are classified on the first axis (clinical psychiatric syndromes).

Based on the history and course of the disorders, two types can be distinguished:

- Disorders in which a phase of previously normal development has occurred prior to manifestation of the disorder. This, for example, applies to the Landau–Kleffner syndrome.
- An additional condition in which the abnormality was present from birth. This is especially true for autism. Autism is classified in the category 'pervasive developmental disorders', which are discussed elsewhere.

In DSM-IV,[3] nomenclature is somewhat different, but generally includes disorders identical or similar to those in ICD-10. In DSM-IV, 'communication disorders' correspond to 'specific developmental disorders of speech and language'. However, they also include stuttering, which is not included in the corresponding category of ICD-10.

'Learning disorders' (DSM-IV) is the category that corresponds to specific developmental disorders of scholastic skills, and 'motor skills disorder' to 'specific disorders of motor function'.

Table 9.2.2.1 shows the terminology used in both classification systems. The headlines of the two systems correspond; however, the subcategories show some differences.

Figure 9.2.2.1 shows a decision tree which includes the three main areas of dysfunction and addresses diagnosis and differential diagnosis.

Specific developmental disorders of speech and language

The main characteristic of these disorders is a disturbance of language acquisition from the early stages of development. The disturbance, however, is not directly attributable to neurological or speech mechanism abnormalities, sensory impairments, intellectual disability, or environmental factors.[1]

There are three main problems in distinguishing these disorders from the normal state and other conditions:

1 **Differentiation from the normal state**: the disorders must be distinguished from normal speech and language development, bearing in mind the great variations seen in the normal pattern. To make the diagnosis, the disorder must clearly be clinically significant, which can be determined by four main criteria: severity, course, pattern, and associated problems.

2 **Differentiation from intellectual disability (mental retardation)**: the degree of speech and language dysfunction must always be considered with respect to the child's cognitive level.

3 **Differentiation from disorders due to sensory impairment or impairments of the central nervous systems**: speech and language disorders resulting from severe deafness, specific

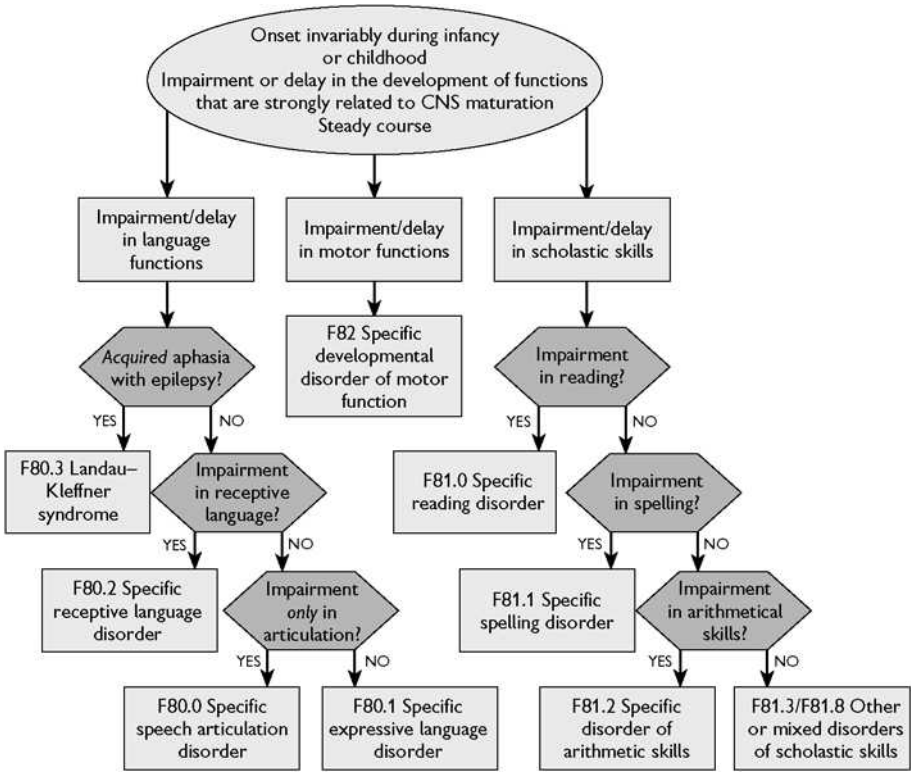

Fig. 9.2.2.1 Specific developmental disorders. CNS, central nervous system.

neurological impairments, or structural brain abnormalities are not classified in the category of specific developmental disorders of speech and language.

Specific speech articulation disorder

(a) Clinical features

The main feature of the disorder is the child's failure to use speech sounds appropriate for his or her mental age, while other language skills are within the normal range. Difficulties include errors in sound production and use, especially substitution of one sound for another. Difficulties in speech sound production usually interfere either with academic achievement or social communication. There are several degrees of severity reaching from mild or no impairment of speech intelligibility to completely incomprehensible speech. Sound substitutions are considered less severe than sound omissions. The sounds most frequently misarticulated are those acquired later during speech development (l, r, s, z, th, ch). However, consonants and vowels that range early in the development sequence may be affected in younger children.

It is very important to relate the misarticulations to normal development. At the age of 4 years, errors in speech and sound production are very frequent, but children are usually understood even by strangers. At 6 to 7 years of age, most speech sounds can be adequately reproduced, and by the age of 11 to 12, children should be capable of almost all speech sounds.

(b) Classification

In ICD-10, speech articulation disorder (F80.0) is classified in the category 'Specific developmental disorders of speech and language'. The counterpart in DSM-IV is the category 'Phonological disorder' (315.39), classified in the category 'Communication disorders' (see Table 9.2.2.1).

(c) Diagnosis and differential diagnosis

The leading feature, the age-appropriate misarticulation of speech sounds with the result that others have difficulties in understanding the child, usually allows one to diagnose the disorder. There are three types of symptoms that can be observed: substitutions, omissions, and distortions of speech sounds. The diagnosis should only be made if the severity of misarticulation is outside the limits of the normal variation for the child's mental age. Further requirements are that non-verbal intelligence and expressive and receptive language skills should be within the normal range.

Differential diagnoses include intellectual disability, hearing impairment, or other sensory deficits or severe environmental deprivation.

(d) Epidemiology

Moderate to severe developmental articulation disorders can be found in 2 to 3 per cent of 6- and 7-year-old children, with less severe disorders even more frequent. The frequency of occurrence falls to 0.5 per cent by the time children are 17 years old (DSM-IV).

(e) Aetiology

As it has been demonstrated that the disorder runs in families, it is assumed that genetic factors are important for its manifestation.

(f) Course and prognosis

The prognosis is favourable if no other associated features such as hearing impairment, neurological conditions, cognitive impairments, or psychosocial problems are present. However, the course

varies depending on the severity and the above-mentioned associated features.

(g) Treatment

Treatment is necessary and appropriate if the child is handicapped in his or her everyday life and cannot be understood by parents, siblings, or other persons. The focus of the therapy depends on whether speech articulation disorder is an isolated phenomenon or if other impairments or dysfunctions are present (e.g. developmental disorder of motor functions). If it is an isolated phenomenon, functional speech therapy can be carried out on the principle that mispronounced sounds should not be repeated when correcting them, but substituted by the correctly pronounced sound.[4] It is advisable to carry out this kind of therapy before the child enters school. If other disorders are present, a comprehensive therapeutic programme that includes speech therapy needs to be developed.

Expressive language disorder

(a) Clinical features and classification

The main feature of this disorder is that the child's ability to use expressive spoken language is reduced below the mental age appropriate level, while language comprehension ranges within normal limits. Abnormalities in articulation may co-occur.

In ICD-10, the following symptoms are considered important for diagnosis (ICD-10, p. 237)[1]:

- delay of the development of expressive language (e.g. absence of single words by the age of 2 years, failure to generate simple two-word sentences by 3 years)
- restricted vocabulary development
- overuse of a small set of general words
- difficulties in selecting appropriate words and word substitutions
- short utterance length and immature sentence structure
- syntactical errors, especially omissions of word endings or prefix
- misuse of or failure to use grammatical features such as prepositions, pronouns, articles, and verb and noun inflexions.

The DSM-IV criteria requires measures of expressive language development being substantially below those obtained from standardized measures of both non-verbal intellectual capacity and receptive language development, interference with academic or occupational achievement, and the exclusion of mixed receptive–expressive disorder and pervasive developmental disorders.

(b) Diagnosis and differential diagnosis

The diagnosis is made by clinical observation, with special emphasis on expressive language functions and the use of individually administered standardized tests of expressive language. The differential diagnosis should rule out mixed receptive–expressive language disorder (DSM-IV), characterized by an impairment of receptive language functions. Autistic disorder may also involve expressed language impairment, but autism can be distinguished by characteristic communication impairments. Finally, intellectual disability and sensory impairments (e.g. hearing impairment or other sensory deficits) need to be ruled out, as well as severe environmental deprivation. The diagnosis is confirmed using intelligence tests, audiometric tests, neurological investigations, and a careful history. Finally, acquired aphasia needs to be ruled out. This can be done by assessing any medical condition that may have caused the disorder.

(c) Epidemiology

In the absence of thorough epidemiological studies, estimates suggest that between approximately 3 and 5 per cent of children may be affected by expressive language disorder of the developmental type. The acquired type seems to be less common.

(d) Aetiology

DSM-IV distinguishes two types of expressive language disorders: the developmental type and the acquired type. In the developmental type, impairment of expressive language begins at a very early age and is not associated with neurological factors, while the acquired type occurs after a period of normal development and is caused by neurological or general medical conditions (e.g. head trauma, encephalitis). It is assumed that the developmental type is caused by genetic factors that influence language development.

(e) Course and prognosis

The course depends on the type of disorder (developmental or acquired type) and severity. Usually, the disorder can be diagnosed by the age of 3 years, while milder forms are often only detected later. According to DSM-IV,[3] approximately half of the children appear to outgrow the developmental type of expressive language disorder, while the other half have persistent difficulties. The outcome of the acquired type depends on the severity and location of the brain pathology.

(f) Treatment

As causal treatment is not possible, treatment measures are based on general principles that have been found to be useful and effective in clinical practice.

1 The first step is to explain clearly to parents the nature of the disorder and the fact that several other disturbances manifested by the child may be a result of the child's communication deficit.

2 The best time to commence speech therapy depends upon the severity of the disorder, the child's cognitive and motivational structure, and other disorders that might be present. Instead of treating children too early (e.g. before the age of 3 years), offering advice and guidance to the parents is extremely important.

3 Treatment itself concentrates on teaching language skills using techniques such as imitation and modelling. The therapist should focus interventions selectively on the areas of difficulty, thus increasing the child's phonological repertoire. Non-verbal communication techniques may be used if verbal communication is substantially impaired. But the therapist should always make sure that non-verbal communication does not dominate the verbal one.

4 In therapeutic programmes, everyday situations are now preferred to very structured programmes. This is because many therapists found that therapeutic progress during sessions was not transferred to everyday life situations. During structured treatment sessions the children are taught to give correct answers to questions that have nothing to do with their situation in everyday life, and it is now thought that structured language training may prevent them using language according to their needs.[5]

5 Alternative communication, such as sign language, should only be used if the child suffers from severe auditory comprehension deficits. The use of a sign language, however, is no longer regarded as an obstacle to the improvement of expressive language skills.[6]

Receptive language disorder

(a) Clinical features and classification

This disorder is characterized by the child's inability or reduced ability to understand language in a way appropriate for his or her mental age. As expressive language production depends on language comprehension; expressive language is also profoundly disturbed and abnormalities in word-sound production can be observed.

The diagnostic guidelines of ICD-10 include the following features:

- failure to respond to familiar names (in the absence of non-verbal clues) by the first birthday
- inability to identify at least a few common objects by 18 months
- failure to follow simple, routine instructions by the age of 2 years
- inability to understand grammatical structures (e.g. questions, comparatives)
- lack of understanding of the more subtle aspects of language (tone of voice, gestures, etc.).

Owing to the disturbances in both receptive and expressive functions, the disorder is called 'receptive–expressive language disorder' in DSM-IV. The diagnostic criteria require scores of both receptive and expressive language development substantially below those obtained from standardized measures of non-verbal intellectual capacity, interference with academic or occupational achievement, and exclusion of pervasive developmental disorders.

(b) Diagnosis and differential diagnosis

Diagnosis is based on three factors: a careful history taken from the child's parents, a thorough clinical investigation including neurological assessment and detailed speech and language assessment, and standardized tests measuring expressive and receptive language functions.

Differential diagnosis should rule out expressive language disorder (which is the case in the presence of language comprehension), specific speech articulation disorder, in which the receptive and expressive language functions are unimpaired, autism (which can be distinguished by the typical communication disturbance), intellectual disability, sensory deficits, and severe environmental deprivation. These disorders can be excluded by intelligence tests, audiometric tests, neurological investigations, and taking a history.

(c) Epidemiology

Owing to the absence of epidemiological studies, the frequency with which the disorder occurs can only be estimated. According to estimations, the disorder occurs in up to 3 per cent of school-age children and is probably less common than expressive language disorder.

(d) Aetiology

As in other developmental language disorders, there is evidence that genetic factors play the most important role in aetiology.[7] The frequent association of disturbed language acquisition with adverse psychosocial factors in the family does not contradict a primarily genetic cause, as many children who grow up under these circumstances show entirely normal developmental patterns of speech and language skills.[8]

(e) Course and prognosis

The long-term prognosis is poor. Only half the patients in the sample studied by Rutter *et al.*[9] had normal conversational skills when they were in their twenties, and there was a decline in non-verbal IQ from childhood to adulthood. The course again depends on the type (developmental or acquired) and severity of the disorder. The disorder is usually detected before the age of 4 years, but earlier in severe cases. The prognosis is poorer than in expressive language disorder. As far as the acquired type is concerned, the prognosis varies depending on severity, location of brain pathology, the child's age, and the level of language development prior to the disorder.

(f) Treatment

Treatment is generally undertaken along the same lines as in expressive language disorders. However, owing to the nature of the disorder, all factors that facilitate language comprehension should especially be encouraged. Non-verbal forms of communication such as sign language can be helpful.

Acquired aphasia with epilepsy (Landau–Kleffner syndrome)

(a) Clinical features and classification

The Landau–Kleffner syndrome is a rare disorder characterized by receptive and expressive language impairment and epileptic seizures, but retained general intelligence, and manifestation after a period of normal development, including language development. The onset of the disorder typically occurs between 3 and 7 years of age and is accompanied by paroxysmal electroencephalographic abnormalities, mainly bilateral spikes in the posterior temporal and parietal regions and epileptic seizures in about 80 per cent of cases.[10] The loss of language may occur gradually over a period of months or abruptly within a few days or weeks.

Aphasia usually starts with receptive language problems occurring together with the characteristic electroencephalographic changes, followed by expressive language difficulties. Usually, the first sign is the impairment of receptive language functions with difficulties in auditory comprehension. During the manifestation period, symptomatology is variable: some children become mute, others express jargon-like sounds, and some produce misarticulations and have difficulties in word fluency. During the manifestation period, emotional and behavioural symptoms are common; they can be regarded as a reaction to the loss of language functions and appear as anxiety reactions, acting-out behaviour, and aggression.

In DSM-IV, the condition is classified in the category mixed receptive–expressive language disorder, acquired type.

(b) Diagnosis and differential diagnosis

The diagnosis can be based upon a detailed history of the child's development, assessment of language functions, careful neurological assessment, and by electroencephalography. The differential diagnosis includes other types of acquired aphasia without epileptic seizures and electroencephalographic abnormalities, and

disintegrative disorders of childhood such as dementia infantilis Heller (Heller's syndrome) (see Chapter 9.2.2).

(c) Epidemiology

The prevalence of the disorder is unknown.

(d) Aetiology

The aetiology is unknown. There is, so far, no indication of any genetic cause. Clinical characteristics suggest that an encephalitis might be considered a causal mechanism. The electroencephalographic changes and the seizures are thought to cause the language and the behavioural and emotional problems.

(e) Treatment

So far, three different approaches to treatment have been undertaken:

1 Anticonvulsant treatment (mainly with carbamazepine): the frequency of seizures can be reduced to some extent with this medication, but paroxysmal electroencephalographic changes are not substantially influenced.

2 Corticosteroids have also been administered,[11] but the benefits remain unclear.

3 Finally, surgical treatment by bilateral temporal transection has been attempted.[12,13]

Specific developmental disorders of scholastic skills

'Specific developmental disorders of scholastic skills' (ICD-10) or 'Learning disorders' (DSM-IV) include disorders characterized by one or more significant impairments in acquisition of reading, spelling, or arithmetical skills. ICD-10 suggests that the category 'Mixed disorder of scholastic skills' (F81.3) be used as an ill-defined, but necessary, category in which arithmetical and reading or spelling skills are significantly impaired, although not because of general intellectual disability or inadequate schooling.

The disorders classified in the category 'Specific developmental disorders of scholastic skills' (**SDDSS**) resemble specific disorders of speech and language. As in these latter disorders, normal patterns of skill acquisition are disturbed and detectable at an age when these functions are required. The disorders are not due to a lack of opportunity to learn or a consequence of brain trauma or disease, but represent a specific type of dysfunction in cognitive processing. The dysfunction affects specific skills, which can be distinguished from the cognitive functions that are usually in the normal range. As in other specific developmental disorders, the condition is more common in boys than in girls.

ICD-10 notes five difficulties regarding diagnosis and differential diagnosis:

1 differentiation of the disorder from normal variations in scholastic achievement (this problem applies to all specific developmental disorders and was discussed in relation to specific developmental disorders of speech and language);

2 consideration of the normal developmental course;

3 interference with learning and teaching;

4 underlying abnormalities in cognitive processing;

5 uncertainties over the best way of subdifferentiating SDDSS.

Based on these considerations, the following diagnostic guidelines for all SDDSS have been suggested (ICD-10):

- **Clinically significant degree of impairment:** this is judged on the basis of severity (e.g. occurrence in less than 3 per cent of school children), developmental precursors (e.g. speech or language disorder in preschool years), and associated problems (e.g. inattention).
- **Specific impairment not explained solely by intellectual disability or by lesser impairments in general intelligence:** for this requirement to be met, individually administered and standardized IQ scholastic achievement tests are obligatory to demonstrate that the child's level of achievement is substantially below the expected level compared to a child of the same mental age and IQ.
- **Developmental nature of the impairment:** this must be demonstrated by the presence of the disorder during the early years of schooling and by exclusion of impairment acquired later. The child's history of school progress is decisive in this respect.
- **Absence of external factors that could explain the impairment:** SDDSS are thought to be mainly based on factors intrinsic to the child's development, and not due to inadequate schooling or any other environmental factors such as absence from school or educational discontinuities. However, such conditions may occur, making the diagnostic process difficult.
- **Exclusion of visual and hearing impairments:** by definition, SDDSS do not occur as a result of impairment of sensory function, such as visual or hearing impairment.

The main differential diagnostic task is distinguishing SDDSS from neurological disorders (e.g. alexia, aphasia, agraphia, apraxia) or impairments that also could influence the development of scholastic skills (e.g. emotional disorder). In cases of normal child development prior to the manifestation of a defined neurological disorder, differential diagnosis is not difficult. However, if minor neurological signs (soft signs) were diagnosed previously, independent of any defined disorder, and the findings persist, it may be difficult to distinguish recent symptoms from previous ones. In such cases, associated disorders or symptoms should be classified separately in the appropriate neurological section of the classification systems.

Specific reading disorder

The ICD-10 classification system distinguishes between 'Specific reading disorder' and 'Specific spelling disorder'. In DSM-IV, 'Specific reading disorder' is distinguished from 'Disorder of Written Expression'. The latter is not identical with the ICD-10 category 'Specific spelling disorder', insofar as that disorder excludes children whose sole problem is one of handwriting.

(a) Clinical features and classification

The main feature of this disorder is a specific and significant impairment in the development of reading skills, which is not solely accounted for by mental age, visual acuity problems, or inadequate schooling.[1]

Other functions may also be affected:

- word recognition
- reading comprehension skills

- oral reading skills
- performance of tasks requiring reading

In many cases, spelling difficulties continue into adolescence and persist in adulthood, even when reading skills improve considerably. The history of children with specific reading disorder frequently reveals a specific developmental disorder of speech and language. Symptoms of these disorders may still be present at elementary school when the specific reading disorder is first diagnosed. Additional frequently associated problems include poor school attendance and problems with social adjustment.

The DSM-IV criteria for reading disorder state that reading achievement as measured by standardized tests should be substantially below the expected level and that the disturbance should interfere with academic achievement or activities of daily living that require reading skills.

(b) Diagnosis and differential diagnosis

The diagnosis is made on the basis of the ICD-10 and/or DSM-IV criteria, which are similar. The ICD-10 diagnostic guidelines require the following:

- A reading performance below the level that is expected on the basis of age, general intelligence, and school placement. For clinical purposes, usually 1.5 standard deviations below the expected level is regarded as a requirement for the diagnosis. For research purposes, two standard deviations are used.
- Performance to be assessed by individually administered standardized tests of reading accuracy, latency, and comprehension.
- Errors demonstrated in oral reading skills and deficits in reading comprehension. Errors in oral reading include:
  - (a) omissions, substitutions, distortions, or additions of words or parts of words;
  - (b) slow reading rate;
  - (c) false starts, long hesitations, or 'loss of place' in text, and inaccurate phrasing;
  - (d) reversals of words in sentences or of letters within words.

Deficits in reading comprehension include:

- (a) an inability to recall facts that have been read;
- (b) inability to draw conclusions or interferences from material that has been read;
- (c) use of general knowledge as background information, rather than of information from a particular story, to answer questions about a story that has been read.

(c) Comorbidity and associated features

It is important to analyse the features of the disorder with a longitudinal perspective. Thus, several associated disorders can be observed: emotional problems during the early school years; hyperactivity and conduct disorders in later childhood and adolescence. Additional frequent features include low self-esteem, adjustment problems at school, and problems in peer relationships. In about 40 per cent of children with reading and/or spelling disorder, other disorders of clinical relevance are present. After finishing school, this rate decreases to 30 per cent, which includes a high proportion of antisocial behaviour and delinquency.[14]

(d) Epidemiology

Specific reading and spelling disorder occur in about 4 per cent of 8- to 10-year-old children, when defined as two standard deviations below non-verbal IQ.[15,16] By using a wider definition, the rates are approximately 7 to 8 per cent, with a predominance of boys (2:1).

(e) Aetiology

Currently, four main aetiological factors have been discussed:

1 genetic influences;

2 deficits in central information processing;

3 general psychosocial factors;

4 specific learning conditions.

Familial clustering in dyslexia was recognized a few years after the first description of the disorder by Hinselwood in 1895. A child with an affected parent has a risk of between 40–60 per cent of developing dyslexia.[17] This risk is increased when other family members are also affected. There is estimated to be a 3 to 10-fold increase in the relative risk for a sibling (λ_s), with an increase in λ_s being observed when stricter criteria are applied.[18]

Twin studies have confirmed that genetic factors are substantially responsible for the familial clustering of dyslexia. It is generally accepted that the proportion of inherited factors involved in the development of dyslexia is between 40–80 per cent, the highest estimates being reported for the phenotype dimensions word reading (up to 58 per cent) and spelling (70 per cent).[19] Whereas shared environmental effects are low for word reading, for with reading, and spelling correlated traits, for example, phonological awareness, shared environmental is substantially higher at about 14 per cent. Based on genome-wide linkage analyses nine candidate gene regions (DYX1-9) could be identified. Most replicated are two regions, 6p22, and 15q21. More recently, four candidate genes, DCDC2, KIAA0319, ROBO1, and DYX1C1 were identified by systematic association analyses. All these genes play a functional role in neuronal migration making them promising candidate genes for dyslexia. However, a functional relevant mutation has not been identified yet.[19]

The hypothesis of deficits in central information processing is based on behavioural and neuroimaging studies that identified cognitive processes that are impaired in dyslexics individuals. These are impaired visual processing, auditory processing, speech perception, phonologic processing, orthographic processing, and motor coordination.[20] The mainstream hypothesis is a phonological processing deficit supported by longitudinal, intervention, and brain imaging studies. Among the latter, the importance of left hemispheric specialization has been widely discussed in the literature, either suggesting maturational lag of the left hemisphere or a structural deficit in white and grey matter.[20]

General psychosocial factors may also play a role in the manifestation of specific reading and spelling disorder. However, the influence seems to be rather marginal. The same applies, more or less, to the special learning condition, because severe deficits in schooling are excluded by definition as a main cause of these disorders. However, given a genetic predisposition for specific reading or spelling disorder, poor learning conditions at school and at home may contribute to the manifestation of these disorders. In summary, the different factors responsible for the manifestation of specific

reading and specific spelling disorder can be best understood in terms of a vulnerability model, in which several genetic predispositions, on the one hand, and general psychosocial factors and special learning conditions, on the other, interact with one another.

(f) Course and prognosis

More than 40 longitudinal follow-up studies have shown similar results:

- There is a high persistence of reading and spelling problems, phonological difficulties, and slowness in word recognition.[23,24]
- Retarded readers make poorer progress than backward readers.[25]
- On the other hand, considerable progress is possible in oral reading and reading comprehension.[26]

As far as schooling is concerned, a substantial proportion of children with specific reading disorder remain behind the school level of their age group. However, there is a difference regarding social background. Children from middle-class homes more frequently show a positive educational outcome compared with children from socially disadvantaged homes.[27]

An epidemiological study in Germany[16] showed that only 3 per cent of children with specific reading disorder were able to attend high school and more than 50 per cent remained at the lowest normal school level. At the age of 18, the rate of unemployment was three times as high as in a normal control group.

(g) Treatment

As mentioned above, treatment is difficult because the disorder tends to persist. Nevertheless, the following principles have been found to be useful:

1 Treatment should start as early as possible in order to avoid a sense of failure and low self-esteem.

2 The treatment should focus on individual instruction and teaching sessions in basic phonetic and other skills such as reading, spelling, and writing. This needs to be done in an age-appropriate way based on the principles of learning theory and starting at a very low level to avoid disappointment and a sense of failure. There are several programmes used in different countries based on these principles, sometimes using computers.

3 Although very popular methods based on training basic perceptual skills like figure-ground discrimination, tone discrimination, temporal auditory processing have not been proven by empirical studies.

4 Even when feelings of failure and, consequently, low self-esteem are present, the instruction in basic skills is the appropriate approach. The child's psychological and learning situation deserves special attention. Psychotherapeutic measures alone are not successful.

5 Parental support is extremely important. Therefore, the parents should not only be educated in detail about the disorder, but also encouraged to listen to their children reading from school books. This has been shown to be a successful approach.[28]

6 There is no specific medication to improve reading and spelling skills, but there is some indication that stimulants may be helpful for poor readers who simultaneously suffer from attention-deficit hyperactivity disorder.

Specific spelling disorder

(a) Clinical features

In ICD-10, the main characteristic of this disorder is a specific and significant impairment in the development of spelling skills in the absence of a history of specific reading disorder, which is not solely accounted for by low mental age, visual acuity problems, or inadequate schooling. The children have difficulties in spelling orally and writing words correctly. For this diagnosis, the following criteria are required (ICD-10):

- The spelling performance of the child should be significantly below the expected level regarding age, general intelligence, and school placement. This has to be assessed individually by administration of a standardized spelling test.
- The reading skills should be within the normal range and there should be no history of preceding reading difficulties.
- The spelling difficulties should not be due to grossly inadequate teaching, to sensory deficits, or to neurological disorders or dysfunctions. They should not be acquired, either as a result of neuropsychiatric or any other disorders.

In DSM-IV, there is no category that corresponds exactly to the ICD-10 category 'Specific spelling disorder'. The DSM-IV category that most closely resembles 'Specific spelling disorder' is 'Disorder of written expression', as defined by measured writing skills substantially below the expected level, interference with academic achievement, and, in the case of a sensory deficit, writing skills greater than the difficulties usually associated with sensory deficits.

It is uncertain whether a pure spelling disorder as described in ICD-10 actually exists or whether spelling skills usually overlap with other functions that constitute scholastic skills. It is, however, possible to assess the different functions separately, as several studies show.

(b) Diagnosis and differential diagnosis

The diagnosis is made according to the criteria mentioned above and by the administration of standardized spelling tests.

Specific disorders of arithmetical skills

(a) Clinical features

The main clinical feature of this disorder (also called dyscalculia) is a specific impairment in arithmetical skills that cannot be explained on the basis of general intellectual disability or inadequate schooling. Dyscalculia is a difficulty in learning and remembering arithmetic facts and executing calculation procedures, with immature problem-solving strategies, long solution times, and high error rates.[29] A number of different skills may be impaired, as understanding or naming mathematical terms, operations, or concepts, and decoding written problems into mathematical symbols.

The impairment affects basic computational skills of addition, subtraction, multiplication, and division, whereas other functions such as reading and writing or motor skills are within the normal range (except in mixed disorder of scholastic skills). The arithmetical difficulties vary, but in most cases include the following features (ICD-10):

- difficulties in understanding the concepts underlying arithmetical operations
- difficulties or lack of understanding of mathematical terms or signs
- difficulties in recognizing numerical symbols

- difficulties in carrying out arithmetical manipulations
- difficulties in aligning numbers or symbols when performing calculations
- poor spatial organization of arithmetical calculations
- reduced ability to learn multiplication tables satisfactorily.

The diagnosis is made according to ICD-10 (Specific disorders of arithmetical skills) or DSM-IV criteria (Mathematics Disorder) and the diagnostic guidelines. The ICD-10 guidelines require the following criteria:

- The child's arithmetical performance should be significantly below the expected level on the basis of age, general intelligence, and school placement, assessed by an individually administered standardized arithmetical test.
- Reading and spelling skills should be within the normal range expected for the child's mental age, also tested by an individually administered standardized test.
- The difficulties in arithmetical skills should not be mainly due to grossly inadequate teaching or the direct effects or defects of visual, hearing, or neurological functions and should not be acquired as sequelae of neurological, psychiatric, or other disorders.

The DSM-IV criteria follow the same principles as with other specific developmental disorders. Required criteria include mathematical ability substantially below the expected level, interference with academic achievement, and in the case of a sensory deficit, the difficulties in mathematical ability greater than the difficulties usually associated with sensory deficits.

(b) Diagnosis and differential diagnosis

The diagnosis is made according to the above-mentioned criteria in the ICD-10 and DSM-IV systems. Differential diagnosis must rule out acquired arithmetical disorder (acalculia), arithmetical difficulties associated with a reading or spelling disorder, and arithmetical difficulties as a result of inadequate teaching.

(c) Epidemiology

It is estimated that between 3 and 6 per cent of all schoolchildren suffer from a specific arithmetical disorder.[30] The sex ratio is approximately equal, in some studies girls are found to be more often affected than boys.[31] An important correlate of maths disorder is dyslexia. It is estimated that 40 per cent of dyslexics also have maths disorder.[31]

(d) Aetiology

Dyscalculia has not been studied with the same intensity as dyslexia. Therefore, knowledge about aetiology, course, and outcome is limited.

Numerical abilities, including arithmetic, are mediated by areas in the parietal lobe. In functional imaging studies performed during mental calculation tasks, a pattern of bilateral activation in the prefrontal, premotor, and parietal cortices has been observed.[32] Neuropsychological evidence indicates that numerical processing is localized to the parietal lobes bilaterally, in particular the intra-parietal sulcus[33] and is independent of other abilities. A reduced volume of grey cortical matter in dyscalculic individuals was found in the sulcus intraparietalis of the left hemisphere.[34,35]

There have been a few studies into genetic contributions to mathematical cognitive ability. Most have studied the possible genetic aetiology of mathematics disorder, at least partly because of its comorbidity with reading disorder.[36]

Results from twin studies were consistent with a genetic basis for mathematics disorder whether combined with reading disability or not and estimates of high heritability of mathematical ability were obtained in a sample of twins with normal intelligence ascertained for reading disability and family samples.[37,38]

So far developmental dyscalculia is likely to be the result of the failure of these brain areas to develop normally and can best be defined as a deficit in the representation or processing of specifically numerical information.[39]

A substantial proportion of children with specific disorder of arithmetical skills have associated emotional problems and difficulties in social interactions. It is not quite clear whether these difficulties are secondary complications of the specific difficulties of arithmetical skills.

(e) Course and prognosis

As a matter of fact, specific disorder of arithmetical skills is only diagnosed at the end of the first year or the beginning of the second year of elementary school, because of the necessity of these skills at that time. Especially in cases when the disorder is associated with a high IQ, children may initially compensate for the difficulties with the result that the dysfunction is discovered only in the third year of elementary school or later. According to some studies,[40,41] children with specific disorder of arithmetical skills show poor visuospatial abilities and also have difficulties in complex and motor tasks. Share *et al.*[42] compared these results with their own. Deficits of right hemisphere functioning were found, but only in boys. These results suggest that boys and girls with specific disorder of arithmetical skills should be studied separately. A further interesting result is the association of the disorder with anxiety.[43] This association is more pronounced in those children in whom arithmetical skills are substantially impaired compared with relatively good reading and spelling skills.

(f) Treatment

The treatment of specific disorders of arithmetical skills follows the same general lines as treatment of specific reading disorder. All treatment components have to focus on the training of skills that are impaired in a way that keeps the child motivated. Treatment strategies should focus on the mathematical disability itself or the mathematics anxiety with which the disorder is frequently associated. In many cases, both facets need to be included in the treatment programme. When treating the mathematical disability according to the child's individual profile of impairment, four aspects should be emphasized: semantic memory, procedural difficulties, visuospatial difficulties, and difficulties with mathematical problem-solving.[44] Mathematics anxiety requires a more psychotherapeutic approach using relaxation techniques, with the aim of reducing anxiety prior to and during maths lessons in order to avoid a sense of failure.

Mixed disorders of scholastic skills

In ICD-10, this is specified as an ill-defined and inadequately conceptualized, but necessary residual, category of disorders in which both arithmetical and reading or spelling skills can be significantly

impaired, and in which the disorder cannot be explained in terms of general intellectual disability or inadequate schooling. This category covers disorders that meet the criteria of 'Specific disorder of arithmetical skills' (F81.2) and either 'Specific reading disorder' (F81.0) or 'Specific spelling disorder' (F81.1). As has been explained earlier, in the case of a mixed disorder of scholastic skills, it is specific arithmetical disorder that seems to dominate both in severity and with respect to associated psychopathological features.

Specific developmental disorder of motor function

Clinical features and classification

Many children to whom this category applies, were previously diagnosed as having 'minimal brain dysfunction'. This term is no longer used. The essential clinical features of the disorder include the following (ICD-10):

- An impairment of motor coordination that is significantly below the expected level on the basis of age and general intelligence assessed by an individually administered and standardized test.
- The difficulties in coordination should already have been present early in development.
- They should not be acquired and not be a direct result of deficits of vision or hearing or of any neurological disorder.

Variability of fine or gross motor coordination is great. The milestones of motor development are usually delayed. In many cases, there is an association with speech difficulties, especially articulation. Parents usually report that the child was slow in learning to sit, run, hop, climb stairs, and ride a bicycle. Many children also have difficulties in learning to tie shoelaces, fasten and unfasten buttons, and throw or catch balls. Some children may be generally clumsy in fine and gross movements others tend to have their main difficulty with fine movements and coordination. In many cases, drawing skills are also impaired and the child's difficulties are particularly obvious during ball games, which require a considerable amount of gross motor coordination.

The DSM-IV criteria are similar, and emphasize a substantial backlog of motor coordination, significant interference with academic achievement, and require general medical conditions and pervasive developmental disorders to be ruled out.

There is growing evidence that specific developmental disorders of motor function are a quite heterogeneous group that needs to be subclassified.[45]

Diagnosis and differential diagnosis

Diagnosis is made according to the criteria and guidelines in ICD-10 and DSM-IV. Differential diagnosis should rule out specific neurological disorders, which can be done with a careful history and neurological examination, pervasive developmental disorders, which can be distinguished by the criteria of these disorders, or attention-deficit hyperactivity disorder. The latter can be distinguished by their pronounced distractibility and impulsivity rather than impairment of motor coordination.

Epidemiology

According to an estimation in DSM-IV about 6 per cent of 5- to 11-year-old children suffer from the disorder.

Aetiology

There are two main factors said to be responsible for the aetiology, genetic influences and brain damage. As far as genetic influences are concerned, there are no valid studies that confirm this assumption. Regarding brain damage, the question arises whether early brain damage can result in a specific impairment of motor functions, while other functions are within normal limits.[46–48] As this appears very unlikely, comorbidity with other disorders should be considered the norm and specificity regarded as the exception.[49]

Course and prognosis

The few follow-up studies have shown that children who suffer from the disorder between 5 and 10 years of age show a high persistence of motor problems in adolescence. Almost all children who were identified as having had motor difficulties at elementary school, had similar problems as teenagers.[50–52]

Treatment

Treatment measures should focus on two main facets: the difficulties and impairments in the different motor functions, and the associated social and emotional difficulties. The first facet requires an active functional treatment of motor functions focusing on the child's individual difficulties. Programmes are available for this kind of treatment, which is usually carried out by physiotherapists and occupational therapists.[53]

In treating the second facet, therapists are confronted with the child's insecurity, which is a direct result of motor difficulties, poor body scheme, avoidance reactions of peers and classmates, and frequent feelings of inferiority and a low self-esteem.[54] These associated problems need to be addressed in psychotherapy, in addition to programmes that focus solely on training motor functions.

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9.2.3 Autism and the pervasive developmental disorders

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Introduction

The pervasive developmental disorders (PDDs) are characterized by patterns of deviance and delay in social-communicative development in the first years of life, which are associated with restricted patterns of interest or behaviour. The prototypic PDD is childhood autism; other conditions included in the PDD class in ICD-10[1] include Rett's syndrome, childhood disintegrative disorder, Asperger's syndrome, and atypical autism. Except for one additional category in ICD-10 (hyperkinetic stereotyped movement disorder), the disorders included in ICD-10 and DSM-IV[2] are essentially identical. In this chapter each of these conditions will be reviewed in terms of their clinical features, definition, epidemiology, course, and aetiology; final sections of the chapter address aspects of treatment and prevention for the group of disorders as a whole (Box 9.2.3.1).

Childhood autism

Autism was first recognized by Kanner[3] in his report of 11 children with 'autistic disturbances of affective contact'. He emphasized two essential diagnostic features: autism and difficulties with change; but he also noted atypical language (when language was present at all). He used Bleuler's term 'autistic' to convey the children's social isolation. Although children with autism had undoubtedly been previously observed,[4] it was Kanner's particular genius to so precisely describe the condition. False leads for research arose since the term autism introduced an unintended confusion with schizophrenia. Also, Kanner assumed that the children had normal intellectual potential. Subsequently, it became clear that autism and schizophrenia were distinct and that autism was often associated with intellectual disability.[5] Although describing autism as inborn, Kanner mentioned that parents were very well educated and successful. In turn this led to the idea, common during the 1950s, that autism might somehow result from deviant patterns of care by unusually successful parents. A large body of evidence shows that this is most certainly not the case.[6] It is clear that families of children with autism come from all social classes and circumstances[7] and that the original impression had been the result of referral bias.

Clinical features

Social deficits of a particular type remain a hallmark of autism. The nature of this deficit varies, somewhat, over time but remains a source of great disability to the affected individual throughout life.[8] In younger and more impaired individuals there may be little interest in social interaction; less impaired individuals may come to a passive acceptance of social interaction while older and more cognitively able individuals are more likely to seem highly eccentric and one-sided.[9] Difficulties are observed in the use of eye contact or other non-verbal social cues, in social emotional reciprocity and empathy, in activities involving shared interests with others, and with peer relationships (see Table 9.2.3.1). As Rutter[10] suggested, these problems do not simply reflect cognitive impairment, which is present in about 60 per cent of individuals affected. Abnormalities in communication (not only in language) are also observed. In a substantial minority (perhaps 30 per cent) of cases, the child never acquires the capacity for communicative speech; among individuals who do talk, various unusual features of language are observed such as echolalia, idiosyncratic language, deficits in prosody, and pronoun reversal.[11] Deficits in pragmatic language are particularly striking. As with the social disturbance, the deficits observed are not solely due to intellectual disabilities. Various unusual behaviours are subsumed under the term 'resistance to change'. These behaviours include literal resistance to change ('insistence on sameness'), stereotyped and repetitive motor mannerisms, strict adherence to non-functional routines, and interest in non-functional parts of objects. Various other features may be observed, such as unusual sensitivities to aspects of the environment and attachments to unusual objects.

Box 9.2.3.1 The pervasive developmental disorders

- Childhood autism/autistic disorder
- Rett's disorder
- Childhood disintegrative disorder
- Asperger's disorder
- PDD-NOS/atypical autism

Definition

In the 1950s and 1960s there was disagreement about autism, e.g. was it a form of schizophrenia or psychosis? Gradually evidence began to accumulate that suggested the role of central nervous system dysfunction in pathogenesis—for example, high risk for developing seizures. Differences in clinical features, onset and course, and family history also supported the distinctiveness of autism apart from childhood schizophrenia.[12,13] By 1978, there was a substantial body of work on the validity of autism. Rutter synthesized this in his influential definition of autism,[10] which required the presence of patterns of delay and deviance in the areas of social and language development that were not simply the result of developmental delay along with the group of unusual behaviours subsumed under Kanner's term 'insistence on sameness'. Onset before 30 months of age was required. In ICD-9[14] infantile autism was still termed a psychotic condition but by 1980 the highly influential DSM-III[15] appeared and recognized autism as a condition apart from schizophrenia, including it in a new class of disorders—the pervasive developmental disorders. The latter term has been the topic of some debate, although a better term has yet to be proposed and, in any case, the term PDD has now come into general usage in both DSM and ICD.[16,17]

The name chosen in DSM-III ('Infantile autism') was consistent with Kanner's original report but reflected a lack of developmental orientation; these concerns were addressed in DSM-III-R,[18] which provided a detailed, and more developmentally oriented, set of diagnostic guidelines. Unfortunately, this definition proved overly inclusive and it became apparent that additional work would be needed. Further impetus was given to this effort by pending changes in ICD-10[1] and DSM-IV. Accordingly, an international field trial was undertaken.[19] Based on the results of this field trial

Table 9.2.3.1 ICD-10 criteria for childhood autism (F84.0)

A. Abnormal or impaired development is evident before the age of 3 years in at least one of the following areas:
1 receptive or expressive language as used in social communication;
2 the development of selective social attachments or of reciprocal social interaction
3 functional or symbolic play
B. A total of at least six symptoms from (1), (2), and (3) must be present, with at least two from (1) and least one from each of (2) and (3)
1 Qualitative impairment in social interaction is manifested in at least two of the following areas:
(a) failure adequately to use eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
(b) failure to develop (in a manner appropriate to mental age, and despite ample opportunities) peer relationships that involve a mutual sharing of interests, activities, and emotions
(c) lack of socio-emotional reciprocity as shown by an impaired or deviant response to other people's emotions; or lack of modulation of behaviour according to social context; or a weak integration of social, emotional, and communicative behaviours
(d) lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g. a lack of showing, bringing, or pointing out to other people objects of interest to the individual)
2 Qualitative abnormalities in communication in at least one of the following areas:
(a) delay in or total lack of development of spoken language that is not accompanied by an attempt to compensate through the use of gestures or mime as an alternative mode of communication (often preceded by a lack of communicative babbling)
(b) relative failure to initiate or sustain conversational interchange (at whatever level of language skill is present), in which there is reciprocal responsiveness to the communications of the other person
(c) stereotyped and repetitive use of language or idiosyncratic use of words or phrases
(d) lack of varied spontaneous make-believe play or (when young) social imitative play
3 Restricted, repetitive, and stereotyped patterns of behaviour, interests, and activities are manifested in at least one of the following:
(a) an encompassing preoccupation with one or more stereotyped and restricted patterns of interest that are abnormal in content or focus; or one or more interests that are abnormal in their intensity and circumscribed in nature though not in their content or focus
(b) apparently compulsive adherence to specific non-functional routines or rituals
(c) stereotyped and repetitive motor mannerisms that involve either hand or finger flapping or twisting or complex whole-body movements:
(d) preoccupations with part-objects or non-functional elements of play materials (such as their odour, the feel of their surface, or the noise or vibration they generate)
C. The clinical picture is not attributable to the other varieties of pervasive developmental disorders; specific developmental disorder of receptive language (F80.2) with secondary socio-emotional problems, reactive attachment disorder (F94.1), or disinhibited attachment disorder (F94.2); mental retardation (F70–F72) with some associated emotional or behavioural disorders; schizophrenia (F20.-) of unusually early onset; and Rett's syndrome (F84.12).

Taken from Disorders of psychological development ***(criteria for research)***, pp. 154–5. © World Health Organization, www.who.int

autism is defined (see Table 9.2.3.1) on the basis of characteristic problems in three areas: social interaction, communication and play, and restricted patterns of interest. By definition, autism must be present by the age of 3 years. ICD-10 provides for various ways in which a diagnosis of atypical autism can be made—for example, because of failure to meet age of onset or behavioural criteria. Data on this system suggest that it has good agreement with the diagnoses of experienced clinicians, avoids the problem of the overdiagnosis of autism in the most mentally handicapped persons, and has reasonably good reliability.

Epidemiology and demographics

Over 40 epidemiological studies of autism and related conditions have been conducted with prevalence estimates ranging from 0.7 per 10 000 to 72.6 per 10 000.[20] In his recent 2005 review, Fombonne notes that prevalence rates are negatively correlated with sample size and there is an apparent trend for increased rates over time. Various considerations (including changes in definition) complicate the interpretation of increased rates. In this review, Fombonne suggests that a reasonable estimate of prevalence is 13 per 10 000 (Box 9.2.3.2). Although higher rates of 1 in 150 are reported if broad definitions are used.

Box 9.2.3.2 Epidemiology of autism and related conditions

Autism. . . . . .13 per 10 000
Rett's disorder. . . . . 1 per 10 000 girls
Childhood disintegrative disorder . . . . . .1.9 per 100 000
Asperger's syndrome . . . . . .4 per 10 000
PDD-NOS/atypical autism . . . . . .1 per 150

Source: Cohen, D.J. and Volkmar, F.R. (eds.) (2005) ***Handbook of autism and pervasive developmental disorders*** (3rd edn.) Wiley, New York.

A number of studies, including both epidemiological and clinically referred samples, report higher rates of autism in boys than in girls (typically 3.5:1 or 4:1). This ratio varies with IQ level, i.e. females with autism who have IQs in the normal range are probably

20 times less common than males.[21] The explanation for this sex difference remains unclear. It is possible that the degree of insult required to produce autism in females must be greater than for males, but other hypothesis have been raised. Ethnic and cultural differences have been little studied.[20,22]

Course and prognosis

Although childhood autism is a chronic disability, there is some suggestion that with early intervention and remediation outcome is improving.[23] For example, the number of individuals with either a 'good' or 'fair' outcome is now about 50 per cent—a noteworthy increase since 1980.[24] However, even in the highest functioning individuals marked social problems persist.

Changes in the degree of social relatedness, communication, and self-help skills are observed with increases in developmental level. Seizure disorders are observed in up to 25 per cent of individuals and may have their onset at any point, but adolescence and early childhood are particularly common times.[25] Factors that predict long-term outcome include the presence of some communicative speech by the age of 5 or 6 years, and non-verbal intellectual level.

Aetiology

Early interest centred on the possibility that experiential factors might somehow cause autism, but a host of research findings suggests that this is not the case. Rather, a fundamental disturbance in the central nervous system is implicated.

(a) Psychological factors

Disabilities affecting attentional mechanisms, arousal, sensory deficits, memory management, and complex information processing, among others, have been proposed as 'primary' deficits underlying the social impairment in autism. Although each of these helps us understand some aspects of the condition, none has, as yet, provided a more comprehensive account of the condition as a whole.[26] Among the most influential recent theories attempting to do that is the hypothesis that posits a lack of a central drive for coherence in children with autism, with the consequent focus on dissociated fragments of their environment rather than integrated 'wholes', leading to a fragmentary and overly concrete experience of the world.[27] Another cognitive account of autism posits that the commonly found difficulties in abstracting rules, inhibiting irrelevant responses, shifting attention, and profiting from feedback as well as in maintaining relevant information 'on-line'—the so-called 'executive functions'—underlie the social, communicative, and behavioural disabilities in autism.[28] Although both these theories—'weak central coherence' and 'executive dysfunction'—provide insightful new views of well-known clinical features, neither phenomena can be seen as specific to autism relative to other developmental disorders.

Probably the most influential current cognitive hypothesis focuses on mechanisms directly impacting on social understanding. This view, the 'theory of mind' hypothesis, posits that autism is caused by the child's inability to attribute mental states such as beliefs and intentions to others. Devoid of this ability, individuals with autism are thought to be unable to infer the thoughts and motivations of others, thus failing to predict their behaviour and adjust their own actions accordingly, which results in a lack of reciprocity in communication and social contact.[29] Although more than 50 studies have documented such deficits in autism, there are still many limitations to this hypothesis. For example, more able individuals with autism do exhibit 'theory of mind' skills—and yet may be totally unable to utilize this capacity in their spontaneous social adjustment. Such phenomena suggest that factors other than a cognitive understanding of mental phenomena are required for a person to meet the demands of everyday social life. For example, the 'enactive mind' hypothesis focuses on early emerging and highly conserved mechanisms of socialization that precede the advent of mentalizing abilities, and which culminate in the development of joint attention and perspective taking skills.[30] Of great interest in the past few years has been the confluence of experimental psychological paradigms and functional neuroimaging studies focusing on the same constructs. This new trend is leading to new insights into brain systems subserving basic social mechanisms such as gaze behaviour, face processing, social-affective responses, and thinking about other people's intentions and beliefs,[31] all of which are greatly compromised in autism.

(b) Biological factors

The importance of biological factors in the pathogenesis of autism is suggested by several lines of evidence. Autism has been associated with a host of medical conditions; but the absence of population data and rigorous diagnostic assessment makes such associations difficult to interpret. For example, early reports suggested an association with congenital rubella, but this now seems questionable given the diagnostic dilemmas and the observation that 'autistic-like' features diminish over time.[32] Gillberg[33] argues that medical conditions may be associated with autism in as many as one-third of the cases, but Rutter and colleagues[34] suggest that a more reasonable figure would be roughly 10 per cent of all cases. The strongest associations are with fragile X syndrome and tuberous sclerosis—both conditions having a strong genetic component (Box 9.2.3.3).

Box 9.2.3.3 Medical conditions associated with autism

- Seizure disorder (epilepsy)
- Fragile X syndrome
- Tuberous sclerosis

Fragile X syndrome is an X-linked intellectual disability syndrome involving a mutation characterized by a triplet repeat of cytosine–guanine–guanine (CGG) that may amplify with succeeding generations. It is associated with a characteristic facial appearance, enlarged testicles, intellectual disability, and some autistic features. Early reports suggesting high rates of fragile X in autism have now been modified; fragile X affects perhaps 1 to 2 per cent of all individuals with autism.[20]

The autosomal dominant disorder tuberous sclerosis is characterized by abnormal tissue growth, or benign tumours (hamartomas), in the brain and in other organs. The condition, which may affect 1 in 10 000 individuals, is variably expressed; the phenotype ranges from minor skin problems or seizures to severe intellectual disability with intractable seizures. The rate of this condition in autism (0.4–2.8 per cent) is significantly increased.[20]

Autism is a strongly genetic condition. Studies of monozygotic and dizygotic twins revealed much higher levels of concordance for monozygotic relative to dizygotic twin pairs, but also elevated

rates of concordance in dizygotic twins relative to population rates.[35] General studies suggest that the recurrence risk of autism in siblings is in the order of between 2 and 10 per cent—which is a substantial increase in risk over population rates. There also appear to be higher rates of social and language problems and rigid patterns of behaviour in siblings and close relatives, raising the possibility that what is inherited is a broader phenotype reminiscent of autism but which also may reflect a more general predisposition to developmental difficulties. Recent work also suggests elevated rates of anxiety and mood disorders in family members. Specific modes of inheritance remain unclear. It now appears that several interacting genes are probably involved in the pathogenesis of autism. Efforts are underway to identify susceptibility genes and trace their impact on brain development. Although several studies have shown increased rates of pre-, peri-, and neonatal complications in children with autism, it is possible that some of these difficulties may reflect a genetic vulnerability in the child or that there may be an interaction of genetic and perinatal factors.[35] A recent report has noted the presence of placental abnormalities in pregnancies of children with autism.

Attempts have been made to identify neuropathological and neuroanatomical correlates of autism. Areas of interest have included the cortical areas responsible for language and social interaction (frontal and temporal lobes) as well as the neostriatum and cerebellum.[36] The report of reduced cerebellar size in the neocerebellar vermal lobules VI and VII has not proven readily replicable. Some individuals with autism have enlarged brains and head sizes, with some evidence that abnormal growth occurs in the first 2 years of life.[37] Neuropathological studies have suggested possible cellular changes in areas of the brain such as the hippocampus and amygdala and changes in the cytoarchitecture of the brain, e.g. in the arrangement of cortical 'minicolumns'.

Various neurotransmitter systems have been studied. Probably the most robust finding has been the observation that about one-third of the children with autism have increased peripheral levels of serotonin. This finding is not specific to autism and its significance remains unclear.[38] Studies of other neurotransmitters such as dopamine produced inconsistent findings. The possible involvement of dopamine is suggested by the high levels of stereotyped behaviours in autism—behaviours, which can be induced in animals by the administration of agents (stimulants) that affect levels of dopamine in the brain. Agents such as the neuroleptics, which block dopamine receptors, are effective in reducing the stereotyped and hyperactive behaviours of many autistic children. Another hypothesis has centred on the possible role of endogenous opioids, in that overproduction of such compounds might lead to social withdrawal and unusual sensitivities and behaviours. This has led to the administration of opioid antagonists such as naltrexone in autism; unfortunately results have been disappointing. Studies of the immune system in autism have been relatively uncommon and findings inconsistent.

Rett's syndrome

In 1966, Andreas Rett described an unusual syndrome in girls characterized by a history of initial normal development, subsequent head growth deceleration, and the development of specific clinical findings such as breathing difficulties, movement problems, and some features suggestive of autism.[39] His findings were replicated and extended by Hagberg and colleagues.[40] As more extensive information became available it became clear that the more 'autistic-like' phase of Rett's syndrome was relatively brief, but this was a major rationale for its inclusion in the PDD class.[41]

Clinical features

Early pre- and perinatal histories are generally unremarkable in Rett's syndrome, as is very early development. Usually within the first or second year of life development begins to slow or actually regress and various motor problems—including characteristic hand-washing stereotypies start to develop.[42] A significant loss of developmental skills occurs and head growth decelerates.[40] The potential for misdiagnosis of autism is greatest during this time; during school age developmental regression often stabilizes and children are more socially responsive. As individuals with Rett's syndrome approach adolescence they are frequently subject to increased spasticity, scoliosis, loss of ambulation, bruxism, hyperventilation, areophagia, apnoea, and seizures.[43] Although debated,[44] the inclusion of Rett's syndrome in the PDD class reflects an awareness of the confusion with autism and the importance of including the condition somewhere.[41]

Definition

Various diagnostic criteria for Rett's syndrome have been developed.[45] As presently defined in ICD-10 (see Table 9.2.3.2) the condition develops after some months of normal development; head circumference is normal at birth but begins to decelerate between 5 and 48 months. Characteristic midline hand-wringing or hand-washing stereotypies develop and purposeful hand movements are lost.

Epidemiology and demographics

Prevalence estimates range from 1 per 12 000 to 1 per 23 000 females; cases are observed in all racial and ethnic groups.[46,47] Rett's syndrome may account for one-quarter to one-third of progressive developmental disabilities among females.[46] A small number of cases have now been reported in males.

Course and prognosis

Various clinical stages of the condition have been identified.[42] Early developmental losses may be subtle initially but become more marked with time. Purposeful hand movements are often then lost, as are speech skills. Ataxia and gait abnormalities are noted in those

Table 9.2.3.2 ICD-10 criteria for Rett's syndrome (F84.2)

A. There is an apparently normal prenatal and perinatal period and apparently normal psychomotor development through the first 5 months and normal head circumference at birth.
B. There is a deceleration of head growth between 5 months and 4 years and loss of acquired purposeful hand skills between 5 and 30 months of age that is associated with concurrent communication dysfunction and impaired social interactions and the appearance of poorly co-ordinated/unstable gait and/or trunk movements.
C. There is severe impairment of expressive and receptive language, together with severe psychomotor retardation.
D. There are stereotyped midline hand movements (such as hand-wringing or 'hand-washing') with an onset at or after the time when purposeful hand movements are lost.

Taken from Disorders of psychological development (***criteria for research***), pp. 154–5.

patients who had acquired the ability to walk, unusual breathing patterns (hyperventilation and/or apnoea), and seizures may also be observed. While social and communication skills may improve in middle childhood motor problems are more pronounced. During the final phase (roughly after age 10) progressive scoliosis and spasticity are observed, while cognitive function stabilizes and seizure activity may diminish. There is a dearth of information on adults with the condition. There does appear to be an increased risk of sudden death due to seizures and/or respiratory difficulties.[48]

Aetiology

Rett[39] originally speculated that the condition might be associated with high peripheral ammonia levels, but this was incorrect. Recent work identified mutations in the MeCP2 gene as the cause of Rett syndrome in most cases; this gene has a major role in regulating various genes during brain development.[49]

Childhood disintegrative disorder

Shortly after the turn of the twentieth century a special educator working in Vienna, Theodor Heller,[50] reported children who had a period of several years of normal development prior to a marked regression with loss of skills in multiple areas and minimal recovery. He initially termed the condition dementia infantilis; subsequently it has been referred to as Heller's syndrome, disintegrative psychosis, or childhood disintegrative disorder.[51] Once it develops the condition is indistinguishable from autism,[52] but it is accorded separate diagnostic status since it appears distinctive in terms of onset and course.

Clinical features

In this condition an 'autistic-like' clinical picture develops after a prolonged period of normal development.[50] More than 100 cases have now been reported; the condition is rare but probably underdiagnosed.[51] Essential clinical features include a relatively long period of normal development followed by a marked developmental regression and development of various 'autistic-like' features; onset is typically between the ages of 3 and 4 years.

Onset can be relatively abrupt or more gradual, and a premonitory phase of non-specific anxiety or agitation may be observed. Parents often note associations between the onset of the condition and various psychosocial or medical events but such associations are probably correlational rather than causative.[53] Once established it resembles autism.[52] Given the behavioural similarity to autism, it might be argued either that the condition does not warrant separate diagnostic recognition or that the condition is inevitably the result of an association with some progressive medical condition. However, the pattern of onset is quite distinctive, the outcome appears to be even worse than in autism, and usually even with very intensive medical evaluations no general medical condition accounting for the regression is identified.[52]

Definition

Criteria for the disorder are listed in Table 9.2.3.3. By definition the disorder cannot coexist with autism or another other explicitly defined pervasive developmental disorder, schizophrenia, elective mutism, or the syndrome of acquired aphasia with epilepsy.

Epidemiology and demographics

Epidemiological data are quite limited. Some limited data suggest a prevalence rate of 1 in 100 000. Initially cases seemed to be equally distributed between males and females, but it now seems likely that cases of Rett's syndrome may have been included in early case series. Recent reviews have noted a preponderance of males.[20]

Table 9.2.3.3 ICD-10 criteria for other childhood disintegrative disorder (F84.3)

A. Development is apparently normal up to the age of at least 2 years. The presence of normal age-appropriate skills in communication, social relationships, play, and adaptive behaviour at age 2 years or later is required for diagnosis.
B. There is a definite loss of previously acquired skills at about the time of onset of the disorder. The diagnosis requires a clinically significant loss of skills (not just a failure to use them in certain situations) in at least two of the following areas: 1 expressive or receptive language 2 play 3 social skills or adaptive behaviour 4 bowel or bladder control 5 motor skills.
C. Qualitatively abnormal social functioning is manifested in at least two of the following areas: 1 abnormalities in reciprocal social interaction (of the type defined for autism) 2 qualitative abnormalities in communication (of the type defined for autism) 3 restricted, repetitive, and stereotyped patterns of behaviour, interests, and activities, including motor stereotypes and mannerisms 4 a general loss of interest in objects and in the environment.
D. The disorder is not attributable to the other varieties of pervasive developmental disorder; acquired aphasia with epilepsy (F80.6); elective mutism (F94.0); Rett's syndrome (F84.2); or schizophrenia (F20.-).

Taken from Disorders of psychological development (criteria for research), pp. 154–5

Course and prognosis

In about 75 per cent of cases the deterioration reaches a plateau, in that there is no further loss of skills, but subsequent gains tend to be minimal. Thus the child who previously was normally socially related, spoke in full sentences, and was toilet-trained becomes indifferent to social interaction, loses all expressive language and toilet skills, and remains mute and relatively low functioning.[52] In the remainder of cases there is more limited recovery, for example the child regains the capacity to speak but only in single words or only echoes language. If a progressive metabolic or neuropathological process is present, the developmental progression may continue until death ensues; such cases often have a later onset.[54] Life expectancy otherwise appears to be normal. In a very small number of cases significant recovery has been noted.

Aetiology

Various lines of evidence suggest the importance of neurobiological factors in pathogenesis. Occasionally medical conditions such as the neurolipidoses, metachromatic leukodystrophy, Addison–Schilder's disease, and subacute sclerosing panencephalitis are associated with the condition. Rates of electroencephalographic abnormalities and seizure disorders are of roughly the same frequency as seen in autism.

Asperger's syndrome

The condition known as Asperger's syndrome was described by a paediatrician with interest in intellectual disability, Hans Asperger,[55] who reported on four boys with marked social problems, unusual perseverative interests, and motor clumsiness but with seemingly good verbal and cognitive abilities. Like Kanner, Asperger used the word autism (autistic psychopathy) to describe this condition. His concept, however, had points of difference, as well as similarity, to autism. For example, verbal abilities tended to be an area of strength, concerns typically did not arise until later in the preschool period, and there was a tendency for the condition to run in families—particularly in fathers. Lorna Wing's[56] report of Asperger's work and publication of a series of cases brought wider attention to the diagnostic concept. The validity of this condition, particularly apart from higher-functioning autism, remains the topic of much debate. A major complication has been the marked differences in definition of the conditions and its potential overlap with other diagnostic concepts (e.g. schizoid personality,[57] nonverbal learning disabilities,[58] semantic–pragmatic disorder,[59] and right hemisphere learning problems).[60] As a result, the literature on this condition is difficult to interpret, although areas of potential differences from autism have been identified, such as neuropsychological profiles[61] and family history.[62]

Clinical features

This condition is characterized by impairments in social interaction and restricted interests and behaviours as seen in autism. However, the child's early development is marked by lack of any clinically significant delay in spoken or receptive language, cognitive development, self-help skills, and curiosity about the environment. Consistent with Asperger's[55] original report, all-absorbing and intense circumscribed interests as well as motor clumsiness are typical, but are not required for diagnosis. The validity of this condition, apart from high-functioning autism and PDD not otherwise specified (PDD-NOS) is controversial. Available research is difficult to interpret given the markedly different ways in which the diagnostic concept has been used. Differences are more likely to be noted relative to autism if a rather stringent diagnostic approach is used. Evidence for external validity of the condition relative to autism includes differences in neuropsychological profiles, patterns of comorbidity, and family history.

Persons with Asperger's syndrome often exhibit a somewhat eccentric social style rather than the more passive or aloof style noted in autism; for example, they may engage others in very one-sided conversations about their area of special interest. They maybe overly reliant on rigid rules for social interaction and may fail to 'see the forest for the trees' in social matters (e.g. an appreciation of exactly when the usual rules do not apply is as important as when they do). Their social oddity and lack of flexibility is a source of much disability.

While early speech-communication skills are apparently normal, certain aspects of communication become more deviant over time. Prosody may be poor, rate of speech may also be unusual, or it may have a somewhat disorganized, tangential, and circumstantial quality. The issue of whether such persons are at increased risk for thought disorder and psychosis remains unresolved, but some part of this impression probably reflects communication problems.

It is rather typical for patients to amass considerable factual information about their topic of interest, which they pursue with great intensity; Asperger originally observed that family life may revolve around the topic of special interest. He also suggested that motor clumsiness was present and, although not required for the diagnosis, there is often a history of motor delay and persistent motor awkwardness—for instance, the child may talk before he walks, have trouble fastening fastener, catching a ball, learning to ride a bicycle, engaging in cursive handwriting, and may also display stiffened gate.

Differences in neuropsychological profiles have been reported.[63] A stringent diagnostic approach may suggest areas of relative strengths (auditory and verbal skills and rote learning) and weakness (visuomotor and visuoperceptual skills); this pattern differs from that observed in higher-functioning individuals with autism[64] and the heritability of social difficulties may be even greater in Asperger's syndrome than in autism.[62]

Interest in the condition has revolved around the possibility that it might represent a transition between autism and other disorders such as schizophrenia. Associated conditions have included depression, anxiety and other mood problems, violence, and other psychotic conditions.[63] Unfortunately, almost all of this literature rests on case reports; controlled studies are needed.

Definition

The ICD-10 criteria for Asperger's syndrome are given in Table 9.2.3.4. As presently defined the social deficit is the same as in autism. In contrast to autism, however, early language, cognitive, and other skills develop typically early in life. By definition, the case does not meet the criteria for childhood autism. Miller and Ozonoff[65] note that several aspects of the current definition can be problematic; Asperger consistently felt that the syndrome he described differed from Kanner's autism (1979). Thus the current definition almost certainly will be refined (or discarded) in future editions of ICD and DSM.

Table 9.2.3.4 ICD-10 criteria for Asperger's syndrome (F84.4)

A. There is no clinically significant general delay in spoken or receptive language or cognitive development. Diagnosis requires that single words should have developed by 2 years of age or earlier and that communicative phrases be used by 3 years of age or earlier. Self-help skills, adaptive behaviour, and curiosity about the environment during the first 3 years should be at a level consistent with normal intellectual development. However, motor milestones may be somewhat delayed and motor clumsiness is usual (although not a necessary diagnostic feature). Isolated special skills, often related to abnormal preoccupations, are common, but are not required for the diagnosis.
B. There are qualitative abnormalities in reciprocal social interaction (criteria as for autism).
C. The individual exhibits an unusual intense circumscribed interest or restricted, repetitive, and stereotyped patterns of behaviour interests and activities (criteria as for autism; however, it would be less usual for these to include either motor mannerisms or preoccupations with part-objects or non-functional elements of play materials).
D. The disorder is not attributable to other varieties of pervasive developmental disorder; simple schizophrenia; schizotypal disorder; obsessive-compulsive disorder; anankastic personality disorder; reactive and disinhibited attachment disorders of childhood.

Taken from Disorders of psychological development (criteria for research), pp. 154–5

Epidemiology and demographics

Estimates of prevalence vary markedly depending on the stringency of the definition used. A stringent approach to diagnosis would suggest a rate in the order of 1 in 2000 or so, but a much less stringent approach may yield one in several hundred.[66]

Asperger[55] originally reported the condition only in boys, although Wing reported on girls with the condition. There does appear to be a male predominance in the order of 20 to 1; similar sex ratios are observed in autism not associated with intellectual disability.

Course and prognosis

Asperger's original[55] impression was of favourable long-term prognosis.[67] Many individuals can attend regular school with some additional support; unfortunately such children may be seen as eccentric and are often prime targets for being victimized. Better verbal skills can mislead educators about the child's vulnerability in other areas, and difficulties academically may be misattributed to wilful non-compliance. There is the impression that these individuals are capable of greater degrees of personal and occupational self-sufficiency than those with autism, but definitive data are lacking. It does appear that the social difficulties persist into adulthood.[68]

Aetiology

Although the cause of Asperger's syndrome remains unknown, the report of high rates of the condition in family members and the reports of occasional familial associations with autism suggests the potential importance of genetic factors. Neurobiological information on the condition is limited. The potential association of the condition with specific neuropsychological profiles is of some interest.

Atypical autism/PDD not otherwise specified

Recent editions of both ICD and DSM have included a 'subthreshold' category (termed either atypical autism or pervasive developmental disorder not otherwise specified—PDD-NOS). In some ways, this notion has historical links to earlier diagnostic concepts.[23] In practice, the term atypical autism in ICD-10 and the term PDD-NOS in DSM-IV refer to what is a residual diagnostic category. ICD-10 provides the possibility for various forms of special coding—for example, failure to meet the onset criteria for autism, failure to meet developmental/behavioural criteria, or failure to meet both.

Research on this diagnostic category has been less advanced than that for other disorders—no doubt reflecting the problems intrinsic to 'subthreshold' disorders. Several attempts have recently been made to identify potential subgroups within the rather heterogeneous disorder, but none has yet achieved general acceptance. Various attempts have been made to identify specific subgroups within this broader category.[69] It is sometimes the case that social and/or communicative skills are relatively preserved but the child exhibits unusual sensitivities, affective responses, and thought processes. It is likely that the term presently encompasses a number of conditions, which may be identified in the future.

Differential diagnosis

Autism and related PDDs must be differentiated from each other, from the specific developmental disorders (e.g. of language), from intellectual disability not associated with PDD, and from other conditions. In intellectual disability not associated with PDD, social and communicative skills are typically on a par with the child's overall intellectual ability. Diagnostic differentiation can be most challenging in persons with severe and profound intellectual disability where assessment is more difficult and stereotyped movements common. Occasionally language and other specific developmental disorders may be confused with autism/PDD. Usually, however, the child's social abilities are preserved and the child is very communicative non-verbally. Schizophrenia rarely has its onset in childhood and almost never before the age of 5 years.

On occasion, selective mutism or social anxiety disorder may be confused with a PDD (particularly PDD-NOS/atypical autism). However, in selective mutism the child can speak in some situations. Similarly, children with anxiety in social situations will usually not exhibit the other symptoms characteristic of autism/PDD. The unusual behaviour and interests of children with obsessive–compulsive disorder may be taken to suggest autism or PDD, but social and language-communication skills are preserved.

In considering the differential diagnosis of conditions that present with regression it is important to review carefully previous diagnostic evaluations. Occasionally, progressive neuropathological conditions may have their onset in childhood. In the Landau–Kleffner syndrome (acquired aphasia with epilepsy) social skills should be relatively preserved even in the face of an extensive aphasia.

Sometimes children who have experienced marked neglect may present with social difficulties, initially suggesting autism or PDD. However, in reactive attachment disorder the history of severe neglect is observed and, as the name of the condition implies, social deficits should remit substantially if an appropriate and nurturing environment is provided.

Treatment

Over the past decade a relatively substantial body of research on treatment of autism has appeared. Recent summaries of this work are available.[70] Much of this work relates to behavioural and educational interventions although a body of well-controlled studies of psychopharmacological agents has appeared as well.[71]

Children with autism and other PDDs generally require an intensive and highly structured intervention programme. More able children may be able to tolerate regular classroom situations, with appropriate support, but more impaired children often need higher levels of teacher supervision and a more intensive classroom setting.[72] For lower-functioning children areas of priority include the ability to tolerate adult guidance and intrusion, to follow routines, to develop communicative abilities, and move from associative to more conceptual learning strategies.[73] The classroom setting can be important, as children with PDD can be readily distracted by extraneous stimuli. The tendency of such children to rely on routines can be used effectively to help promote more systematic learning. Generalization of skills learned is particularly important since the child may have difficulties in applying skills learned in new settings. Speech and communication are a critically important aspect of any intervention programme.[11] Techniques to foster communication through non-verbal means such as sign language, picture-exchange, visual schedules, and other augmentative methods can be very helpful to non-verbal children.[74] The use of such methods does not preclude, and in fact may foster, the use of spoken communication.

Behaviour modification techniques are helpful in increasing the frequency of desired behaviours while simultaneously diminishing problem behaviours. Typically, a functional analysis of the target behaviour is initially performed, and then a plan developed for prompting or decreasing the behaviour.[73] While there is general agreement that children with autism/PDD profit from a behaviourally based intervention, there is more controversy over the degree to which progress can be made; for instance, there have been some claims for dramatic improvement and even 'cures' of autism.

Neuroleptics have been the most intensively studied psychopharmacological agents in this population, and there have been several well-designed double blind, placebo-controlled trials.[71] The main mechanism of action appears to be dopamine-receptor blockade. The agents may reduce maladaptive behaviours such as stereotypies,[75] but side effects include sedation, irritability, and movement problems (including tardive dyskinesia). Recently, interest has centred on the newer atypical neuroleptics.[76]

Management

Goals for treatment include promoting learning and reducing behaviours that interfere with learning. Treatment is best based on a comprehensive view of the child and his or her strengths and areas of need. A structured and individualized intervention programme is needed. Various professionals such as speech pathologists, special educators, occupational and physical therapists may be involved.[72,73] Goals for intervention will vary depending on developmental level, life circumstance, and clinical context—for example, vocational factors will be more important during adolescence, and for individuals with conditions like Rett's syndrome the efforts of other professionals (orthopaedists and respiratory therapists) may be needed.

For higher functioning and older individuals the acquisition and generalization of social skills are particularly important. The use of rehearsal and social scripts may be indicated. Teaching must be explicit and can include modelling and rehearsal within individual instruction and small group settings, with the use of naturalistic settings to encourage generalization whenever possible. For higher functioning students, including those with Asperger's syndrome, this can include explicit analyses of challenging social situations, videotaping for self-observation, role playing, and the use of individualized social stories.[77]

There is no evidence that unstructured psychotherapy is useful in autism and related conditions. Structured and supportive psychotherapy may be appropriate for some carefully selected, higher-functioning individuals, particularly if it focuses on explicit problem-solving strategies for frequently troublesome situations.

Pharmacotherapy interventions are not curative, but they may provide considerable help with specific problematic symptoms.[71] The best evidence relates to the atypical neuroleptics but data on other agents are less substantive. The balance of potential benefits and risks should be considered, and informed consent obtained from parents or, whenever possible, the affected child.

Mood stabilizers and antidepressants have sometimes been used, given the increase in affective lability, anxiety, and depression in individuals with autism and PDDs. However, the response to antidepressants has been somewhat variable. Lithium and other mood stabilizers are sometimes used clinically, particularly if there is a strong family history of bipolar disorder, but there have been few controlled studies of these agents.

Selective serotonin-reuptake inhibitors (SSRIs) were of initial interest in autism, given the repeated reports of high group levels of peripheral serotonin in this population as well as of the high levels of repetitive behaviours observed in this group (i.e. reminiscent of those seen in obsessive–compulsive disorder). Several reports have suggested that, at least in adults, these agents may be helpful in lowering the levels of obsessive–compulsive-like behaviours, although activation is sometimes observed; Studies of children have been less frequent, but there is some suggestion that they may also respond.[71]

Various other agents have been used in autism, including anxiolytics, β-blockers, clonidine, and naltrexone. Unfortunately, it is difficult to draw firm conclusions from the limited data available, but at present the clinical efficacy of these agents is not well established in this population. Surprisingly few studies have systematically evaluated the use of stimulants in autism. However, the observation that these agents induce stereotyped behaviours in animals would suggest their potential for increasing levels of stereotyped behaviours.

Prevention

At present information on the prevention of autism and related PDDs is clearly quite limited. Apart from the association with two strongly genetic conditions (fragile X syndrome and tuberous sclerosis) no specific biological markers for autism have yet been found although some promising leads for early screening or children at risk (i.e. siblings) have appeared. It is likely that early diagnosis may change dramatically in the not so distant future as potential susceptibility genes are identified or are combined with innovative approaches to screening—this will have potentially major importance since there is some suggestion that early intervention may significantly improve outcome.

Further information

www.autism.fm. Regularly updated website with links to other sites.

Quackwatch www.quackwath.com. Information about non-conventional treatments.

Jacobson, J.W., Foxx, R.M., and Mulick, J.A. (2005). *Controversial therapies for developmental disabilities: fad, fashion and science in professional practice*. Lawrence Erlbaum Associates, Mahwah, NJ. A comprehensive guide to nonconventional treatments.

Center for disease control—autism related information:

http://www.cdc.gov/ncbddd/dd/ddautism.htm This website provides basic information for physicians (including early warning signs of autism) in both English and Spanish.

Federal autism research networks website: www.autismresearchnet.work.org provides links to US federally funded autism research projects.

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9.2.4 Attention deficit and hyperkinetic disorders in childhood and adolescence

Eric Taylor

Introduction

The concept of ADHD arose from neurological formulations, but does not entail them, and the modern definition simply describes a set of behavioural traits. The historical evolution of the concept was described by Schachar.[1] It began with the idea that some behavioural problems in children arose, not from social and familial adversity, but from subtle changes in brain development. The term 'minimal brain dysfunction (MBD)' was often applied, and covered not only disorganized and disruptive behaviour but other developmental problems (such as dyspraxias and language delays) presumed to have an unknown physical cause. MBD, however, stopped being a useful description when studies of children with definite and more-than-minimal brain damage made it plain that they showed a very wide range of psychological impairment, not a characteristic pattern (see Harris, this volume); and therefore it was invalid to infer the presence of brain disorder from the nature of the psychological presentation.

The successor to the concept of MBD was attention deficit and hyperactivity: defined, observable behaviour traits without assumption of cause. 'Attention Deficit/Hyperactivity Disorder' (ADHD) in DSM-IV, and 'Hyperkinetic Disorder' in ICD-10, describe a constellation of *overactivity, impulsivity* and *inattentiveness*. These core problems often coexist with other difficulties of learning, behaviour or mental life, and the coexistent problems may dominate the presentation. This coexistence, to the psychopathologist, emphasizes the multifaceted nature of the disorder; to the sociologist, a doubt about whether it should be seen as a disorder at all; to the developmentalist, the shifting and context-dependent nature of childhood traits. For clinicians, ADHD symptoms usually need to be disentangled from a complex web of problems. It is worthwhile to do so because of the strong developmental impact of ADHD and the existence of effective treatments. Public controversy continues, but professional practice in most countries makes ADHD one of the most commonly diagnosed problems of child mental health.

Clinical features

Overactivity

The idea of overactivity refers simply to an excess of movement. It is not totally dependent on context and cannot be reduced to non-compliance: physical measures of activity level have indicated that it is higher in children with ADHD than in controls, even during sleep.[2] It is, however, partly dependent upon context: it is often inhibited by a novel environment, creating a pitfall for the inexperienced diagnostician who may exclude it incorrectly because it is not manifested during observation at a first clinic visit. It may not be evident in situations where high activity is expected, such as the games field. The key situations where it is evident are familiar to the child and where calm is expected, such as visiting family friends, attending church, mealtimes, homework and—often the most troublesome—at school, during class.

(a) Impulsivenes

Impulsiveness means action without reflection—often described as a failure to 'stop and think'. The term covers premature, unprepared and poorly timed behaviours—such as interrupting others, and giving too little time to appreciate what is involved in a school task or a social situation.

(b) Inattentiveness

Inattentiveness means disorganized and forgetful behaviour: short-sequence activities, changing before they are completed, with a lack of attention to detail and a failure to correct mistakes. All these are behavioural observations, not psychological constructs. At a cognitive level, 'Attention deficit' is a rather poor descriptor; the performance of affected children does not fade with time on a task any more than that of ordinary people and the presence of irrelevant information ('distractors') does not worsen their performance disproportionately to that of other people.[3] There are cognitive changes of a different kind (see 'Aetiology' below); but the diagnosis of inattentiveness depends on descriptions and observations of behaviour rather than on tests of performance.

Many other behavioural changes characterize some children with ADHD. They are, for instance, often irritable and their emotions can flash very rapidly when provoked. They may sleep badly (and this in turn can contribute to poor concentration). They can be aggressive to other people and non-compliant to authority. They can also be charming, humourous, inquisitive and intuitive. None of these, however, are either constant in ADHD or confined to those with ADHD. They are worth noting, but they do not make the diagnosis.

Classification

Attention Deficit/Hyperactivity Disorder in DSM-IV is defined as the presence of a number, above a cut-off, of behaviours considered to reflect the cardinal features described above to a degree that is developmentally inappropriate and gives rise to some impairment in more than one setting (e.g. school and home).[4] Overactive and impulsive behaviours are considered together as a single construct of 'hyperactivity-impulsiveness', and for convenience the combined dimension will be referred to in this chapter as 'hyperactivity'. Examples of inattentive behaviour are added together and form a separate dimension. There are therefore three subtypes: hyperactive-impulsive, inattentive and combined.

The same problems characterize the ICD-10 definition of 'Hyperkinetic Disorder' (HD),[5] but with added requirements: especially, that all three cardinal features are present, pervasively across home, school and other situations. HD is therefore, in effect, a subtype of ADHD.[6,7] The subtypes of hyperkinetic disorder are based on the presence or absence of conduct disorder—and indeed the presence of conduct disorder is an important factor with which to reckon in the course.

Diagnosis

Description of the symptoms makes them sound easy to recognize, and indeed the problems are usually very salient, disruptive to

other people, and common causes of referral to health and special education services. Nevertheless, there are pitfalls in the diagnosis, making it necessary for a specialist assessment to be undertaken before the diagnosis is given.

Ambiguous criteria

The behavioural problems, described in outline above, are translated into detailed criteria in DSM-IV and ICD-10, and some of them can be ambiguous. For example, 'does not follow instructions' is a DSM item intended to imply that instructions are forgotten or not attended to; but the behaviour can also be shown for reasons of wilfulness and therefore a part of oppositional/defiant disorder. Careful description or witnessing the behaviours complained of is necessary.

Confusion of cardinal and associated features

Many behavioural problems—such as temper tantrums, sleeplessness, aggression and disobedience to adult authority—are common in children with ADHD, and may be the key reasons for presentation. It is easy to make the mistake of diagnosing ADHD when only disruptive behaviour is present. Much of the confusion comes from the way impulsiveness is operationalized in the diagnostic schemes. Behaviours, such as calling out in class and interrupting others, can indeed come from a difficulty in holding oneself back; but they can also represent deliberate flouting of the rules, and in a London survey of 6- to 8-year-old boys they were as common in non-hyperactive but defiant children as in the hyperactive.[8] Direct observation can usually make the distinction—watching either the children tackling tasks requiring them to stop and think in the clinic, or their natural behaviour in the classroom. Inattentive behaviour also helps to make the diagnosis of ADHD and is less confounded by oppositionality.

(a) Reliance on non-expert judgements

The behaviours of ADHD are continuously distributed in the population (see 'Epidemiology'). The level that is considered normal or acceptable will vary from one culture to another and from one rater to another. To be diagnosed, they should be excessive not only for the child's age but also for the developmental level; and this demands considerable familiarity with the usual range of variation. The diagnostician will acquire this in the course of training and experience; experienced teachers will be excellent judges; but inexperienced or overstressed parents may identify the problems at a low level of hyperactive behaviour, or suppose that an abnormal level is only to be expected in childhood. It is usually helpful to obtain a detailed behavioural account rather than rely on an overall judgment of 'overactivity' or 'failure to concentrate'. Contradiction between sources may occur, and leads to arguments between parents and teachers. This may be due to different expectations, the emotional relationship of raters with the child, or children behaving very differently in contexts that vary in the demands placed on the children. The clinician needs to understand the full context of the way involved adults describe the child.

(b) Problems of recognition in the presence of coexistent problems

It is commonplace for children whose problems meet the criteria for ADHD to show other patterns of disturbance as well. This is often, confusingly, called 'comorbidity' – confusing because it assumes that the other pattern is a distinct disorder, which is only one of the explanations for coexistent problems. Clinicians need to understand the relationships for two reasons; so that they do not make or miss the diagnosis of ADHD; and so that they can make good strategies for treating ADHD in the presence of other disorders and other disorders in the presence of ADHD (see 'Treating Complex cases', below).

(c) Conduct and oppositional disorders

The commonest association and the best researched is with conduct and oppositional disorders. Nearly half the children with hyperactive behaviour in a community survey showed high levels of defiant and aggressive conduct as well; but the associations of the two problems were different, with hyperactivity (but not conduct problems) being associated with delays in motor and language development.[8] Genetic research indicates higher heritability for hyperactivity than for defiant and aggressive behaviour; but there are some genetic influences that are common to both.[9] Hyperactivity is more responsive to stimulant medication than are less hyperactive forms of conduct problem.[10]

When both ADHD and conduct problems are present, then the combined diagnosis ('hyperkinetic conduct disorder' in ICD-10) shows the associations of both disorders. ADHD is therefore not to be diagnosed by the absence of conduct disorder features, but by the clear presence of the core problems of inattentivess and disorganization.

(d) Tourette disorder and multiple tics

A different kind of differential is presented by children with Tourette disorder. Their motor restlessness may indeed represent the coexistence of ADHD, but can result directly from tics. If a child's tics are very frequent and there are a large number of them, then their repetitive and stereotyped nature may not be apparent and they may be seen simply as restless fidgetiness. Again, direct observation of the pattern of overactivity is the key. When there is doubt, videorecording the child and subsequent slow-motion review may make repetitive patterns evident.

(e) Autism spectrum disorders

Children with autism have clear and characteristic impairments of language, communication, and social development. Spectrum disorders, however, can raise diagnostic challenges. Children with ADHD alone often show language delays (usually of an expressive nature with over-simple utterances, by contrast with the receptive difficulties and idiosyncratic patterns of autism). Their attention difficulties may make them unresponsive to the overtures of others in a way that can simulate the social obliviousness of people in the spectrum of autism, and they are often friendless—not because of lack of interest in others but because of the capacity of hyperactive behaviour to irritate other people. Indeed, attention problems can extend to perseverativeness on certain activities such as video games that may be mistaken for the restricted interests of autism. All these factors can lead to ADHD being mistaken for autism, but the reverse can happen too. There are other reasons for overactive behaviour in autism. First, stereotyped patterns of driven overactivity can be seen: they are not disorganized or impulsive and are often made worse by change and novelty (which usually reduce the overactivity of ADHD). Second, episodic bursts of extreme activity can be seen and may be best regarded and treated as catatonic. Third, akithisia may result from neuroleptic medication, or irritable

restlessness from anticonvulsants, and it will be necessary to establish a clear history that ADHD has been a persistent trait.

(f) Attachment disorders

Reactive attachment disorder (RAD) may share with ADHD a disinhibited style of relating to other people (an unreserved but shallow making of social contact). Children with RAD, however, tend to be controlling rather than disorganized, and vigilant rather than inattentive; and inattention and impulsiveness are not cardinal features of RAD; so it is not difficult to recognize both patterns when present in an individual child. The confusion in practice often comes from theoretical misconceptions. Those caring for neglected or abandoned children may consider that the diagnosis of ADHD cannot be accurate because the cause of the children's problems is clearly to be found in their early deprivation. The causal pathway may indeed be that of neglect (though genetic inheritance and fetal exposure to toxins also need considering); but ADHD is a descriptive category, not an explanatory one. If the pattern of ADHD is present it still needs recognizing—not least because the cause of the ADHD behaviour does not seem to determine the response to stimulant medication, and children who have encountered neglect or abnormal early attachment may still have their ADHD problems reduced by medication.[10]

(g) Bipolar disorders

Both ADHD and manic conditions are characterized by overactivity, overtalkativeness, a sensation of whirling thoughts, and often by irritable mood. The distinction is made by the presence in bipolar disorder of episodicity, euphoria, and grandiosity. A suggestion that these distinguishing features are not in fact present in childhood bipolar disorder has naturally led to great overlap between the expanded childhood bipolar diagnosis and ADHD with poor emotional regulation, and further research will be needed to clarify whether there is a distinction.

In all these differential diagnoses, the principle is to establish that the child shows not only overactive behaviour, but the specific pattern of ADHD. Experienced judgement may be required, and the practice of diagnosing on the basis of questionnaire scores alone risks overidentification.

In adult life, there are still more possibilities for misdiagnosis. The commonest reasons for uncertainty are in distinguishing from atypical bipolar disorder and the effects of substance misuse. 'Personality disorder' is sometimes applied; and indeed ADHD shares with personality disorders a long-standing trait quality, but can also be a more precise way of describing the difficulties presented. Differentiation from the normal range of variation can be difficult in the absence of clear standards. The task of the diagnostician is harder when adults are presenting for the first time if only self-report is available; the self-description of hyperactivity may be a form of self-depreciation.

Methods of recognition

(a) Rating scales and informant interviews

Questionnaire ratings by parents or teachers are very useful for screening purposes, and in group studies they give a fairly good discrimination between people with a clinical diagnosis of ADHD and controls from the ordinary population.[11] Many are available[12,13] and the most famous are those from Conners, which yield several different scoring systems; and derivatives such as the Iowa Conners, the SWAN and SNAP scales.[14] They do however leave a fair number of individuals misclassified, and are not suitable as the sole means of establishing a diagnosis. A detailed interview with parents establishes what actual behaviours are the basis for ratings, allows professional judgement to be included, and remains the most informative single method.

(b) Psychiatric interview

Interview with the child is valuable for the observation of attention and social interaction that it yields, and for understanding a child's view of their predicament. Children, however, are not good witnesses about their own concentration and impulse control, and even affected adults are not good at describing themselves in these terms. The experience of ADHD is usually one of suffering the reactions evoked from other people, or an experience of repeated failure. Adults often describe an experience of whirling and interrupted thoughts (in the absence of manic features); and some children will say the same, especially if treatment has enabled them to make a comparison with another way of being.

(c) Investigating underlying causes

Assessment needs not only to distinguish ADHD from related disorders, but to consider whether the ADHD pattern may result from remediable causes. The anamnestic history is by far the most productive investigation. It should include whether hearing problems have been excluded by previous testing (and, if not, an expert assessment should be arranged), and any injuries or diseases are potentially damaging the brain. The strengths and weaknesses of the family environment need to be assessed; they may dictate the choices of treatment. Physical examination should be sufficient to detect congenital anomalies, skin lesions, and motor abnormalities that can be the pointers to a neurological cause. Psychometric assessment is desirable whenever there are problems at school, both to generate an idea of developmental level against which the 'developmental inappropriateness' of behavioural symptoms can be judged, and to detect barriers to learning that may be the reason for inattentiveness. Special physical investigations are not routinely necessary. EEG often yields evidence of immaturity, but this does not advance assessment much and is not routinely indicated. It is valuable in the investigation of epilepsy and in the rare cases when deterioration of function suggests the possibility of a degenerative disorder. Blood tests should be planned only on the basis of history and examination, but may include tests of thyroid function, lead (in high-lead areas) chromosomal integrity (including fragile-X probe) when there is other evidence of developmental delay, and specific DNA tests when there is clinical suspicion of a phenotype such as that of Williams syndrome.

Epidemiology

Prevalence estimates vary widely; but most of the variation between studies comes from differences in definition[6] A community survey in London of more than 2000 6–8-year-old boys found a continuum of severity on rating scales: at each successively higher level of hyperactive behaviour there were successively fewer number of children[8] The genetic evidence also supports a continuum: in a population-based twin study, the influences on hyperactive behaviour were similar over the whole range of variation.[15] Estimates of prevalence are therefore critically dependent upon the cut-off point chosen.

Two major influences on the cut-off are the diagnostic criteria applied and the cultural attitudes of raters. Attention Deficit/Hyperactivity Disorder has a rate in the school age population usually given at about 5 per cent, but varies from 2.4 to 9 per cent[6,16] probably depending on how rigorously 'impairment' is defined. The ICD-10 diagnosis of Hyperkinetic disorder yields rates around 1 to 2 per cent of the school age population.[6,17] Sex differences are marked: population surveys suggest that 2–3 boys are affected for every girl.[18]

The frequency of hyperactive behaviour in the population, at least as indexed by rating scales in surveys, has not been increasing over the last two decades.[19] By contrast, there have been large increases in the frequency with which hyperactivity as a medical condition has in practice been recognized—most obviously evidenced by a great increase in the rates of stimulant prescription between 1995 and 2005 in the UK (Wong *et al*, in submission) and a continuing increase in the USA.[20] The studies suggest that stimulant medication is given for about 3 children per 1000 in the UK (i.e. about 12 per cent of those in the community meeting ADHD criteria with impairment) and about 40 per 1000 in the USA. It is likely that health service organization plays a part in determining recognition. In the USA survey, a diagnosis of ADHD was more likely to have been made for children whose families Carried Health Insurance.[20] In a UK survey, children with high hyperactivity as rated by teachers and parents seldom received a diagnosis, with the main filter coming at the level of recognition by primary health care services.[21]

In adult life, those who were hyperactive as children still have an elevated rate of hyperactivity and related social impairment (reviewed systematically by Faraone *et al.*).[22] Indeed, a cross-sectional population survey of adults described a surprisingly high prevalence rate of about 4 per cent, with a high rate of co-existent psychological morbidity.[23] More evidence is needed on the extent of the adult problem. It is however clear that a substantial number of adults, who were not diagnosed in childhood, may be affected, and an increasing number are presenting for the first time to adult services.

Aetiology

Genetic inheritance

Genetic influences are strong: Twin studies suggest a heritability around 80 per cent, making it one of the psychological disorders most strongly influenced by genetic inheritance,[24,25] and adoptive family studies concur in emphasizing the strength of association with biological relatives.[26] Indeed, several DNA variants in genes coding for relevant proteins have now been identified and replicated.[27] In particular, the genes coding for the dopamine D4 and D5 receptors, the dopamine transporter, SNAP25 (affecting synaptosomal protein), the serotonin 1b receptor and the serotonin transporter have all been associated with ADHD by more than one group of investigators. Several kinds of caution are, however, needed in interpreting these findings. The odds ratios are all quite small (between 1.1 and 1.5), no polymorphism so far found is either necessary or sufficient; it is possible that there are subtypes of ADHD with different genetic influences[28]

Current research continues to seek more associated genes, especially by genome scans and positional cloning and to emphasize the likely importance of gene-environment interactions. Individual studies have reported that the risk alleles for genes in the dopamine system magnify the effects on the foetus of maternal smoking and alcohol consumption during pregnancy,[29,30] and catechol o-methyl transferase (COMT) of low birth weight[31]

Environment

Environmental influences are reviewed by Taylor & Warner Rogers.[32] There are associations with several kinds of adversity in fetal and early postnatal life; and genetic factors may influence the exposure to some hazards (e.g. to lead, via playing in contaminated areas) as well as their impact. Many of the insults have generalized effects on brain development and can also lead to low IQ.

Prenatal

The prenatal factors implicated include smoking and drinking in pregnancy,[33] cocaine,[34] maternal stress during pregnancy,[35] anticonvulsant use[36] and the factors causing very low birth weight.[37] For some of these, there is experimental evidence for a harmful effect in animals. Smoking, for example, has high biological plausibility: the substances inhaled have an effect in animal models, and there is a dose-response relationship in human studies.[38] It is important to recognize these risk factors in assessing a referred child, because one may be able to prevent a subsequent child from suffering the same injury. Interpretation of a positive history, however, is not straightforward, because of the likely effects of genetic influences as well. There is no doubt of the existence of the fetal alcohol syndrome, nor that it can include ADHD symptoms, but the effect of lesser degrees of exposure is uncertain. Apparent associations could be magnified by gene-environment correlations. Maternal drinking may be influenced by the same genes that influence ADHD; the genes and the pregnancy toxin may be handed down together. Knopic *et al.*[39] investigated this by studying the offspring of mothers who were identical twins yet differed in whether they had a history of alcohol abuse: ADHD was common in both groups: the suggestion was that the genes were more important than the presumed exposure to alcohol.

Postnatal

In postnatal life, the best defined risks are at extreme levels of misfortune. Head injury and brain disease have to be severe before they have a causative effect; minor injury is often a result of hyperactivity rather than a cause.[32] Children who experienced extreme deprivation in the orphanages of Romania showed increased rates of pervasive and persistent overactivity and inattention in later childhood, even though they had been adopted into English families before the age of 4 years.[40] Minor degrees of psychological adversity have not been shown to cause ADHD (though they may well be associated with coexistent conduct disorder). Indeed, the twin studies that show genetic influences can also be used to distinguish between the environment that all children in the family share (such as a chaotic family life style or the use of television), and the environmental influences that affect one child but not another; only the latter play a part.

Diet is often blamed for hyperactivity. There is some truth in it, but the effects seem to be modest. The main evidence comes from therapeutic trials (see under 'Treatment') which indicate that a range of foodstuffs can be harmful for individual children – including cow's milk, wheat flour, eggs, and artificial colourings and additives. Individual idiosyncrasies seem more important than a damaging

effect of the substances on everyone. Experimental trial, however, giving colourings including tartrazine to an unselected population of preschool children, suggests that the substances have a small but measurable adverse effect on behaviour across the whole range and so ought to be seen as mildly toxic.[41]

Pathogenesis

The effect of these aetiological influences on the developing brain is being clarified by the neuroimaging possibilities being created by magnetic resonance and other non-invasive techniques. Several brain areas are smaller in ADHD than controls.[42] The difference persists through adolescence into adult life and is more marked in those who have never received medication than those who have. The areas most affected—frontal, striatal and cerebellar—are involved in self-organizational abilities that fail in those with ADHD.

At a neuropsychological level, there have been extensive comparisons between young people referred for, and diagnosed with ADHD in the USA and age-matched controls without psychopathology. 'Executive function'—which has become a broad and ill-defined term for psychological processes by which people modify their responsiveness to stimuli or the organization of their responses—has received special attention and is reviewed by Willcutt *et al.*[43] In general summary, many such functions show significant differences between ADHD and controls, but the effect sizes are modest and do not suggest that research has yet hit on either a fundamental deficit or on a means of diagnosis to replace behavioural description.

Motor inhibition and cognitive inhibition have received particular attention, deriving from the behavioural observation that children with ADHD can be described as 'disinhibited', and from an influential suggestion by Barkley[44] that failures of inhibitory process could underlie the other cognitive deficits – such as inefficient planning ahead, and poor self-control by internal language. There is not much doubt that experiments reliably produce poor performance in ADHD on tests of suppressing motor responses.[45] Indeed, functional neuroimaging has found that people with ADHD, as a group, show less activation of brain structures involved in response suppression, even when they are performing at a satisfactory level on a simple test.[46] There is more uncertainty about whether this form of impulsiveness does indeed derive from deficits in inhibition or from other kinds of psychological alteration, such as reluctance to put effort into planning responses of any kind, or to be patient during a period of waiting. This last idea, 'delay aversion', has been elaborated and tested[47] and suggests that some children with hyperactive behaviour are still capable of delaying a response when appropriate provided that the length of time they have to wait for the reward is controlled. A head-to-head comparison of inhibition failure action (in a 'stop' test) and delay aversion (in a test of delaying gratification) has been carried out, with the result that either test on its own produced a moderate distinction between ADHD and controls, but combining the two resulted in a much better discrimination, with sensitivity and specificity around 80 per cent.[48]

The clinical applicability of the extensive research investment in psychological testing is rather small. The tests have for the most part lacked either standardization or establishment of test–retest reliability; the interpretation of an individual child's score, accordingly, lacks quantitative support. There are a few tests of related abilities that have normative values with age standardization (e.g. the Tests of Everyday Attention for Children: TEACH). Their place in practice is not to make a diagnosis of ADHD, but to suggest which of several possible cognitive weaknesses apply in the individual child. In principle, useful advice for education could follow from such testing; but evaluations—of the uptake by teachers, of the advice or the impact on the child—are lacking.

Course and prognosis

First 3 years

A 'difficult temperament' in early childhood includes overactivity and poor self-regulation, and can have a harmful effect on parent–child relationships; but the concept of inattentiveness is hard to apply at this age and the diagnosis would be insecure.

Age 3–6

ADHD behaviours are clearly recognizable by this age, and there is a strong likelihood of persistence into the school years.[49] Parent training is an effective intervention (see 'Treatment') and should be available for parents with children at risk, without waiting for formal diagnosis.

Age 7–11

School and peer demands make ADHD behaviours impairing; the tolerance of families and the culture at large help to determine whether ADHD is seen as a problem; and this is a very common age for referral and diagnosis. Hyperactivity (as opposed to inattentiveness alone) becomes important in generating aggressive and antisocial behaviour and delinquency.[50] The extent to which there is a poor social outcome may depend upon genetic influences,[51,52] on environmental influences such as a hostile home atmosphere,[53] and on gene–environment interactions (a COMT gene polymorphism together with a low birth weight predicted the development of antisocial symptoms in those with ADHD).[31]

Age 12–18

During adolescence, there is a maturing in the abilities of self-control, and some children with ADHD will lose their problems; but the demands for self-control rise as well, and so the children are still more impulsive and inattentive than their peers and four times as likely to merit a psychiatric diagnosis.[54] Indeed, about half of cases diagnosed in childhood will retain the full diagnosis in adolescence.[55]

Those who continue to show hyperactivity are at risk for other problems, notably aggressive and antisocial behaviour and delinquency,[50] and motor traffic accidents.[56]

Adult life

By adult life, most will no longer meet full diagnostic criteria for ADHD; but, equally, most will retain some functional impairment related to hyperactivity.[22] This should imply a falling prevalence, but survey of adults has found high rates (about 4 per cent).[23] Some part of this discrepancy may derive from adults developing impairment for the first time; they may have had ADHD symptoms as children, but the symptoms were not impairing, and have only become impairing when adult life imposes responsibility and high expectations.

The implications for practice are that from childhood to early adult life, and perhaps longer, severe levels of hyperactivity and inattentiveness should be seen as potentially chronic disability; and that intervention should not target only the core symptoms but also the surrounding tangle of adverse personal relationships and educational failure.

Treatment evaluations

Medication

There have been many trials of central nervous stimulants (especially methylphenidate, with some work on dexamfetamine and pemoline) and atomoxetine. A systematic review was undertaken by NICE (National Institute for Clinical Excellence).[57] Sixty-five trials met quality criteria and were assessed. Quantitative review indicated heterogeneity among the trials, so a meta-analysis was not attempted; but there was no doubt about the superiority of methylphenidate, atomoxetine, and dexamfetamine to placebo. Economic analyses were undertaken and were not very robust, but suggested that all three gave acceptable cost per Quality-Adjusted Life Year. All three should be in clinical use, with the decision regarding which product to use to be based on comorbidity, adverse effects, compliance, potential for drug diversion, and individual preferences with differences in cost as a secondary consideration.

Several proprietary preparations of methylphenidate have appeared that offer an extended release through the day; they differ in the physics of their delivery systems and therefore in their speed of onset and duration of action. Banaschewski *et al.*[58] made a systematic review of trials on them and on atomoxetine, which also has a sustained effect through the day. They indicated that the effect size of extended-release methylphenidate preparations was comparable to that of immediate-release—around 0.8–1.1 SD; but, not surprisingly, the effect of an 8-hour preparation was somewhat smaller than that of a 12 preparation on parent ratings, though similar on teachers' ratings of child behaviour. The effect size of atomoxetine was around 0.6 SD.

Most studies have been carried out on children and adolescents of school age. In children under 6 years, the limited trial evidence suggests that stimulants are more effective than placebo in reducing hyperactivity and the level of stress in family relationships[59] The safety of the drugs in this age group is uncertain. For adults, enough randomized controlled trials have appeared for stimulants and atomoxetine that meta-analysis has been possible, with the conclusion that they are more effective than placebo.[58,60]

Psychological evaluations

Behaviour therapy has received several trials, but no satisfactory systematic review has yet appeared. Miller *et al.*[61] attempted one, but decided to exclude most of the trials because they did not meet the quality criteria that were imposed. Nevertheless, reasonably good effect sizes have been reported in randomized trials comparisons for the comparisons of behaviour modification programmes (usually delivered on an individual family basis) with no treatment or treatment as usual.[62,63] Group programs of parent training—which typically include supportive education in behavioural management—are also effective, perhaps particularly for preschool children.[64,65] Cognitive therapy, by contrast, has been disappointing in trials.[66]

Elimination diets

Several trials of eliminating foods that seem to be incriminated for an individual child, followed by double-blind administration of those foods in experimental design, have found that the identified foods can worsen that child's behaviour more than a placebo.[67] The implication, as for food effects on disorders such as eczema, is of idiosyncratic intolerances so that each child needs investigating individually. This is troublesome for families, and perhaps only applicable to younger children whose diet is still under parental control.

Drug vs. psychosocial intervention

There has been controversy over the relative merits of medication and behaviour therapy. In the USA, the debate has been sharpened by a perceived over-prescription of drugs and led to a large-scale random-allocation non-blind trial.[68] The trial compared rather idealized versions of: medication (with very careful and systematic monitoring of dose and response), behaviourally oriented psychosocial therapy (delivered with high intensity and a combination of approaches to teachers, parents and the young people themselves), both interventions given together, and a 'treatment as usual' policy of referring back to community agencies (which usually resulted in medication). At the prime outcome point—14 months after randomization—the outcome for those given the research style of medication was better than those given behavioural treatment only and considerably better than those given treatment as usual, even when that included medication. Adding medication to behaviour therapy improved the outcome for the primary measures of hyperactive behaviour; adding behaviour therapy to medication did not—but did yield better control of aggression at home, improvement in the overall sense of satisfaction of parents, lower medication dosage, and a higher rate of very good outcomes. These improvements in the combination treatment were real, but very expensive to achieve, and it remains uncertain whether such benefits could be matched by behaviour therapy delivered under the constraints of ordinary practice. The marked superiority of careful medication to other forms of intervention did not persist at later follow-up points. At 2 and 3 years after the start of the trial, those who had been allocated to all arms of the trial showed rather similar outcomes. None were untreated, and all groups showed less hyperactivity than at the beginning of the trial, so the finding should not lead to therapeutic nihilism. The likely reasons for the waning of the medication effect are that the drug loses its effect, stops being taken, or depends upon careful and skilled adjustment of dosage in the longer term.

Management

Psychoeducation

Unlike most psychiatric conditions, a diagnosis of ADHD is often sought by parents and welcomed by them. The image, of being a physically caused neurological disease, is often perceived as a relief from the stigma of mental disorder. On the other hand, the media controversy over whether it is a 'real' disorder, and over the use of controlled drugs, leaves some parents confused and fearful.

Assessment on the principles above will have led to an individual formulation of the nature and causes of the impairment. Extended explanation is worthwhile in the longer term. An over-simple

description in terms of a chemical deficiency in the brain may seem a useful starting point but can lead to unrealistic expectations for treatment and frustration with the doctor or, worse, with the child. A model of chronic disability is in keeping with the evidence from longitudinal studies; but needs to be modulated by the good outcome for some children, the improvement for most, and the ability of warm and encouraging parenting to reduce the risks of antisocial behaviour in later childhood and adolescence.[53]

Children's understanding of their problems is also worth a good deal of effort. Little research has so far addressed the issue, but it is important to their ability to cope. They need to know that their problem is understood, that treatments are available, that they can influence their outcome by their own actions, and that the people around them understand all this and can be encouraging. Positive role models are useful: some successful sports stars, performers, politicians and business people have outed themselves as having, and sometimes using, ADHD. Explanations need to be repeated as the young people mature and expect a fuller and more interactive discussion.

Explanation is often needed by teachers as well. They may need to revise their expectations of the level of challenge with which the child can cope; and for some frustration can lead to antagonism towards the child's family. If they already see ADHD as a neurological disease, then the frequent observations of changeability in the children, and of ability to cope sometimes with difficult tasks, may make them reject a neurological cause—and with it the diagnosis and the validity of drug treatment. They may need to know that physical and psychological factors can both enter into the child's presentation and that the effect of medication does not depend on the aetiology.[10]

After explanation comes basic advice on helping the children's development. The first steps with parents are to establish whether there is already a framework of frequent warm interactions and effective ways of giving instructions and following up children's actions with consistent patterns of reward or loss of reward. If this does not already exist, then a parent training group is often helpful. Both a supportive atmosphere and the teaching of skills in behaviour modification seem to be necessary. The target behaviours for modification are often the ones most troublesome to parents—disobedience and aggression—rather than restlessness or inattentiveness specifically.

Liaison with schools should include advice on the severity of the problem and the intensity and nature of extra help that will be required. Teachers will often be able to share good practice in classroom management. One of the principles is to maintain good stimulus control, for instance by having the affected child at the front of the class under the teacher's eye. Another is to find opportunities for the children to let off physical energy (they can sometimes be used as messengers between classrooms) and to learn in short chunks. Variety and interest in the material to be learned or understood is useful. Transitions between activities in the classroom are often the time for children to become disorganized, and the child with ADHD should be the first to change activity with the teacher's supervision. Individual attention is probably the most effective resource in the classroom, but it is also very demanding: a classroom assistant may help to achieve it. Star charts for younger children and token economy systems for older ones are often recommended, but usually depend upon the system used for the rest of the class.

Specific interventions

When straightforward advice is not enough, then the two best-evaluated treatment approaches are *behaviour modification* and *medication management*. The choice of which to start with will depend on several conclusions from the assessment: the severity of the problem (with more severe problems responding preferentially to medication rather than behaviour therapy[7] the availability of treatment; the willingness and ability of parents (or teachers) to engage in psychological intervention; the urgency of the problem (with medication affording a more rapid change); and the wishes of the family. Whichever approach is taken first, the other should be available without undue delay if the response is below expectation.

(a) Behaviour modification

The principles of behaviour modification do not differ from those used in other kinds of behaviour problem (e.g. 62). Target behaviours should be clearly specified and monitored; the antecedents and consequences of the behaviours should be understood and modified as appropriate; clear schemes of reward and punishment should be established, understood by the child, and applied consistently. There are, in addition, some modifications to suggest for the specific needs that come from the nature of ADHD. The rapid delay-of-reward gradient calls for contingencies to be applied with particular attention to speed. For example, a kitchen timer can be set for an appropriate length of activity depending upon the individual child (for instance, 5 min application to homework, or 30 min spent free of aggression to siblings). When the timer sounds, an obvious reward (such as a token) is given within a very few seconds. The reward may swiftly lose its reinforcing quality with repetition, so frequent changes in the reward (or the backup to a token) are needed. Impairment in error correction may make it all the more necessary to be explicit and swift in explaining to the child which of their behaviours has earned the reward, or its loss. Response cost (such as loss of tokens) is usually advocated in conjunction with the reward scheme.

(b) Medication

Prescription of medicines can be guided by published schemes (e.g.[13]). Specialist assessment is highly desirable when problems are at the level that warrants medication—not because the treatment is specially risky, but because it is important that remediable causes and associated conditions are not overlooked. The first choice of medicine is usually methylphenidate. If immediate-release is chosen, then one usually begins with doses, three times a day about 5 mg to 10 mg, depending on the child's weight. If there are no adverse effects, then the dose is increased upwards (probably weekly) until there is a good response, or adverse effects become troublesome, or the ceiling of 0.7 mg/kg/dose is reached—whichever comes first. If an extended-release preparation is chosen, then a similar policy is followed of starting at a low level (e.g. 10–20 mg as a single dose) and titrating in the light of response.

The choice of immediate—or extended—release preparation should be discussed with the family. School children often have a strong preference for a single tablet to be taken in the morning before school, so as to avoid stigmatization. Schools should also be part of the decision making, because of the organizational problems for them of maintaining secure storage and accurate administration. On the other hand, advantages of immediate-release include lower cost and the possibility of accurate control of the profile of action through the day.

Individual variation in drug response is considerable, so good monitoring is a key to achieving good effects. A simple rating scale such as the abbreviated Conners is suitable: a short scale is more likely to be completed than a long one. The wide variety of presentations means that key problems for the individual child may not be included on a standard scale. An individualized scale can therefore be constructed as part of the assessment and used as the prime outcome measure. Ratings by teachers are particularly important, but communication problems can mean that their voice is not heard. If the dose is set only by the level seen as optimal by the parents, then there is a danger of over-treatment. The child's behaviour will be seen at home in the mornings and evenings of schooldays, i.e. at times when the blood level of medication is lower than during school hours. The best dose for mornings and evenings may then lead a child being over-controlled and unspontaneous during school hours. Internet feedback from class teacher can be quick and accessible, but care is needed to maintain confidentiality. Telephone monitoring is useful, especially to allow frequent adjustments in the initial phase of setting dosage, but cannot replace individual contact.

Physically, blood pressure and height and weight need regular checks; mentally, the examiner should be alert to the possibilities that agitation, depression, loss of spontaneity and perseveration can appear as a result of medication and not only as part of the condition.

Under some circumstances, atomoxetine is the medication of first choice. It is not a controlled drug, and does not maintain an illicit market, so it may be preferred if there is a substance-misusing family member. The media controversy over the use of stimulants has entailed that atomoxetine may be acceptable to some families who reject 'Ritalin'. It may also be preferred in the presence of Tourette disorder and perhaps of high levels of anxiety. Children who have failed to respond to a stimulant may nevertheless show a good response to atomoxetine. The balance of adverse effects is somewhat different and atomoxetine may therefore be preferred when, for example, insomnia has resulted from stimulants or is a major problem in itself. The action of atomoxetine may take some weeks to appear, and close titration is not recommended. Rather, a test dose around 0.5 mg/kg is given (in case adverse effects appear even on a small dose), and is followed after a week by an increase to 1.2 mg/kg.

Treatment in comorbid conditions

In general, and as considered in 'Diagnosis' above, the cluster of ADHD symptoms is similar whether or not co-existent disorders are present. The principles are for the most part the same as when treating uncomplicated ADHD. In the most frequent combination—of ADHD and conduct disorder—stimulant treatment can reduce antisocial problems as well as the core of ADHD[57] and is often worth trying even before conduct disorder is addressed.

(a) Anxiety

In the combination of ADHD and anxiety states, there is some trial evidence that the superiority of stimulant to placebo is less than in ADHD without anxiety.[10,69] There may need to be particular attention to monitoring both problems in establishing the correct dose level, and atomoxetine will sometimes be chosen. The reasons for anxiety should be sought and corrected.

(b) Pervasive developmental disorders

When an autism spectrum disorder is also present, then treatment of ADHD with stimulants is possible, but particular attention needs to be given to the possibility of exacerbating social withdrawal and repetitive patterns of behaviour, and monitoring these should be given a priority as high as detecting the desired effects. The RUPP Autism Network[70] treated 72 cases in a design with a 1-week test period, 4 weeks randomized crossover, and 8 weeks of continued treatment for those who responded well. Methylphenidate produced a better reduction of hyperactive behaviour than did placebo. The most satisfactory dose level was a modest 0.25 mg/kg.

(c) Substance misuse

In the presence of substance misuse, many clinicians are wary of prescribing the potentially misusable stimulants. There may be too much hesitation. People with ADHD taking stimulants show lower rates of substance misuse than those who do not take prescribed medication.[71]

(d) Epilepsy

The presence of epilepsy raises extra needs in assessment before treatment. In poorly controlled epilepsy, ADHD symptoms may be the direct result of very frequent small seizures ('absence status' at the extreme) or of very frequent seizures at night, so ambulant and sleeping EEGs are useful. In less extreme cases, brief lapses of attention can be the result of minor seizures causing transient cognitive impairment; simultaneous recording of EEG with behaviour observation and/or psychological test performance is the best way of getting the answer. Anticonvulsant medications can also cause disturbances of attention and irritability; the clues come from high blood levels of anticonvulsants, low folate levels, polypharmacy, and a temporal relationship between drug changes and hyperactivity or inattention. Once these are excluded, then treatment of ADHD can proceed as usual. Methylphenidate, it has been copied from textbook to textbook, can worsen epilepsy. I have not been able to find empirical evidence for this, do not find that it matches with clinical experience and regard methylphenidate as safe in controlled epilepsy. Atomoxetine has had numerous reports of seizures following its use, but very few of first seizures. In uncontrolled epilepsy, and especially where there is a risk for status epilepticus, I prefer to use dexamfetamine.

Review after a satisfactory response

When a child has responded well to the first treatment chosen, a specialist's review of the case is in order.

Is the improvement sufficient? Impulsiveness and inattentiveness may not have disappeared entirely; but the goal should be that they are no longer impairing. If the problems are still more than minor, then the other main intervention should be explored.

What has happened to any co-existent disorders: are they satisfactorily resolved or is further treatment or referral indicated?

Has the improvement led to different understanding of the child? Old habits of reacting to the children or setting expectations for them may need to be modified. At home, parents may well have found that they set disciplinary sanctions at a high level when the need was to have an impact on an inattentive person. Those sanctions may be too severe, for a person who is now more responsive to reward and punishment and lead to distress or discouragement.

At school, there may be opportunities for normalizing the curriculum. Social skills learning, which may have been abandoned in the past because of the child's failure to profit, may now be well worth another try.

Does the child understand the nature of the improvement? This, like the nature of the disability, will need discussing in different ways as the child matures. The initial reaction may well be one simply of relief at being out of trouble. If, however, medicines are seen as a tablet 'to make me good', this attitude may lead to a rejection of medicine in adolescence when rejecting other aspects of adult authority. There are usually many decisions to make about medication—whether to take it during the school holidays and at weekends, whether to vary the dose in line with environmental demands, and whether to continue taking it even though indulging in alcohol or cannabis. There is every reason to involve the child as an active agent in these decisions and to help them to learn from the consequences.

For how long should the treatment continue? Scientific study has not given a secure answer to this question, and individual decision making is needed. Periodic spells off medication—perhaps for a fortnight every two years—is a good way of deciding whether it is still needed. These are also good times for the patient to review why he is taking it, and perhaps to seek the reactions of others to his state off medication.

Are there satisfactory follow-up arrangements? Shared care between primary care and the specialist service is the ideal, with physical monitoring (perhaps 6-weekly) and minor dose adjustments carried out in primary care, and psychological monitoring and strategy decisions about therapy (perhaps 6-monthly) carried out by the specialist team.

Review in refractory cases

A case can be considered refractory when the problems are still impairing after the exhibition of methylphenidate (or dexamfetamine), atomoxetine and behavioural therapy.

There are several reasons for failure at the first line, of which failure to follow treatment is the commonest. If behaviour modification was the first line tried, and the child was too hyperactive for it to be adequately delivered, then it may well be worth another try in combination with medication, even if the medication alone was not obviously successful. If medication was not taken, then the reason may have been stigma and careful discussion may allow a more successful attempt. Public controversy about ADHD and medication has been intense for decades and has not been resolved by increasing knowledge. It is right for there to be strong debate, not least because the issues raised—of whether it is legitimate to make changes in one's learning and social abilities through physical methods—arise in many other areas of public concern. Unfortunately, however, some journalism accuses parents and teachers of bad faith, in pretending a physical cause to disguise failings in parental childrearing or inadequacies in schools. This is understandably disquieting for children and those around them. Involvement with a user group can help to maintain a positive attitude to overcoming disability, and counter some of the noxious attitudes expressed in some of the media.

Another reason for failure of medication is the appearance of adverse effects, either precluding the treatment or limiting the dose to subtherapeutic levels. Symptomatic treatment of adverse reactions is often possible.

Appetite loss can result from stimulants (and less commonly from atomoxetine). This will often disappear towards the end of the day, as the medication is cleared from the body, so increased intake in the evenings (or at weekends or other holidays from stimulant medication) is often enough to prevent faltering in growth.

Insomnia is a frequent complaint, but should be carefully recorded at baseline as it often precedes medication. The commonest problem to result from stimulants is a delay in settling and falling asleep. Sleep hygiene measures can help, for instance, a planned deceleration of activity towards bedtime (perhaps in a place other than the bedroom, to avoid conditioning the bedroom environment to wakefulness); a reduction in light intensity (including sitting farther away from the television or computer screen); and prescription of melatonin shortly before settling. A switch from stimulants to atomoxetine may be needed.

Tics can be worsened or produced by stimulants. Some people find that mild tics are a price worth paying for the beneficial actions, or not even notice them; but they can be disfiguring and stigmatizing. A switch to atomoxetine will often be the first action; or the combination of methylphenidate with clonidine may be useful in both reducing tic severity and reducing the necessary dose of methylphenidate.

Treatment can also fail because the initial assessment was incomplete and set the wrong targets for therapy. Another disorder may have masqueraded as ADHD (see 'Diagnosis') and a failure to respond should raise the index of suspicion for the presence of autistic or hypomanic overactivity. (The converse, however, does not hold; a response to methylphenidate does not make the diagnosis of ADHD, because qualitatively similar changes can be seen when ordinary children receive a stimulant, vide Rapoport *et al.*)[72] A variant of this comes when ADHD was present, but not the main problem, and the main problem perceived by parents or teachers is not drug-sensitive.

When the above reasons for failure to respond to treatment have been considered and dealt with, then the psychiatrist should consider some of the wide variety of unlicensed medications that have been shown effective in randomized controlled trials.

Some noradrenergic agents (clonidine, guanfacine), may act to stimulate presynaptic autoreceptors and may downregulate noradrenergic activation. They can be useful when there is a great deal of agitation as part of the symptom pattern (e.g. in autism). They do not, however, improve the cognitive aspects of inattentiveness.

Modafinil has effects in reducing hyperactivity and may enhance cognition as well. A licence has been applied for, but was interrupted by the possible emergence of skin disorders as a complication.

Tricyclic antidepressants (e.g. imipramine, protriptyline) and some other antidepressants (bupropion, but not SSRIs) are more effective than placebo in reducing hyperactivity; their effect often wanes after a few weeks or months, but they can be useful for short periods, e.g. to allow a period off stimulants in a child with a growth problem.

Monoamine oxidase inhibitors also reduce hyperactivity; they are in general unsafe for use in children, partly because of the difficulty in maintaining dietary restrictions, but the reversible MAOIs such as meclobomide are somewhat safer and could be considered. Nicotine patches can be considered for their combination of cognitive and behavioural effects, but quite often produce nausea and local irritation.

Risperidone and other atypical neuroleptics are often used, especially in intellectually impaired populations, but have not been evaluated for the treatment of ADHD. Their power, and the reason they are prescribed, is in the symptomatic reduction of severely aggressive and agitated behaviour rather than the improvement of attentive and reflective behaviour. Clarity of indication is therefore important, and their benefits should be set against the many hazards.

Some efficacious drugs have been contraindicated because of rare but severe, adverse effects: pemoline after reports of liver failure, desipramine because of cardiac toxicity. In general, the use of unlicensed or unevaluated drugs should be embarked on only by prescribers with specialist experience, who obtain carefully informed consent and monitor appropriately. The most effective medication protocol yet evaluated was that of the MTA approach (see above), in which stimulants were sufficient for about 90 per cent of cases and there was little recourse to the second line of drugs.

Treatment in adult life

A key problem in current knowledge is that of making an accurate diagnosis when adults present for the first time. Adults may be mistaken in identifying themselves (see above); but their recall of their childhoods is a reasonably reliable predictor of their parents' ratings.[73] Self-report scales have emerged[74, 75] but are not yet fully validated. The account of somebody who knows the patient well—perhaps a spouse or a partner—is very desirable, but does of course need interpreting in the light of their own interests in a diagnosis.

Psychosocial treatment for adults is not yet well evaluated. In principle, adults ought to have greater capacities for cognitive and other self-instructional approaches. Young and Bramham[76] provide a useful guide. Simply the giving of a diagnosis comes as a relief to some who have puzzled over the reasons for their failures, and can liberate problem-solving approaches.

Treatment with stimulant drugs and atomoxetine has been evaluated by several randomized controlled trials in adults (reviewed by Faraone *et al.* and Banaschewski *et al.*).[58,60] The drugs are efficacious. Only atomoxetine has a licence in Europe, and that only when treatment was started in childhood; but it does not seem reasonable to withhold a therapy because it was unavailable to the person earlier. Their use follows similar principles to those described above for children, and Asherson[77] provides a guide.

In conclusion, this chapter has presented a picture of ADHD and its severe form, hyperkinetic disorder, as disabilities that change with development and are often accompanied by other problems that can mask it or themselves be masked by it. They are rewarding challenges for diagnosis and treatment in adulthood as well as during childhood and adolescence.

Further information

The National Institute of Mental Health (NIMH) Website: http://www.nimh.nih.gov/health/topics/attention-deficit-hyperactivity-disorder-adhd/index.shtml

The National Attention Deficit Disorder Information and Support Service (ADDISS) Website: www.addiss.co.uk

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9.2.5 Conduct disorders in childhood and adolescence

Stephen Scott

Introduction

The term conduct disorder refers to a persistent pattern of antisocial behaviour in which the individual repeatedly breaks social rules and carries out aggressive acts which upset other people. It is the commonest psychiatric disorder of childhood across the world, and the commonest reason for referral to child and adolescent mental health services in Western countries. Antisocial behaviour has the highest continuity into adulthood of all measured human traits except intelligence. A high proportion of children and adolescents with conduct disorder grow up to be antisocial adults with impoverished and destructive lifestyles; a significant minority will develop antisocial personality disorder (psychopathy). The disorder in adolescence is becoming more frequent in Western countries and places a large personal and economic burden on individuals and society.

Relation to other disorders

Conduct disorder is one of the two *disruptive disorders* of childhood, (also known as *externalizing disorders*); the other is the hyperkinetic syndrome (ICD 10), a more severe form of attention-deficit hyperactivity disorder (ADHD, DSM IV-R). Conduct disorder and the hyperkinetic syndrome are distinct disorders but often co-occur. As discussed in Chapter 9.1.1 on classification, disruptive disorders can be distinguished on a number of criteria from the other main grouping of child psychiatric conditions, the *emotional disorders* (also known as *internalizing disorders*). For example, unlike emotional disorders, disruptive disorders are commoner in boys, the socially disadvantaged, children from large families, and where there is parental discord.

Juvenile delinquency is a legal term referring to an act by a young person who has been convicted of an offence which would be deemed a crime if committed by an adult. Most, but not all, recurrent juvenile offenders have conduct disorder. In this chapter the term conduct disorder is used as defined by ICD 10 diagnostic criteria; the term conduct problems will be used for less severe antisocial behaviour.

Social problem or medical diagnosis?

Infringement of the rights of other people is a requirement for the diagnosis of conduct disorder. Since the manifestations include a

failure to obey social rules despite apparently intact mental state and social capacities, many have seen the disorder as principally socially determined. They therefore believe the responsibility for its cause and elimination lies with people who can influence the socialization process, such as parents, schoolteachers, social service departments, and politicians. Due to the impossibility of their seeing all cases, there is some debate within child and adolescent psychiatry as to whether doctors and mental health professionals should be involved in any but the most complex presentations.[1] Some have argued that involvement of medical personnel carries the risk of their becoming agents of social control through the misapplication of diagnostic labels, which may lead to abuses of the kind seen in some totalitarian regimes.

However, advances in the last decade have shown there are substantial genetic and biological contributions to conduct disorder, and in some cases the symptoms may be responsive to medication. Work in the last 25 years mainly from the field of child and adolescent mental health has clarified many of the mechanisms contributing to the development and persistence of antisocial behaviour, and has led to the development of effective treatments. As yet these are not being widely used with the children and adolescents who need them. Therefore psychiatrists need to be able to contribute to the planning and delivery of an appropriate service.

Clinical features

Aggressive and defiant behaviour is an important part of normal child and adolescent development which ensures physical and social survival. Indeed, parents may express concern if a child is too acquiescent and unassertive. The level of aggressive and defiant behaviour varies considerably amongst children, and it is probably most usefully seen as a continuously distributed trait. Empirical studies do not suggest a level at which symptoms become qualitatively different, nor is there a single cut-off point at which they become impairing for the child or a clear problem for others. There is no hump towards the end of the distribution curve of severity to suggest a categorically distinct group who might on these grounds warrant a diagnosis of conduct disorder.

Picking a particular level of antisocial behaviour to call conduct disorder is therefore necessarily arbitrary. For all children, the expression of any particular behaviour also varies according to child age, so that for example physical hitting is at a maximum at around 2 years of age but declines to a low level over the next few years. Therefore any judgement about the significance of the level of antisocial behaviour has to be made in the context of the child's age. Before deciding that the behaviour is abnormal or a significant problem, a number of other clinical features have to be considered:

- Level: severity and frequency of antisocial acts, compared with children of the same age and gender
- Pattern: the variety of antisocial acts, and the setting in which they are carried out
- Persistence: duration over time
- Impact: distress and social impairment of child; disruption and damage caused to others.

Change in clinical features with age

The type of behaviour seen will depend on the age and gender of the individual.

Younger children, say from 3 to 7 years of age, usually present with general defiance of adults wishes, disobedience of instructions, angry outbursts with temper tantrums, physical aggression to people especially siblings and peers, destruction of property, arguing, blaming others for things that have gone wrong, and a tendency to annoy and provoke others.

In *middle childhood*, say from 8 to 11, the above features are often present but as the child grows older, stronger, and spends more time out of the home, other behaviours are seen. They include: swearing, lying about what they have been doing, stealing of others belongings outside the home, persistent breaking of rules, physical fights, bullying of other children, cruelty to animals, and setting of fires.

In *adolescence*, say from 12 to 17, more antisocial behaviours are often added: cruelty and hurting of other people, assault, robbery using force, vandalism, breaking and entering houses, stealing from cars, driving and taking away cars without permission, running away from home, truanting from school, extensive use of narcotic drugs.

Not all children who start with the type of behaviours listed in early childhood progress on to the later, more severe forms. Only about half continue from those in early childhood to those in middle childhood[2]; likewise only about a further half of those with the behaviours in middle childhood progress to show the behaviours listed for adolescence. However, the early onset group are important as they are far more likely to display the most severe symptoms in adolescence, and to persist in their antisocial tendencies into adulthood. Indeed over 90 per cent of severe, recurrent adolescent offenders showed marked antisocial behaviour in early childhood. In contrast, there is a large group who only start to be antisocial in adolescence, but whose behaviours are less extreme and who tend to desist by the time they are adults.

Girls

Severe antisocial behaviour is less common in girls who are less likely to be physically aggressive and engage in criminal behaviour, but more likely to show spitefulness, emotional bullying (such as excluding children from groups, spreading rumours so others are rejected by their peers), frequent unprotected sex leading to sexually transmitted diseases and pregnancy, drug abuse, and running away from home.

Pattern and setting

Prognosis is determined by the frequency and intensity of antisocial behaviours, the variety of types, the number of settings in which they occur (e.g. home, school, and in public), and their persistence. For general populations of children, the correlation between parent and teacher ratings on the same measures is only 0.2 to 0.3, so that there are many children who are perceived to be mildly or moderately antisocial at home but well behaved at school, and vice versa. However, for more severe antisocial behaviour, there are usually manifestations both at home and at school.

Impact

At home the child often is subject to high levels of criticism and hostility, and sometimes made a scapegoat for a catalogue of family misfortunes. Frequent punishments and physical abuse are not uncommon. The whole family atmosphere is often soured and siblings also affected. Maternal depression is often present, and families who are unable to cope may, as a last resort, give up the child to be cared for by the local authority. At school, teachers may

take a range of measures to attempt to control the child and protect the other pupils, including sending the child out of the class, sometimes culminating in permanent exclusion from the school. This may lead to reduced opportunity to learn subjects on the curriculum and poor examination results. The child typically has few if any friends, who get fed up with their aggressive behaviour. This often leads to exclusion from many group activities, games, and trips, so restricting the child's quality of life and experiences. On leaving school the lack of social skills, low level of qualifications, and presence of a police record make it harder to gain employment.

Classification

The ICD-10 classification has a category for conduct disorders, F91. The *Clinical descriptions and diagnostic guidelines*[3] state:

> Examples of the behaviours on which the diagnosis is based include the following: excessive levels of fighting or bullying; cruelty to animals or other people; severe destructiveness to property; firesetting; stealing; repeated lying; truancy from school and running away from home; unusually frequent and severe temper tantrums; defiant provocative behaviour; and persistent severe disobedience. Any one of these categories, if marked, is sufficient for the diagnosis, but isolated dissocial acts are not. (p. 267)

An enduring pattern of behaviour should be present, but no time frame is given and there is no impairment or impact criterion stated.

The ICD-10 *Diagnostic criteria for research*[4] differ, requiring symptoms to have been present for at least 6 months, and the introductory rubric indicates that impact upon others (in terms of violation of their basic rights), but not impairment of the child, can contribute to the diagnosis. The research criteria take a menu-driven approach whereby a certain number of symptoms have to be present. 15 behaviours are listed to consider for the diagnosis of **Conduct Disorder**, which usually but not exclusively apply to older children and teenagers. They can be grouped into four classes:

(a) Aggression to people and animals

- often lies or breaks promises to obtain goods or favours or to avoid obligations
- frequently initiates physical fights (this does not include fights with siblings)
- has used a weapon that can cause serious physical harm to others (e.g. bat, brick, broken bottle, knife, gun)
- often stays out after dark despite parenting prohibition (beginning before 13 years of age)
- exhibits physical cruelty to other people (e.g. ties up, cuts, or burns a victim), and
- exhibits physical cruelty to animals.

(b) Destruction of property

- deliberately destroys the property of others (other than by firesetting) and
- deliberately sets fires with a risk or intention of causing serious damage).

(c) Deceitfulness or theft

- steals objects of non-trivial value without confronting the victim, either within the home or outside (e.g. shoplifting, burglary, forgery).

(d) Serious violations of rules

- is frequently truant from school, beginning before 13 years of age
- has run away from parental or parental surrogate home at least twice or has run away once for more than a single night (this does not include leaving to avoid physical or sexual abuse)
- commits a crime involving confrontation with the victim (including purse-snatching, extortion, mugging)
- forces another person into sexual activity
- frequently bullies others (e.g. deliberate infliction of pain or hurt, including persistent intimidation, tormenting, or molestation), and
- breaks into someone else's house, building, or car.

To make a diagnosis, three symptoms from this list have to be present, one for at least 6 months. There is no impairment criterion. There are three subtypes: *conduct disorder confined to the family context* (F91.0), *unsocialized conduct disorder* (F91.1, where the young person has no friends and is rejected by peers), and *socialized conduct disorder* (F91.2, where peer relationships are normal). It is recommended that age of onset be specified, with *childhood onset type* manifesting before age 10, and *adolescent onset type* after. Severity should be categorized as *mild*, *moderate*, or *severe* according to number of symptoms *or* impact on others, e.g. causing severe physical injury, vandalism, theft.

For younger children, say up to 9 or 10 years old, there is a list of eight symptoms for the subtype known as **Oppositional Defiant Disorder** (F91.3):

1 has unusually frequent or severe temper tantrums for his or her developmental level
2 often argues with adults
3 often actively refuses adults' requests or defies rules
4 often, apparently deliberately, does things that annoy other people
5 often blames others for his or her own mistakes or misbehaviour
6 is often touchy or easily annoyed by others
7 is often angry or resentful
8 is often spiteful or resentful.

To make a diagnosis of the oppositional defiant type of conduct disorder, four symptoms from *either* this list *or* the main conduct disorder 15 symptom list have to be present, but no more than two from the latter. Unlike the main variant, there is an impairment criterion: the symptoms must be amaladaptive and inconsistent with the developmental level (p. 161).

Where there are sufficient symptoms of a comorbid disorder to meet diagnostic criteria, the ICD-10 system discourages the application of a second diagnosis, and instead offers a single, combined category. There are two major kinds: mixed disorders of conduct

and emotions, of which **Depressive Conduct Disorder** (F92.0) is the best researched; and **Hyperkinetic Conduct Disorder** (F90.1). There is modest evidence to suggest these combined conditions may differ somewhat from their constituent elements.

The DSM IV-R system[5] follows the ICD-10 research criteria very closely and does not have separate clinical guidelines. The same 15 behaviours are given for the diagnosis of conduct disorder 312.8, with almost identical wording. As for ICD-10, three symptoms need to be present for diagnosis. Severity and childhood or adolescent onset are specified in the same way. However, unlike ICD-10, there is no division into socialized/unsocialized, or family context only types, and there *is* a requirement for the behaviour to cause a clinically significant impairment in social, academic, or social functioning. Comorbidity in DSM IV-R is handled by giving as many separate diagnoses as necessary, rather than by having single, combined categories.

In DSM IV-R, oppositional defiant disorder is classified as a separate disorder on its own, and not as a subtype of conduct disorder. Diagnosis requires four symptoms from a list of eight behaviours which are the same as for ICD-10, but unlike ICD-10, all four have to be from the oppositional list, and none may come from the main conduct disorder list. It is doubtful whether oppositional defiant disorder differs substantially from conduct disorder in older children in any associated characteristics, and the value of designating it as a separate disorder is arguable. In this article, the term conduct disorder will henceforth be used as it is in ICD-10, to refer to all variant including oppositional defiant disorder.

Differential diagnosis

Making a diagnosis of conduct disorder is usually straightforward but comorbid conditions are often missed. The differential diagnosis may include:

1 *Hyperkinetic syndrome/Attention-deficit hyperactivity disorder*. These are the names given by ICD-10 and DSM IV-R respectively for similar conditions, except that the former is more severe. For convenience the term *hyperactivity* will be used here. It is characterized by impulsivity, inattention, and motor overactivity. Any of these three sets of symptoms can be misconstrued as antisocial, particularly impulsivity which is also present in conduct disorder. However, none of the symptoms of conduct disorder are a part of hyperactivity so excluding conduct disorder should not be difficult. A frequently made error however, is to miss comorbid hyperactivity when conduct disorder is definitely present. Standardized questionnaires are very helpful here, such as the Strengths and Difficulties Questionnaire, which is brief, and just as effective at detecting hyperactivity as much longer alternatives.[6]

2 *Adjustment reaction to an external stressor*. This can be diagnosed when onset occurs soon after exposure to an identifiable psychosocial stressor such as divorce, bereavement, trauma, abuse, or adoption. The onset should be within 1 month for ICD-10, and 3 months for DSM IV-R, and symptoms should not persist for more than 6 months after the cessation of the stress or its sequelae.

3 *Mood disorders*. Depression can present with irritability and oppositional symptoms but unlike typical conduct disorder mood is usually clearly low and there are vegetative features; also more severe conduct problems are absent. Early manic depressive disorder can be harder to distinguish, as there is often considerable defiance and irritability combined with disregard for rules, and behaviour which violates the rights of others. Low self-esteem is the norm in conduct disorder, as is a lack of friends or constructive pastimes. Therefore it is easy to overlook more pronounced depressive symptoms. Systematic surveys reveal that around a third of children with conduct disorder have depressive or other emotional symptoms severe enough to warrant a diagnosis.

4 *Autistic spectrum disorders*. These are often accompanied by marked tantrums or destructiveness, which may be the reason for seeking a referral. Enquiring about other symptoms of autistic spectrum disorders should reveal their presence.

5 *Dissocial/antisocial personality disorder*. In ICD-10 it is suggested a person should be 17 or older before dissocial personality is considered. Since at age 18 most diagnoses specific to childhood and adolescence no longer apply, in practice there is seldom difficulty. In DSM IV-R conduct disorder can be diagnosed over 18 so there is potential overlap. A difference in emphasis is the severity and pervasiveness of the symptoms of those with personality disorder, whereby all the individual's relationships are affected by the behaviour pattern, and the individual's beliefs about his antisocial behaviour are characterized by callousness and lack of remorse.

6 *Subcultural deviance*. Some youths are antisocial and commit crimes but are not particularly aggressive or defiant. They are well adjusted within a deviant peer culture that approves of recreational drug use, shoplifting, etc. In some localities a third or more teenage males fit this description and would meet ICD-10 diagnostic guidelines for socialized conduct disorder. Some clinicians are unhappy to label such a large proportion of the population with a psychiatric disorder. Using DSM IV-R criteria would preclude the diagnosis for most youths like this due to the requirement for significant impairment.

Multiaxial assessment

ICD-10 recommends that multiaxial assessment be carried out for children and adolescents, while DSM IV-R suggests it for all ages. In both systems axis one is used for psychiatric disorders which have been discussed above. The last three axes in both systems cover general medical conditions, psychosocial problems, and level of social functioning respectively; these topics will be alluded to below under aetiology. In the middle are two axes in ICD-10, which cover specific (Axis two) and general (Axis three) learning disabilities respectively; and one in DSM IV-R (Axis two) which covers personality disorders *and* general learning disabilities.

Both specific and general learning disabilities are essential to assess in individuals with conduct problems. Fully a third of children with conduct disorder also have specific reading retardation[7] defined as having a reading level two standard deviations below that predicted by the person's IQ. While this may in part be due to lack of adequate schooling, there is good evidence that the cognitive deficits often precede the behavioural problems. General learning disability (mental retardation) is often missed in children with conduct disorder unless IQ testing is carried out. The rate of conduct disorder rise several-fold as IQ gets below 70.

Epidemiology

Between 5 per cent and 10 per cent of children and adolescents have significant persistent oppositional, disruptive, or aggressive behaviour problems.[8,9] With respect to historical period, a modest rise in diagnosable conduct disorder over the second half of the twentieth century has also been observed comparing assessments of three successive birth cohorts in Britain.[10] There is a marked social class gradient.[9] With respect to ethnicity, youth self-reports of antisocial behaviours, and crime victim survey reports of perpetrators' ethnicity show an excess of offenders of black African ancestry. Importantly, Hispanic Americans in the United States of America and British Asians in the United Kingdom do not tend to show an excess of offending compared to their white counterparts.

Sex differences in prevalence

The sex ratio is approximately 2:5 males for each female overall, with males further exceeding females in the frequency and severity of behaviours. On balance, research suggests that the causes of conduct problems are the same for the sexes, but males have more conduct disorder because they experience more of its individual-level risk factors (e.g. hyperactivity, neurodevelopmental delays). However, recent years have seen increasing concern amongst clinicians about treating antisocial behaviour amongst girls.[11]

Developmental subtypes

Life-course persistent versus adolescence-limited

There has been considerable attention paid to the distinction between conduct problems that are first seen in early childhood versus those that start in adolescence[2] and these two subtypes are encoded in the DSM-IV. Early onset is a strong predictor of persistence through childhood, and early onset delinquency is more likely to persist into adult life. Those with early onset differ from those with later onset in that they have lower IQ, more attentional and impulsivity problems, poorer scores on neuropsychological tests, greater peer difficulties and they are more likely to come from adverse family circumstances.[2] Those with later onset become delinquent predominantly as a result of social influences such as association with other delinquent youths. Findings from the follow-up of the Dunedin cohort support relatively poorer adult outcomes for the early onset group in domains of violence, mental health, substance abuse, work, and family life.[2] However the 'adolescence-limited' group were not without adult difficulties. As adults they still engaged in self-reported offending, and they also had problems with alcohol and drugs. Thus, the age-of-onset subtype distinction has strong predictive validity, but adolescent onset antisocial behaviours may have more long-lasting consequences than previously supposed, and so both conduct problems warrant clinical attention.

Aetiology

Individual-level characteristics

(a) Identified genotypes

The search for specific genetic polymorphisms is a very new scientific initiative, and little has yet been accomplished. The most-studied candidate gene in relation to conduct problems is the MAOA promoter polymorphism. The gene encodes the MAOA enzyme, which metabolizes neurotransmitters linked to aggressive behaviour. Replicated studies show that maltreatment history and genotype interact to predict antisocial outcome.[12]

(b) Perinatal complications and temperament

Recent large-scale general population studies have found associations between life-course persistent type conduct problems and perinatal complications, minor physical anomalies, and low birth weight.[13] Most studies support a biosocial model in which obstetric complications might confer vulnerability to other co-ocurring risks such as hostile or inconsistent parenting. Smoking in pregnancy is a statistical risk predictor of offspring conduct problems,[13] but a causal link between smoking and conduct problems has not been established. Several prospective studies have shown associations between irritable temperament and conduct problems.[14]

(c) Neurotransmitters

In general the findings with children have not been consistent.[15] For example, in the Pittsburgh Youth cohort, boys with long-standing conduct problems showed downward changes in urinary adrenaline level following a stressful challenge task, whereas prosocial boys showed upward responses. However other studies have failed to find an association between conduct disorder and measures of noradrenaline in children.[15] It should be borne in mind that neurotransmitters in the brain are only indirectly measured, most measures of neurotransmitter levels are crude indicators of activity, and little is known about neurotransmitters in the juvenile brain.

(d) Verbal deficits and autonomic reactivity

Children with conduct problems have been shown consistently to have increased rates of deficits in language-based verbal skills.[16] The association holds after controlling for potential confounds such as race, socio-economic status, academic attainment, and test motivation. Children who cannot reason or assert themselves verbally may attempt to gain control of social exchanges using aggression; there are likely also to be indirect effects in which low verbal IQ contributes to academic difficulties which in turn mean that the child's experience of school becomes unrewarding, rather than a source of self-esteem and support.

A low resting pulse rate or slow heart rate has been found consistently to be associated with antisocial behaviour, and a meta-analysis of 40 studies suggested it is the best replicated biological correlate of antisocial behaviour.[17] Other psychophysiological indicators show that antisocial and psychopathic boys are also slowest to show a skin-conductance response to aversive stimuli.[17] The explanation for the link between slow autonomic activity and antisocial behaviour remains unclear.

(e) Information-processing and social cognition

Dodge proposed the leading information-processing model for the genesis of aggressive behaviours within social interactions.[18] The model hypothesises that children who are prone to aggression focus on threatening aspects of others' actions, interpret hostile intent in the neutral actions of others, and are more likely to select and to favour aggressive solution to social challenges. Several studies have demonstrated that aggressive children make such errors of social cognition.[18]

Risks outside the family

(a) Risks in the neighbourhood

It has long been assumed that bad neighbourhoods have the effect of encouraging children to develop conduct problems. Many parents

strive to secure the best neighbourhood and school for their child that they can afford. Although it is obvious that some local areas have higher crime rates than others, it has been difficult to document any direct link between neighbourhood characteristics and child behaviour, for a number of reasons. For example, neighbourhood characteristics were conceptualized in overly simple structural-demographic terms such as percentage of non-white residents or percentage of single-parent households. Moreover, research designs could not rule out the alternative possibility that families whose members are antisocial tend to selectively move into bad neighbourhoods. A new generation of neighbourhood research is addressing these challenges, and suggests that the neighbourhood factors that are important include social processes such as 'collective efficacy' and 'social control', do influence young children's conduct problems, probably by supporting parents in their efforts to rear children.

(b) Peer influences

Children with conduct problems have poorer peer relationships than non-disordered children in that they tend to associate with children with similar antisocial behaviours, they have discordant interactions with other children, and experience rejection by non-deviant peers. Three processes have been identified, namely that children's antisocial behaviours lead them to have peer problems, deviant peer relationships lead to antisocial behaviours, and thirdly some common factor leads to both.[19]

Risks within the family

(a) Concentration of crime in families

Fewer than 10 per cent of the families in any community account for more than 50 per cent of that community's criminal offenses, which reflects the coincidence of genetic and environmental risks. There is now solid evidence from twin and adoption studies that conduct problems assessed both dimensionally and categorically are substantially heritable.[20] However, knowing that conduct problems are under some genetic influence is less useful clinically than knowing that this genetic influence appears to be reduced, or enhanced, depending on interaction with circumstances in the child's environment. Several genetically sensitive studies have allowed interactions between family genetic liability and rearing environment to be examined. Both adoption and twin studies have reported an interaction between antisocial behaviour in the biological parent and adverse conditions in the adoptive home that predicted the adopted child's antisocial outcome, so that the genetic risk was modified by the rearing environment.

(b) Family poverty

There is an association between severe poverty and early childhood conduct problems. Early theories proposed direct effects of poverty related to strains arising from the gap between aspirations and realities, and from lacking opportunity to acquire social status and prestige. Subsequent research has indicated that the association between low income and childhood conduct problems is indirect, mediated via family processes such as marital discord and parenting deficits.

(c) Parent–child attachment

Parent–child relationships provide the setting for the development of later social functioning, and disruption of these attachment relationships, for example through institutional care, is associated with subsequent difficulties in relating. Thus, conduct problems might be expected to arise from infant attachment difficulties. One study found that ambivalent and controlling attachment predicted externalizing behaviours after controlling for baseline externalizing problems[21]; disorganized child attachment patterns seem to be especially associated with conduct problems. Although it seems obvious that poor parent–child relations in general predict conduct problems, it has yet to be established whether attachment difficulties as measured by observational paradigms have an independent causal role in the development of behaviour problems; attachment classifications could be markers for other relevant family risks.

(d) Discipline and parenting

Patterns of parenting associated with conduct problems were delineated by Patterson in his seminal work *Coercive Family Process*.[22] Parents of antisocial children were found to be more inconsistent in their use of rules, to issue more, and unclear, commands, to be more likely to respond to their children on the basis of mood rather than the characteristics of the child's behaviour, to be less likely to monitor their children's whereabouts, and to be unresponsive to their children's prosocial behaviour. Patterson proposed a specific mechanism for the promotion of oppositional and aggressive behaviours in children. A parent responds to mild oppositional behaviour by a child with a prohibition to which the child responds by escalating his behaviour, and mutual escalation continues until the parent backs off thus negatively reinforcing the child's behaviour. The parent's inconsistent behaviour increases the likelihood of the child showing further oppositional or aggressive behaviour. In addition to specific tests of Patterson's reinforcement model there is ample evidence that conduct problems are associated with hostile, critical, punitive, and coercive parenting.[23]

In considering the role of coercive processes in the origins or maintenance of conduct problems, we need to consider possible alternative explanations, (i) that the associations reflect familial genetic liability towards children's psychopathology and parents' coercive discipline, (ii) that they represent effects of children's behaviours on parents, and (iii) that coercive parenting may be a correlate of other features of the parent/child relationship or family functioning that influence child behaviours. There is considerable evidence that children's difficult behaviours do indeed evoke parental negativity. The fact that children's behaviours can evoke negative parenting does not however mean that negative parenting has no impact on children's behaviour. The E-risk longitudinal twin study of British families examined the effects of fathers' parenting on young children's aggression.[24] As expected, a prosocial father's *absence* predicted more aggression by his children. But in contrast, an antisocial father's *presence* predicted more aggression by his children, and his harmful effect was exacerbated the more time each week he spent taking care of the children.

(e) Exposure to adult marital conflict and domestic violence

It is likely that family processes other than parenting skills and quality of parent–child attachment relationships have a role. Many studies have shown that children exposed to domestic violence between adults are subsequently more likely to themselves become aggressive. Cummings and Davies[25] proposed that marital conflict influences children's behaviour because of its effect on their regulation of emotion. For example a child may respond to frightening emotion arising from marital conflict by down-regulating his own emotion through denial of the situation. This in turn may

lead to inaccurate appraisal of other social situations and ineffective problem-solving. Repeated exposure to family conflict is thought to lower childrens' thresholds for psychological dysregulation, resulting in greater behavioural reactivity to stress.[25] Children's aggression may also be increased by marital discord because children are likely to imitate aggressive behaviour modelled by their parents. Through parental aggression children may learn that aggression is a normative part of family relationships, that it is an effective way of controlling others, and that aggression is sanctioned, not punished.

(f) Maltreatment

Physical punishment is widely used, and parents of children with conduct problems frequently resort to it out of desperation. Overall, associations between physical abuse and conduct problems are well established.[15] In the Christchurch longitudinal study, child sexual abuse predicted conduct problems, after controlling for other childhood adversities.[26] Links with conduct problems are not however straightforward. The risk for conduct problems does not apply equally to all forms of physical punishment. The E-risk longitudinal twin study was able to compare the effects of corporal punishment (smacking, spanking) versus injurious physical maltreatment using twin-specific reports of both experiences.[27] Results showed that children's genetic endowment accounted for virtually all of the association between their corporal punishment and their conduct problems. This indicated a 'child effect', in which children's bad conduct provokes their parents to use more corporal punishment, rather than the reverse. Findings about injurious physical maltreatment were the opposite. There was no child effect provoking maltreatment and moreover, significant effects of maltreatment on child aggression remained after controlling for any genetic transmission of liability to aggression from antisocial parents.

From risk predictor to causation

Associations have been documented between conduct problems and a wide range of risk factors. A variable is called a 'risk factor' if it has a documented predictive relation with antisocial outcomes, whether or not the association is causal. The causal status of most of these risk factors is unknown; we know what statistically predicts conduct-problem outcomes, but not how or why. Establishing a causal role for a risk factor is by no means straightforward, particularly as it is unethical to experimentally expose healthy children to risk factors to observe whether those factors can generate new conduct problems. There is no one solution to the problem, although the use of genetically sensitive designs and the study of within-individual change in natural experiments and treatment studies have considerable methodological advantages for suggesting causal influences on conduct problems.

Course and prognosis

Of those with early onset conduct disorder (before eight) about half persist with serious problems into adulthood. Of those with adolescent onset, the great majority (over 85 per cent) desist in their antisocial behaviour by their early twenties.

Many of the factors which predict poor outcome are associated with early onset (Table 9.2.5.1).

To detect protective factors, children who do well despite adverse risk factors have been studied.

Table 9.2.5.1 Factors predicting poor outcome

Onset	**Early onset of severe problems, before 8 years of age**
Phenomenology	Antisocial acts which are severe, frequent, and varied
Comorbidity	Hyperactivity and attention problems
Intelligence	Lower IQ
Family history	Parental criminality; parental alcoholism
Parenting	Harsh, inconsistent parenting, with high criticism, low warmth, low involvement, and low supervision
Wider environment	Low-income family in poor neighbourhood with ineffective schools

These so-called 'resilient' children, however, have been shown to have lower levels of risk factors, for example a boy with antisocial behaviour and low IQ living in a rough neighbourhood but living with supportive, concerned parents. Protective factors are mostly the opposite end of the spectrum of the same risk factor, thus good parenting, high IQ are protective. Nonetheless there are factors which are associated with resilience independent of known adverse influences. These include a good relationship with at least one adult, who does not necessarily have to be the parent; a sense of pride and self-esteem; and skills or competencies.

Adult outcome

Studies of groups of children with early onset conduct disorder indicate a wide range of problems not only confined to antisocial acts, as shown in Table 9.2.5.2.

What is clear is that not only are there substantially increased rates of antisocial acts, but that the general psychosocial functioning of children with conduct disorder grown up is strikingly poor. For most of the characteristics shown in Table 9.2.5.2, the increase

Table 9.2.5.2 Adult outcome

Antisocial behaviour	**More violent and non-violent crimes, e.g. mugging, grievous bodily harm, theft, car crimes, fraud**
Psychiatric problems	Increased rates of antisocial personality, alcohol and drug abuse, anxiety, depression and somatic complaints, episodes of deliberate self-harm and completed suicide, time in psychiatric hospitals
Education and training	Poorer examination results, more truancy and early school leaving, fewer vocational qualifications
Work	More unemployment, jobs held for shorter time, jobs low status and income, increased claiming of benefits and welfare
Social network	Few if any significant friends, low involvement with relatives, neighbours, clubs, and organizations
Intimate relationships	Increased rate of short-lived, violent cohabiting relationships; partners often also antisocial
Children	Increased rates of child abuse, conduct problems in offspring, children taken into care
Health	More medical problems, earlier death

compared to controls is at least double for community cases who were never referred, and three to four times for referred children.[18]

Pathways

The path from childhood conduct disorder to poor adult outcome is neither inevitable nor linear. Different sets of influences impinge as the individual grows up and shape the life-course. Many of these can accentuate problems. Thus a toddler with an irritable temperament and short attention span may not learn good social skills if he is raised in a family lacking them, and where he can only get his way by behaving antisocially and grasping for what he needs. At school he may fall in with a deviant crowd of peers, where violence and other antisocial acts are talked up and give him a sense of esteem. His generally poor academic ability and difficult behaviour in class may lead him to truant increasingly, which in turn makes him fall further behind. He may then leave school with no qualifications and fail to find a job, and resort to drugs. To fund his drug habit he may resort to crime, and once convicted, find it even harder to get a job. From this example, it can be seen that adverse experiences do not only arise passively and independently of the young person's behaviour; rather, the behaviour predisposes them to end up in risky and damaging environments. Consequently, the number of adverse life events experienced is greatly increased.[28] The path from early hyperactivity into later conduct disorder is also not inevitable. In the presence of a warm supportive family atmosphere it is far less likely than if the parents are highly critical and hostile.

Other influences can however steer the individual away from and antisocial path. For example, the fascinating follow-up of delinquent boys to age 70 by Laub and Sampson[29] showed that the following led to desistence: being separated from a deviant peer group; marrying to a non-deviant partner; moving away from a poor neighbourhood; military service which imparted skills.

Treatment

Evidence-based treatments

Proven treatments include those which singly or in combination address (i) Parenting skills, (ii) Family functioning, (iii) Child interpersonal skills, (iv) Difficulties at school, (v) Peer group influences, and (vi) Medication for coexistent hyperactivity.

(a) Parenting skills

Parent management training aims to improve parenting skills. There are scores of randomized controlled trials showing that it is effective for children up to about 10 years old.[30] They address the parenting practices identified in research as contributing to conduct problems. A more detailed account is given by Scott.[30] Typically, they include five elements:

(i) Promoting play and a positive relationship

In order to cut into the cycle of defiant behaviour and recriminations, it is important to instil some positive experiences for both sides and begin to mend the relationship. Teaching parents the techniques of how to play in a constructive and non-hostile way with their children helps them recognize their needs and respond sensitively. The children in turn begin to like and respect their parents more, and become more secure in the relationship.

(ii) Praise and rewards for sociable behaviour

Parents are helped to reformulate difficult behaviour in terms of the positive behaviour they wish to see, so that they encourage wanted behaviour rather than criticize unwanted behaviour. For example, instead of shouting at the child not to run, they would praise him whenever he walks quietly; then he will do it more often. Through hundreds of such prosaic daily interactions, child behaviour can be substantially modified. Yet some parents find it hard to praise, and fail to recognize positive behaviour when it happens, with the result that it become less frequent.

(iii) Clear rules and clear commands

Rules need to be explicit and constant; commands need to be firm and brief. Thus shouting at a child to stop being naughty doesn't tell him what he *should* do, whereas for example telling him to play quietly gives a clear instruction which makes compliance easier.

(iv) Consistent and calm consequences for unwanted behaviour

Disobedience and aggression need to be responded to firmly and calmly, but for example putting the child in a room for a few minutes. This method of timeout from positive reinforcement sounds simple but requires considerable skill to administer effectively. More minor annoying behaviours such as whining and shouting often respond to being ignored, but again parents often find this hard to achieve in practice.

(v) Reorganizing the child's day to prevent trouble

There are often trouble spots in the day which will respond to fairly simple measures. For example, putting siblings in different rooms to prevent fights on getting home from school; banning TV in the morning until the child is dressed; and so on.

Treatment can be given individually to the parent and child which enables live feedback in light of the parent's progress and the child's response. Alternatively, group treatments with parents alone have been shown to be equally effective.[31] Trials show that parent management training is effective in reducing child antisocial behaviour the short-term, with moderate to large effect sizes of 0.5 to 0.8 standard deviations, and there is little loss of effect at 1 or 3 year follow-up.[32]

(b) Family functioning

Functional Family Therapy, Multisystemic Therapy, and *Treatment Foster Care* aim to change a range of difficulties which impede effective functioning of teenagers with conduct disorder. Functional family therapy addresses family processes which need to be present such as improved communication between parent and young person, reducing interparental inconsistency, tightening up on supervision and monitoring, and negotiating rules and the sanctions to be applied for breaking them. Functional family therapy has been shown to reduce reoffending rates by around 50 per cent.[33] Other varieties of family therapy have not been subjected to controlled trials for young people with conduct disorder or delinquency, so cannot be evaluated for their efficacy.

In multisystemic therapy,[34] the young person's and family's needs are assessed in their own context at home and in their relations with other systems such as school and peers. Following the assessment, proven methods of intervention are used to address difficulties and promote strengths. Multisystemic therapy differs from most types of family therapy such as the Milan or systemic approach as usually practised in a number of regards. Firstly, treatment is delivered in the situation where the young lives, e.g. at

home. Secondly, the therapist has a low caseload (4–6 families) and the team is available 24 h a day. Thirdly, the therapist is responsible for ensuring appointments are kept and for making change happen—families cannot be blamed for failing to attend or 'not being ready' to change. Fourthly, regular written feedback on progress towards goals from multiple sources is gathered by the therapist and acted upon. Fifthly, there is a manual for the therapeutic approach and adherence is checked weekly by the supervisor. Several randomized controlled trial attest to the effectiveness, with reoffending rates typically cut by half and time spent in psychiatric hospitalization reduced further.[34]

Treatment foster care is another way to improve the quality of encouragement and supervision that teenagers with conduct disorder receive. The young person lives with a foster family specially trained in effective techniques; sometimes it is ordered as an alternative to jail. Outcome studies show useful reductions in reoffending.[35]

(c) Anger management and child interpersonal skills

Most of the programmes to improve child interpersonal skills derive from cognitive behaviour therapy. A typical example is the *Coping Power* Programme.[36] This and other programmes have in common, in training the young person to:

i) slow down impulsive responses to challenging situations by stopping and thinking,

ii) recognize their own level of physiological arousal, and their own emotional state,

iii) recognize and define problems,

iv) develop several alternative responses,

v) choose the best alternative based on anticipation of consequences,

vi) reinforce himself for use of this approach.

Over the longer-term they aim to increase positive social behaviour by teaching the young person to:

i) learn skills to make and sustain friendships,

ii) develop social interaction skills such as turn-taking and sharing,

iii) express viewpoints in appropriate ways and listen to others.

Typically, given alone, treatment gains with interpersonal skills training are good within the treatment setting, but only generalize slightly to 'real-life' situations such as the school playground. However, when they are part of a more comprehensive programme which has those outside the young person reinforcing the approach, they add to outcome gains.[36]

(d) Difficulties at school

These can be divided into learning problems and disruptive behaviour. There are proven programmes to deal with specific learning problems such as specific reading retardation, such as. reading recovery. However, few of the programmes have been specifically evaluated for their ability to improve outcome in children with conduct disorder, although trials are in progress. Preschool education programmes for high risk populations have been shown to reduce arrest rates and improve employment in adulthood (see below).

There are several schemes for improving classroom behaviour, which vary from those which stress improved communication such as 'circle time', and those which work on behavioural principles or are part of a multimodal package. Many of these schemes have been shown to improve classroom behaviour, and some specifically target children with conduct disorder.[37]

(e) Peer group influences

A few interventions have aimed to reduce the bad influence of deviant peers. However, a number attempted this through group work with other conduct disordered youths, but outcome studies showed a *worsening* of antisocial behaviour. Current treatments therefore either see youths individually try to steer them away from deviant peers, or work in small groups (say 3–5 youths) where the therapist can control the content of sessions. Some interventions place youths with conduct disorder in groups with well-functioning youths, and this has led to favourable outcomes.[38]

(f) Medication for coexistent hyperactivity

Where there is comorbid hyperactivity in addition to conduct disorder, several studies attest to a large (effect size of 0.8 standard deviations or greater) reduction in both overt and covert antisocial behaviour,[39] both at home and at school. However, the impact on long-term outcome is unstudied.

Management

Engagement of the family is particularly important for this group of children and families as dropout from treatment is high, at around 30–40 per cent. Practical measures such as assisting with transport, providing childcare, holding sessions in the evening, or at other times to suit the family will all help. Many of the parents of children with conduct disorder may themselves have difficulty with authority and officialdom and be very sensitive to criticism. Therefore the approach is more likely to succeed if it is respectful of their point of view, does not offer overly prescriptive solutions, and does not directly criticize parenting style. Practical homework tasks increase changes, as do problem-solving telephone calls from the therapist between sessions.

Parenting interventions may need to go beyond skill development to address more distal factors which prevent change. For example, drug or alcohol abuse in either parent, maternal depression, and a violent relationship with the partner are all common. Assistance in claiming welfare and benefits and help with financial planning may reduce stress from debts.

A multimodal approach is likely to get larger changes. Therefore involving the school in treatment by visiting and offering strategies for managing the child in class is usually helpful, as is advocating for extra tuition where necessary. If the school seems unable to cope despite extra resources, consideration should be given to moving the child to a different school which specializes in the management of behavioural difficulties. Avoiding antisocial peers and building self-esteem may be helped by getting the child to attend after school clubs and holiday activities.

Where parents are not coping or a damaging abusive relationship is detected, it may be necessary to liaise with the social services department to arrange respite for the parents or a spell of foster care. It is important during this time to work with the family to increase their skills so the child can return to the family. Where there is permanent breakdown, long-term fostering, or adoption may be recommended.

Opportunities for prevention

Conduct disorder should offer good opportunities for prevention since:

1 it can be detected early reasonably well,

2 early intervention is more effective than later,

3 there are a number of effective interventions.

In the United States of America, a number of comprehensive interventions based on up to date empirical findings are being carried out. Perhaps the best known is Families and Schools Together.[40] Here the most antisocial 10 per cent of 5–6 year olds in schools in disadvantaged areas were selected, as judged by teacher and parent reports. They were then offered intervention which was given for a whole year in the first instance and comprised:

i) weekly parent training in groups with videotapes

ii) an interpersonal skills training programme for the whole class

iii) academic tutoring twice a week

iv) home visits from the parent trainer

v) a pairing programme with sociable peers from the class.

Almost 1000 children were randomized to receive this condition or controls, and the project has cost over $50 million. However, so far, preliminary reports of outcome have been limited with no improvement of antisocial behaviour at home on questionnaire measures and modest improvements in the classroom. There are a number of possible reasons for the smaller effects compared to those obtained in trials with clinically referred populations. The motivation of families may be less as they don't perceive they have a problem; starting levels of antisocial behaviour are lower, so there is not so far to go to reach normal levels; and keeping up the quality of the intervention across several sites is harder. It remains to be seen whether longer-term effects will be greater.

In the United States of America, preschool education programmes for disadvantaged children have shown good outcomes in small demonstration projects, but replication on a larger scale has generally proved rather disappointing. In the United Kingdom, the government stressed the importance of helping parents of children in the first 3 years of life and put substantial resources (£540 million) into *SureStart* centres in specifically targeted high risk neighbourhoods to support parenting. Early evaluation of outcome showed no change on 24 of 25 variables; maternal acceptance of the child was the only measured outcome to change, child antisocial behaviour did not.[41] Separate from conduct disorder prevention but related is crime prevention, which can include reducing the opportunities for antisocial behaviour by tighter policing, reducing access to drugs and guns, and so on.

Conclusion

Much is known about the risk factors leading to conduct disorder and effective treatments exist. The challenge is to make these available on a wide scale, and to develop approaches to prevention which are effective and can be put into practice at a community level.

Further information

To access the US Federal Government's site National Youth Violence Prevention Resource Center which has recent research findings, visit http://www.safeyouth.org/scripts/index.asp

To access the US Surgeon Generals' thorough report on youth violence, visit http://www.surgeongeneral.gov/library/youthviolence/

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9.2.6 Anxiety disorders in childhood and adolescence

Daniel S. Pine

Introduction

The term 'fear' refers to the brain state evoked by dangerous stimuli that are avoided because they are capable of harming the organism. The term 'anxiety', in contrast, refers to the brain state evoked by 'threats', stimuli that signal the *possibility* of danger at some point in the near future. Fear and anxiety represent adaptive responses to overt dangers and threats, in that these responses typically reduce the potential for harm to the organisms. Anxiety *disorders* represent conditions where the level of fear is maladaptive either because it leads to clinically significant distress or impairment in function. These effects can result from the production of an anxiety response in a situation not perceived as dangerous by healthy people or by the production of an extreme anxiety response in a situation that healthy people would find mildly anxiety provoking.

The current chapter summarizes recent research on paediatric anxiety disorders. A focus on developmental aspects of anxiety is important since most clinically impairing forms of anxiety typically begin during childhood.[1] Moreover, childhood anxiety disorders show associations with a range of adult psychopathologies beyond anxiety, including most prominently various mood disorders. This fact has stimulated considerable debate concerning the degree to which childhood anxiety disorders reflect early manifestations of adult anxiety disorders. Separation anxiety disorder (SAD) represents the only specific anxiety disorder that primarily occurs in children and adolescents but not adults. Two other disorders frequently co-occur with SAD, social phobia (SOPH), and generalized anxiety disorder (GAD). The current chapter focuses specifically on these three conditions. The chapter also reviews in somewhat less detail data for specific phobia (SPH), a typically minimally impairing condition, and panic disorder (PD), a condition that occurs primarily in adults.[1] Other chapters review material for conditions that frequently co-occur with these five anxiety disorders. This includes major depression (see Chapter 9.2.7), obsessive–compulsive disorder (Chapter 9.2.8), and trauma-related disorders (Chapters 9.3.2). Material on SAD, SOPH, GAD, SPH,

and PD are reviewed in three sections. The first, most detailed, section reviews clinical features of these disorders, including typical presentations and diagnosis. The second somewhat briefer section reviews pathophysiology, and the final section briefly reviews therapeutics.

Clinical features

Clinical presentation

Children presenting with symptoms of anxiety typically manifest signs of various disorders. In fact, in the clinical setting, presentation with a 'pure' form of anxiety is relatively rare. This suggests that current classifications group children into categories that are unlikely to represent distinct pathophysiologies. Nevertheless, while the current nosology is likely to change as understandings of pathophysiology advance, current classification schemes remain quite useful in that they facilitate communication among individuals working with a child and allow clinicians to draw on research in therapeutics using a common diagnostic system. The current section describes clinical presentation of five specific anxiety disorders, as defined in the fourth edition of the *Diagnostic and Statistical Manual* (DSM-IV). These definitions are similar to those used in the 10th edition of the *International Classification of Disease* (ICD-10), although ICD-10 provides single diagnosis for children with multiple anxiety disorders, whereas and DSM-IV provides multiple diagnoses.

(a) Separation anxiety disorder

The key feature of SAD involves presentation of anxiety related to fear that harm will befall an attachment figure. In severe forms, SAD typically presents with avoidance of situations, such as school, where separation is required. The term 'school phobia' had been used on occasion for these presentations, but current approaches no longer use this term. Symptoms of SAD often are severe at night, leading many children to refuse to sleep alone or at friends' homes. Considerable research examines the relationship between childhood SAD and adult panic disorder.

(b) Social phobia

The key features of SOPH involve intense fear or anxiety in situations where the individual is scrutinized. This presents either as extreme form of pervasive shyness or as extreme fear in particular social situations, such as during class presentations. SOPH can also be classified as a 'generalized subtype', indicating that most social situations are feared. The condition can markedly interfere with function by leading children to avoid important academic exercises that must be performed in social settings or by markedly impacting on social development. This effect on social relationships has led to some controversy concerning the boundaries between SOPH and pervasive developmental disorders (PDD). Classically, this distinction can be made based on the presence of language dysfunction and stereotypic behaviour in PDD but not SOPH. Considerable research examines the relationship between late-childhood SOPH and early-childhood temperament.

(c) Generalized anxiety disorder

The key feature of GAD involves a pervasive sense of worry about various events or circumstances. For example, children with GAD frequently worry about their competence, as might manifest on school or athletic performances. These worries are associated with other symptoms, such as muscle tension or other somatic complaints, irritability, and trouble sleeping. Because children with SAD and SOPH also present with worry, clinicians face difficulties when attempting to determine if worries reflect aspects of these disorders or another problem. GAD is diagnosed only when worries cannot be accounted for by another diagnosis. Considerable research examines the relationship between GAD and major depressive disorder (MDD).

(d) Specific phobia

The key feature of SPH involves fear of a specific stimulus or object. SPH can manifest to a range of objects, such as potentially dangerous animals or natural scenarios, and SPH can be categorized into one of five types, based on the content of the fear. Children rarely present for clinical care when they suffer from SPH in the absence of another anxiety disorder, despite the fact that SPH does present relatively commonly in pure forms in the community. This suggests that SPH typically is associated with relatively mild degrees of distress and impairment, unless SPH is associated with another anxiety disorder.

(e) Panic disorder

The key feature of PD involves spontaneous panic attacks. The term 'panic attack' refers to crescendo paroxysms of severe anxiety that occur suddenly and are associated with somatic and cognitive sensations, such as rapid heart beat, shortness of breath, and a strong desire to flee. Panic attacks occur in many situations and with various clinical syndromes. The key feature of PD is that at least some of these attacks occur in the absence of any cue or trigger. As such, the patient cannot attribute the attack to fear of any specific circumstance. PD virtually never occurs prior to puberty, and the disorder is also very rare before adulthood.

Assessment

The assessment for anxiety involves input from multiple sources. Clearly obtaining information directly from the patient is vital. Children with anxiety disorders may be reluctant to report the precise nature of their fears. As a result, adults may be unaware of vital symptoms. On the other hand, children also often show reluctance to acknowledge their anxiety, either because they are unaware of their degree of incapacitation or because they are highly embarrassed about their symptoms. In this instance, adults provide vital information concerning specific objects or situations that might be feared by children or adolescents.

Various forms of standardized assessment are available for paediatric anxiety.[2] This includes rating scales that can be directly completed by parents, teachers, or children, as well as scales that are completed by clinicians based on their interview of the child and parent. Moreover, standardized observational batteries typically are used for the assessment of temperament, in very young children, that relate to anxiety disorders in older children. Temperament also can be measured by parent or self-report.[3] In general, while high scores on various rating scales does provide some indication regarding the presence of an anxiety disorder, structured psychiatric interviews, completed by a trained clinician, represents the gold standard for arriving at a diagnosis.

Prevalence and demographics

As a group, paediatric anxiety disorders probably represent the most common form of developmental psychopathology. It is

difficult to provide precise data concerning their overall prevalence, as the rate of anxiety disorders is highly variable across studies, most likely due to variations in assessment. Rates of anxiety disorders are unusually sensitive to even subtle changes in assessments of impairment.[4] In general, overall lifetime rates of paediatric anxiety probably fall in the 10–20 per cent range.[5] Rates of individual disorders vary with age. Thus, SAD represents the most common condition, with prevalence typically in the 5 per cent range, before puberty, whereas GAD and SOPH become more prevalent during adolescence, again with rates in the 5 per cent range. Rates of SPH are highly variable, depending on the stringency of impairment criteria, with some estimates surpassing 20 per cent. As noted above, PD is very rare before late adolescence.

In terms of demography, anxiety disorders show a strong female predominance. This gender difference manifests for all of the conditions examined here, and, unlike data for MDD, it emerges before puberty. While the overall rate of anxiety disorders changes relatively little from childhood to adolescence, the nature of disorders does change. Thus, SAD is most common in young children, whereas SOPH is most common in adolescence. Data concerning associations with social class appear somewhat mixed. While some inconsistent reports note higher rates among individuals in the relatively lower social strata, the data appear most consistent for SPH, with weaker or absent associations in other conditions.[1] Consistent with weak relationships, recent work suggests that abrupt changes in family economics do not lead to changes in rates of anxiety disorders, despite strong associations with changing rates of other disorders.[6]

Comorbidity

Data concerning comorbidity reveal distinct trends in the clinic relative to the community, most likely due to the effects of referral biases on data from the clinic. Thus, in the clinic, paediatric anxiety disorders have been linked to virtually every form of psychopathology. This includes mood disorders, behaviour disorders, attention deficit hyperactivity disorder, and substance use disorders. In the community, however, associations appear particularly strong with a more restricted group of conditions. The most common comorbidity represents associations with other anxiety disorders, with odds ratios typically appearing in the three-to-five range.[1] Associations between SOPH and GAD appear particularly strong in this work. Comorbidity with mood disorder, particularly MDD, is only slightly weaker than comorbidity among the anxiety disorders.[7] Other forms of psychopathology show far weaker associations.

Clinical course

Paediatric anxiety disorders predict an increased risk for a range of adverse psychiatric outcomes in adults. This includes most prominently risk for adult anxiety disorders and MDD.[1,8] In general, children and adolescent with one or another anxiety disorder face a two- to five-fold increased risk for adult anxiety or MDD. These relationships reflect the fact that most adults with various forms of mood or anxiety disorder show the initial signs of their problem during childhood or adolescence, manifest as a paediatric anxiety disorder. However, the overall magnitude of these longitudinal relationships between paediatric anxiety and any form of adult psychopathology appears somewhat weaker than longitudinal relationships for other developmental psychopathologies, such as the behaviour disorders.[9]

Relatively few studies consider the long-term outcome of the specific paediatric anxiety disorders. In the few studies that do examine this issue, the overall weight of the evidence suggests that risk for poor outcome is similar among all paediatric anxiety disorders.[5] However, some inconsistent data do note specific associations among individual child and adult disorders. For example, some evidence documents a particularly strong association between paediatric GAD and adult MDD,[1] though studies following adolescents into their 30s suggest a comparable risk for MDD in adolescent SOPH.[8] Similarly, some studies note an association between childhood SAD and adult PD, but the overall weight of the evidence does not provide strong support for this link.[10] Finally, some inconsistent evidence also suggests that the outcome of paediatric SPH appears relatively good, as compared with other anxiety disorders.

Pathophysiology

Neuroscience and fear

Work on the pathophysiology of anxiety disorders benefits from a wealth of research examining brain regions involved in fear and anxiety among rodents and non-human primates.[11] Data also document strong cross-species parallels in the effects of threats and danger on behaviour, physiology, and information processing. This suggests that neural circuits implicated in fear and anxiety among rodents or non-human primates are likely involved in fear and anxiety among humans.

Basic science work delineates a distributed neural circuit engaged by various forms of dangerous or threatening stimuli. This includes stimuli recognized as dangerous through learning, as classically studied in the 'fear conditioning' paradigm, whereby a neutral conditioned stimulus is paired with an aversive unconditioned stimulus.[12] This also includes stimuli innately recognized as dangerous, even in the absence of prior training.[13] Finally, work in immature rodents and non-human primates demonstrates strong developmental influences on the neural circuitry associated with both learned and innate fears.[11] Specifically, in immature relative to mature organisms, both genetic and environmental manipulations show the capacity to exert more robust, long-standing effects on anxiety and fear-related behaviours as well as the underlying neural circuitry mediating these behaviours.

Figure 9.2.6.1 illustrates the core components of the underlying circuitry associated with fear and anxiety. The amygdala represents a hub in the fear circuit. As shown in Fig. 9.2.6.1, this collection of nuclei lies within the medial temporal lobe, where it receives input from various sensory cortices as well as brain-stem monoamine systems, and where it sends output to the hypothalamus and other structures that orchestrate the organism's response to danger. While some debate continues concerning the precise role played by the amygdala in fear, the structure has been implicated in both learned and innate fears as well as various positive-valence emotions. Some data suggest that the amygdala plays a vital role in regulating attention when organisms learn to associate neutral stimuli with salient events.

Fear and anxiety represent complex states that reflect influences from other brain regions beyond the amygdala. Figure 9.2.6.1 shows the location of two particularly important regions. Thus, the hippocampus also plays a role in fear and anxiety, with data most clearly implicating this brain region in the representation of spatial

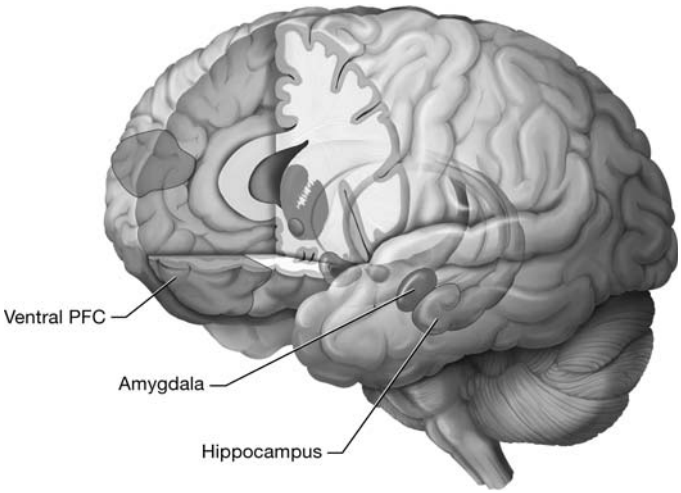

Fig. 9.2.6.1 Displays key anatomical components of brain circuitry engaged when various organisms encounter threats or dangers. Functional aspects of this circuitry show strong cross-species conservation, and Figure 9.2.6.1 depicts the location in the human brain of three particularly important neural structures: the ventral prefrontal cortex (PFC), amygdala, and hippocampus.

contexts associated with threat. As shown in Fig. 9.2.6.1, the hippocampus lies posterior to the amygdala in the medial temporal lobe. Various components of the prefrontal cortex (PFC) also are involved in fear and anxiety. As shown in Fig. 9.2.6.1, particularly strong associations occur with ventral PFC, including both lateral and medial expanses. PFC is thought to play a regulatory role for fear and anxiety responses, serving to delineate the temporal context where fear and anxiety are either necessary or not appropriate, given the organism's goals.

Familial aggregation and genetics

Most complex behaviours, including fear and anxiety, represent phenomena that result from influences of both genes and the environment. The associations of genes and the environment with anxiety can be examined directly with behavioural indicators of anxiety or psychiatric diagnosis. Alternatively, these associations can be examined with constructs beyond symptoms that show closer relationships to brain function. The term 'endophenotype' has been used to describe such underlying constructs linked to both disorders and their underlying risks.[14]

Family studies consistently demonstrate strong associations among various anxiety disorders in parents and anxiety disorders in their children. More than 10 studies show that children born to parents with PD, SOPH, or GAD face a two- to four-fold increased risk for anxiety disorders.[5] As with data from longitudinal studies, some non-specificity emerges in other family studies. Children born to parents with MDD even in the absence of anxiety face the same elevated risk for paediatric anxiety as children born to parents with anxiety disorders. These data are consistent with a wealth of data among adults documenting strong familial associations.[15]

These findings on familial aggregation might reflect the effects of either genes or the environment. Data from twin and adoption studies suggest that genes account for approximately 40 per cent of the risk for anxiety both among children and adults.[5,15] Much like for longitudinal and family aggregation studies, twin studies suggest pathophysiologic similarities in the genetics of anxiety and depression. In particular, GAD and MDD appear to share many of the same genes.[16] In terms of specific genes, the field has only begun to examine associations with specific paediatric anxiety disorders. While considerable enthusiasm pertains to research on serotonin-related genes, this enthusiasm emerges predominantly from studies in adults.[11]

The effects of genes on risk for paediatric anxiety are not thought to moderate overt symptomatic expressions. Rather, genes, either as main effects or through interactions with the environment, are hypothesized to produce disruptions in the underlying function of the neural circuit illustrated in Fig. 9.2.6.1. These disruptions are expected to produce perturbations in physiologic regulation and information processing functions, examples of endophenotypes for paediatric anxiety disorders. Work on endophenotypes in paediatric anxiety disorders generally focus on three related profiles.

First, considerable work examines variations in children's temperaments, as they relate to both parental histories of anxiety as well as children's risk for anxiety, manifest later in life. This work shows that children who react with fear and hesitation in novel social scenarios face a high risk for anxiety.[3] This temperamental classification is known as 'behavioural inhibition'. Some evidence suggests that these associations pertain particularly strongly to the association with later-life SOPH.

Second, other work examines associations with variations in physiologic or cognitive responses to various threats. While various forms of fear and anxiety produce robust changes in autonomic physiology, inconsistent data document strong associations between individual differences in anxiety among humans and the magnitude of these physiological responses. Particularly interest focuses on between-group differences in conditioned physiologic reactions, but findings in this area appear weak and inconsistent.[17] Some of the strongest findings emerge from research on the startle response. This defensive reflex shows strong cross-species similarities in the degree to which it can be modulated by the presence of a threat. In general, adult anxiety disorders are characterized by enhanced startle in some contexts, though data in paediatric anxiety disorders appear inconsistent. Moreover, children born to parents with either PD or MDD also show enhanced startle responses under some circumstances.[18,19] Other work focuses on biases in various cognitive processes, such as attention and memory, where associations with specific disorders generally appear stronger than for studies of physiology. Here some work suggests that perturbations in face processing predict both risk for anxiety and the presence specifically of paediatric SOPH.[20]

Finally, perturbations in respiration have been linked most convincingly to the diagnosis of PD.[21] Data among adults show these respiratory perturbations aggregate within families. Moreover, findings in children and adolescents show that respiratory perturbation is associated with SAD but not other paediatric anxiety disorders, such as SOPH. Given evidence of familial aggregation between parental PD and childhood SAD, these data suggest that respiratory perturbation may confer a familial risk for panic disorder. Nevertheless, data examining respiratory function in offspring of PD patients are not consistent with this possibility.[22]

Stress

Work in animal models demonstrates strong relationships between exposure to various forms of physical or emotional stress

and individual differences in anxiety or fear.[11] These associations appear particularly strong in juvenile organisms. For studies among children and adolescents, strong associations also emerge with various measures of stress including either stress that occurs within the family or in other social contexts.[23] Nevertheless, it remains unclear the degree to which these association reflect specific connections with anxiety, given that stress is associated with a range of other psychopathologies besides anxiety.[24] Moreover, considerable heterogeneity exists in terms of the relationship between stress and anxiety, given that some individuals exposed to extreme stress are resilient, whereas other individuals exposed to mild stress develop anxiety. At least some of these individual differences are though to reflect the influences of genes on underlying neural circuitry, such that the development of paediatric anxiety reflects influences of gene–environment interactions.[11]

Brain imaging

Through studies of brain imaging, it is now possible to examine associations between paediatric anxiety disorders and perturbations in brain structure or function. Relatively few brain-imaging studies have examined paediatric anxiety disorders, and the few studies that do focus on neural structures depicted in Fig. 9.2.6.1.

Without question, the amygdala stands as the most frequently investigated brain structure in paediatric anxiety. Two studies examine amygdala morphometry in paediatric anxiety disorders, with one of these studies reporting reduced volume and the other reporting enlarged volume, both focusing mostly on children with GAD.[25,26] Such inconsistencies are consistent with mophometry studies in adults, where inconsistent evidence of amygdala enlargement or reduction emerges across a range of studies. Two other studies use functional magnetic resonance imaging (fMRI) to examine amygdala function in paediatric anxiety disorders.[27,28] Here, the findings are more consistent, much like in a larger series of studies in adults. Specifically, both studies reported enhanced amygdala activation in paediatric GAD, consistent with data in adult SOPH, MDD, as well as post-traumatic distress disorder or behavioural inhibition. Finally, imaging work focused on other structures documents abnormalities with less consistency. This includes structural and functional studies of the PFC and hippocampus.[5]

Therapeutics

A range of approaches has been suggested as useful in the treatment of paediatric anxiety disorders. The current chapter restricts considerations to a review of modalities studied with the randomized controlled trial (RCT). Two modalities have been studied in sufficient detail to provide conclusions on efficacy: cognitive behavioural psychotherapy treatment (CBT) and selective serotonin reuptake inhibitors (SSRIs). The data for these two treatments are reviewed in most detail, whereas other treatments are mentioned only briefly.

CBT relies on the principles of extinction, whereby an individual with an anxiety disorder undergoes exposure to a feared object or situation while relying on cognitive techniques taught as part of the therapy. In general, CBT is easiest when a child presents with a relatively specific set of fears and worries that allow the therapist to work with the child to create a fear hierarchy. The child then gradually undergoes exposure to situations on this hierarchy that are increasingly anxiety provoking. More than 10 studies use an RCT design to examine the efficacy of CBT, and the overwhelming majority of these document strong efficacy.[5] Nevertheless, most of these studies compare CBT to a wait-list control condition, a condition that may actually be aversive. The few RCTs of CBT using more suitable control conditions generally find weaker advantages for CBT. These studies suggest that CBT is a viable treatment option for any of the anxiety disorders considered in this chapter.

Five RCTs examine the efficacy of one or another SSRI in paediatric anxiety disorders, all using placebo control.[5] As with the data for CBT, this work provides strong justification for using SSRIs, in that robust treatment effects emerge. Moreover, these studies rely on placebo, a more credible control than in the CBT studies relying on wait-list comparison. Thus, the strength of the evidence supporting efficacy is probably somewhat greater in SSRIs than it is for CBT. Nevertheless, serious concerns about the safety of SSRIs emerged in 2002, due to the suggestion that SSRIs were associated with an increased risk over placebo for suicidal thoughts or behaviour. This ultimately led the Food and Drug Administration to place a 'black box' warning on the use of SSRIs in children, a warning recently extended to adults aged 25 and younger. Given these concerns, CBT probably represents the most reasonable first-line treatment for paediatric anxiety disorders. Among children who either cannot complete a course of CBT or who fail to respond to CBT, SSRIs represent an eminently reasonable treatment.

A range of other treatments have been considered for paediatric anxiety disorders. These include both psychotherapies, such as dynamically oriented therapy, and medications, such as various non-SSRI antidepressants or benzodiazepines. Due to either the dearth of data on efficacy or concerns with safety, all of these treatments should be considered third-line options, after CBT and SSRIs.

Conclusions

The current chapter reviews data for paediatric anxiety disorders in three sections. The longest section reviews clinical characteristics of these disorders. This section describes the clinical features, demography, and outcome of paediatric anxiety disorders, the most common class of mental syndrome afflicting children and adolescence. The first section is followed by a shorter section focused on pathophysiology. Here, data on neural circuits implicated in anxiety are reviewed most comprehensively. Finally, treatment is briefly reviewed in the third section. More detailed considerations of therapeutics for a range of paediatric psychiatric disorders can be found in section 9.5. This includes a discussion of both psychotherapies and medication.

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9.2.7 Paediatric mood disorders

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In this chapter, we describe the nosology and epidemiology of paediatric unipolar and bipolar disorders, risk factors and predictors of course, and the evidence base for pharmacological and psychosocial treatments. We conclude this chapter by suggesting areas for future research.

Clinical picture

Mood disorders may be classified on three dimensions: (a) severity; (b) course; and (c) presence or absence of mania/hypomania.[1] Depressed children and adolescents may not describe their mood as sad, but instead as, 'grouchy', 'bored', 'having no fun', or 'empty'.[2] The most severe depressive condition is major depression, which requires at least 2 weeks of a depressed, sad, bored, or anhedonic mood for most of the time, and four additional depressive symptoms involving impairment in concentration, suicidal thoughts, difficulty making decisions, impaired sleep and appetite, guilt, and a decreased sense of self-worth (Box 9.2.7.1). Patients with depressive symptoms, but whose clinical picture is below the threshold for major depression (so-called minor depression or depression NOS) can still show significant impairment.[3] Dysthymic disorder is more chronic and intermittent than major depression, with periods of depression interspersed with normal mood, but with duration of at least 1 year (Box 9.2.7.2). Adjustment disorder with depressed

Box 9.2.7.1 Criteria for the diagnosis of a major depressive episode

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

   Note: Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

   1 Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g. feels sad or empty) or observation made by others (e.g. appears tearful). *Note*: In children and adolescents, can be irritable mood.

   2 Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others).

   3 Significant weight loss when not dieting or weight gain (e.g. a change of more than 5 per cent of body weight in a month), or decrease or increase in appetite nearly every day. *Note*: In children, consider failure to make expected weight gains.

   4 Insomnia or hypersomnia nearly every day.

   5 Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).

   6 Fatigue or loss of energy nearly every day.

   7 Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional nearly every day (not merely self-reproach or guilt about being sick).

   8 Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).

   9 Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms do not meet criteria for a Mixed Episode (see p. 365).[1]

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication (or a general medical condition (e.g. hypothyroidism).

E. The symptoms are not better accounted for by bereavement, i.e. after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

(Modified from APA (2000), *Diagnostic and statistical manual of mental disorders* (4th edn), American Psychiatric Association Press, Washington, DC.)

Box 9.2.7.2 Criteria for the diagnosis of dysthymic disorder

A. Depressed mood for most of the day, for more days than not, as indicated either by subjective account or observation by others, for at least 2 years. *Note*: In children and adolescents, mood can be irritable and duration must be at least 1 year.

B. Presence, while depressed of two (or more) of the following:

   1 poor appetite or overeating

   2 insomnia or hypersomnia

   3 low energy or fatigue

   4 low self-esteem

   5 poor concentration or difficulty making decisions

   6 feelings of hopelessness

C. During the 2-year period (1 year for children or adolescents) of the disturbance, the person has never been without the symptoms in Criteria A and B for more than 2 months at a time.

D. No Major Depressive Episode (see p. 356)[1] has been present during the first 2 years of the disturbance (1 year for children and adolescents); that is the disturbance is not better accounted for by chronic Major Depressive Disorder, or Major Depressive Disorder, In Partial Remission. *Note*: There may have been a previous Major Depressive Episode provided there was a full remission (no significant signs or symptoms for 2 months) before development of the dysthymic disorder. In addition, after the initial 2 years (1 year in children or adolescents) of dysthymic disorder, there may be superimposed episodes of Major Depressive Disorder, in which case both diagnoses may be given when the criteria are met for a Major Depressive Episode.

E. There has never been a Manic Episode (see p. 362),[1] a Mixed Episode (see p. 365),[1] or a Hypomanic Episode (see p. 368),[1] and criteria have never been met for Cyclothymic Disorder.

F. The disturbance does not occur exclusively during the course of a chronic Psychotic Disorder, such as Schizophrenia or Delusional Disorder.

G. The symptoms are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition (e.g. hypothyroidism).

H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

(Modified from APA (2000), *Diagnostic and statistical manual of mental disorders* (4th edn), American Psychiatric Association Press, Washington, DC.)

mood is a milder and self-limited disturbance of mood that follows a significant life stressor (Box 9.2.7.3).

The presence of clinically significant manic or hypomanic symptomatology suggests bipolar spectrum disorder. The symptomatology of mania can be thought of as the mirror image of depression, with mood characterized by elation or grandiosity. Mania is associated

Box 9.2.7.3 Criteria for the diagnosis of adjustment disorder with depressed mood

A. The development of emotional or behavioural symptoms in response to an identifiable stressor(s) occurring within 3 months of the onset of the stressor(s).

B. These symptoms or behaviours are clinically significant as evidenced by either of the following:

1 marked distress that is in excess of what would be expected from exposure to the stressor

2 significant impairment in social or occupational (academic) functioning

C. The stress-related disturbance does not meet the criteria for another specific Axis I disorder and is not merely an exacerbation of a pre-existing Axis I or Axis II disorder.

D. The symptoms do not represent bereavement.

E. Once the stressor (or its consequences) has terminated, the symptoms do not persist for more than an additional 6 months.

(Modified from APA (2000), *Diagnostic and statistical manual of mental disorders* (4th edn), American Psychiatric Association Press, Washington, DC.)

with clear impairment, whereas hypomania, while associated with a change in functioning, does not always result in impairment per se. Bipolar individuals, especially in the paediatric age group, frequently do not show the classic distinct alternating manic and depressive periods found in adult bipolar patients. Instead they may either experience depression and manic symptoms simultaneously, so-called mixed episodes, or alternations of mania and depression that may occur within a month, a week, or even a day, e.g. rapid cycling.[4] Common symptoms of paediatric bipolar disorder are pressure of speech, increased energy, and decreased need for sleep. Risk-taking behaviour showing poor judgement (e.g. gambling, hypersexuality, excessive spending) and joking and excessive humour are very specific for paediatric bipolar disorder, but less common in paediatric samples. While irritability is a common symptom of paediatric bipolar disorder, it is very non-specific and is commonly found in many other conditions, such as depression, oppositional defiant disorder, and attention deficit disorder. The DSM-IV requires a relatively long duration of mania (7 days) and hypomania (4 days) in order to meet criteria. Many paediatric patients may show the same symptom pattern but have very rapid cycling and therefore, do not meet these criteria. If altered function is present, such patients should receive a diagnosis of Bipolar Disorder NOS. Bipolar NOS is a common diagnosis for children with manic symptoms because very often, paediatric bipolar illness does not fulfil the duration criteria for mania, in part due to the frequency of rapid cycling conform to the classic adult patterns of distinct patterns of mania and depression.[4] However, in children and adolescents, Bipolar disorder NOS does not appear to be different from Bipolar I or II with regard to impairment, rate of comorbid disorders, response to treatment, or family history of bipolar disorder, and many patients with BP-NOS upon longitudinal follow-up go on to develop BP-I or BP-II disorders.[4] Individuals who have had a history of full mania plus major depression receive a diagnosis of Bipolar I disorder, those with hypomania plus major depression receive a diagnosis of Bipolar II disorder, and those with hypomania and dysthymia receive a diagnosis of cyclothymic disorder (see Boxes 9.2.7.4 and 9.2.7.5).

While some in the field continue to raise questions about the validity of the diagnosis of paediatric bipolar disorder, the convergent evidence from longitudinal and high-risk studies is that there it is the essentially an earlier manifestation of the same illness as is found in adults, is highly familial, and shows a chronic and consistent course.[4,5]

Differential diagnosis

Attention deficit hyperactive disorder (ADHD) and disruptive disorders

Patients with ADHD, oppositional disorder, and conduct disorder are often irritable, show a low frustration tolerance, and can become demoralized due to school failure and peer rejection. However, in the absence of true depression, their mood will be restored as soon as the source of their frustration has been remedied. While both ADHD and depression are associated with poor concentration, the age of onset of ADHD is usually earlier than in mood disorders. Patients with ADHD have other accompanying difficulties such as hyperactivity and impulsivity that are part of the depressive picture. Conversely, depressed patients will show changes in sleep, energy level, appetite, mood, and self-worth that are not part of the picture of ADHD. The symptoms of ADHD, such as poor concentration, hyperactivity, and impulsivity can also be seen in bipolar disorder but patients with ADHD rarely have concomitant hypersexuality, grandiosity, and decreased need for sleep.[4,5] However, hypersexuality may also be seen in victims of sexual abuse, but in contrast with the hypersexuality of bipolar disorder, is not accompanied by clinically significant grandiosity, pressure of speech, increased energy, and diminished need for sleep. Often, the diagnostic difficulty is not simply distinguishing between disruptive and mood disorders, but in the proper attribution of shared symptoms in patients with comorbidity, as is very often the case. When patients have both mood disorder and ADHD, usually the ADHD antedates the mood disorder. A diagnosis of a mood disorder can only be made when the shared symptoms, such as impaired concentration become worse in association with depressed or manic mood.

Anxiety disorders

Patients with anxiety disorder may also become quite dysphoric, but when the anxiogenic situation is removed, normal mood frequently ensues. Anxiety is a frequent antecedent of paediatric depression and bipolar disorder.[5,6] Symptoms that are shared between disorders, such as difficulty with sleep, or impaired concentration, are attributed to the mood disorder only if they become worse with the onset of a depressed or manic mood state. Panic disorder is often comorbid with paediatric bipolar disorder.[5] However, it is important to distinguish between the symptoms of panic disorder, that are prominently somatic and associated with thoughts and feelings of dread, and rapid cycling and a mixed state, which are marked with mood instability and the presence of simultaneous, or rapidly alternating depressive and manic symptoms.

Box 9.2.7.4 Criteria for the diagnosis of bipolar disorder

A. Currently (or most recently) in a Manic Episode (see p. 362).[1]

B. There has previously been at least one Major Depressive Episode (see p. 356),[1] Manic Episode (see p. 362),[1] or Mixed Episode (see p. 365).[1]

C. The mood episodes in Criteria A and B are not better accounted for by Schizoaffective Disorder and are not superimposed on Schizophrenia, Schizophreniform Disorder, Delusional Disorder, or Psychotic Disorder Not Otherwise Specified.

Past or current history of a Manic Episode is characterized by:

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least 1 week (or any duration if hospitalization is necessary).

B. During the period of mood disturbance, three (or more of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:

1 inflated self-esteem or grandiosity

2 decreased need for sleep (e.g. feels rested after only 3 h of sleep)

3 more talkative than usual or pressure to keep talking

4 flight of ideas or subjective experience that thoughts are racing

5 distractibility (i.e. attention to easily drawn to unimportant or irrelevant external stimuli)

6 increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation

7 excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g. engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments)

C. The symptoms do not meet criteria for a Mixed Episode (see p. 365).[1]

D. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

E. The symptoms are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication, or other treatment) or a general medical condition (e.g. hyperthyroidism).

Note: Manic-like episodes that are clearly caused by somatic antidepressant treatment (e.g. medication, electroconvulsive therapy, light therapy, should not count toward a diagnosis of Bipolar I disorder.

(Modified from APA (2000), *Diagnostic and statistical manual of mental disorders* (4th edn), American Psychiatric Association Press, Washington, DC.)

Box 9.2.7.5 Criteria for the diagnosis of cyclothymic disorder

A. For at least 2 years, the presence of numerous periods with hypomanic symptoms (see p. 368)[1] and numerous periods with depressive symptoms that do not meet criteria for a Major Depressive Episode. *Note*: In children and adolescents, the duration must be at least 1 year.

B. During the above 2-year period (1 year in children and adolescents), the person has not been without the symptoms in Criterion A for more than 2 months at a time.

C. No Major Depressive Episode (p. 356),[1] Manic Episode (p. 362),[1] or Mixed Episode (see p. 365)[1] has been present during the first 2 years of the disturbance.

Note: After the initial 2 years (1 year in children and adolescents) of Cyclothymic Disorder, there may be superimposed Manic or Mixed Episodes (in which case both Bipolar I disorder and Cyclothymic Disorder may be diagnosed) or Major Depressive Episodes (in which case both Bipolar II disorder and Cyclothymic Disorder may be diagnosed).

D. The symptoms in Criterion A are not better accounted for by Schizoaffective Disorder and are not superimposed on Schizophrenia, Schizophreniform Disorder, Delusional Disorder, or Psychotic Disorder Not Otherwise Specified.

E. The symptoms are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition (e.g. hyperthyroidism).

F. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

(Modified from APA (2000), *Diagnostic and statistical manual of mental disorders* (4th edn), American Psychiatric Association Press, Washington, DC.)

Substance abuse

The use of marijuana, alcohol, or opiates can mimic the symptoms of depression, such as difficulty with concentration, motivation, low energy, and dysphoria. Amphetamine and cocaine abuse can mimic mania. Depressed and bipolar patients are at greatly increased risk of abusing substances, so that the presence of substance abuse does not rule out a mood disorder or vice versa, but in fact, should raise the suspicion of possible comorbidity.

Eating disorder

Patients with a restricting eating disorder who are nutritionally compromised may show symptoms that overlap with depression, including decreased appetite, low energy, and sad mood. Often the sadness is found in patients with anorexia who are being forced to gain or maintain weight against their will. A diagnosis of depression, unless there is a clear historical precedent that antedates the eating disorder, should only be made when the nutritional status of the patient has been normalized. Bulimic disordered patients often have difficulties with impulse control that need to be differentiated from bipolar disorder.

Borderline personality disorder

Although there is evidence that borderline personality disorder can be reliably diagnosed in adolescents,[7] diagnostic convention requires that this diagnosis only be applied for adults. Still, there is general agreement that many adolescents, particularly those with mood disorders, have 'borderline features', such as mood lability, impulsivity, suicidal thoughts and behaviour, chaotic interpersonal relationships, and risky behaviour that has a high likelihood of resulting in personal harm. Others have argued that borderline personality disorder is really a form of bipolar spectrum disorder, although family studies have not confirmed this.[5] Instead, the high degree of overlap between personality disorder and bipolar disorder suggests that care be taken in not attributing symptoms that more appropriately are associated with a lifelong personality style to bipolar disorder. Conversely, in the presence of a clear and unremitting paediatric mood disorder, personality disorder should not be diagnosed.

Psychosis

Although rare in childhood, incipient schizophrenia can present with sad and detached mood, sleep disturbance, and social withdrawal. Psychotic symptoms that evolve in schizophrenia are more likely to be mood-incongruent. In contrast, psychosis in depression and bipolar disorder is more often, but not always, mood-congruent.[2,5] This is a diagnosis that often can only be made upon careful longitudinal follow-up. Since psychosis is often seen in youth with mood disorders, and schizophrenia is rare at this age group, any child or adolescents with psychosis needs to be carefully assessed for the presence of a mood disorder, particularly bipolar illness.

Comorbidity

Comorbidity is the rule, rather than the exception.[8] Anxiety disorder frequently antedates paediatric depression and bipolar disorder, with common precursors being social phobia and panic disorder, respectively. ADHD is frequently comorbid with both conditions. Substance abuse is often a complication of mood disorder, although this condition in turn lengthens episodes and increases the risk for recurrence.

Medical comorbidity

Medications used to treat epilepsy, inflammatory bowel disease, and rheumatic and allergic disease can have profound effects on mood. Corticosteroids can induce depression or mania. Phenobarbital is associated with depression, as is use of interferon.[9] Moreover, systemic aspects of the diseases themselves may increase the risk for depression, in epilepsy, asthma, diabetes, and thyroid illness. Oral contraceptives can also result in mood changes.

Descriptive epidemiology

The point prevalence of major depression in around 1–2 per cent in prepubertal samples, and between 3–8 per cent in adolescent samples.[10] The prevalence of bipolar disorder is around 1 per cent in paediatric populations, although the rate of 'soft' bipolar disorder, which has some, but not all of the core features of bipolar disorder, has been reported to be as high as 5 per cent in some adolescent samples.[11] The male to female ratio is around 1:1 for prepubertal depression, but increases to around 1:3 for depression after puberty. In contrast, the males and females have similar risk for bipolar disorder, regardless of pubertal status. The increased rate of depression after puberty is accounted for almost entirely by the increased risk in females, and may be related to changes in estradiol and testosterone associated with puberty.[12] Prepubertal major depression is an admixture of two subtypes: one is highly familial, with a high risk for recurrence and for eventual paediatric mania, and the second with comorbid with disruptive disorders, a low risk for depressive recurrence, an association with parental criminality, substance abuse, and family discord, and a course more similar to conduct disorder than to mood disorder.[13] A clear clinical syndrome of depression has been reported in children as young as aged 3, particularly in those young children with a family history of mood disorders.[14]

Course

Paediatric mood disorders tend to be both chronic and recurrent. While prepubertal depression comorbid with conduct disorder is likely not to be recurrent, studies of child-onset depression with a family history of depression show high rates of recurrence, with risks of recurrence of 40 per cent in 2 years, and over 70 per cent within 5 years.[2] The average length of a depressive episode is around 4–6 months in community samples, and 6–8 months in clinical samples.[15] The duration of dysthymic disorder is much longer, on average, around 5 years, according to one careful longitudinal study.[2] In patients with comorbid dysthymic disorder and depression, so-called double depression, the risk for prolonged episodes and recurrence are both very high.[2] Longer episodes are also predicted by comorbidity with substance abuse, conduct disorder, or anxiety disorder, family conflict, and parental depression.[10,16]

Paediatric bipolar disorder does not often present with 'classic' periods of alternating depression and mania. Instead, such patients frequently present with either a mixed state, e.g. simultaneous occurrence of depression and mania, or rapid cycling, with brief and alternating periods of depression and mania.[4] In comparing the course of paediatric and adult bipolar patients, paediatric bipolar patients have many more episodes per year, and spend less time in remission.[4] Consistent with these longitudinal observations are findings from adult pedigrees that age of onset in bipolar disorder appears to be familial, and that earlier age of onset is associated with higher rates of drug abuse, alcohol abuse, rapid cycling, and suicide attempts.[17] Much of the impairment in paediatric bipolar disorder is associated with depressive symptoms that often never completely remit. As noted above, the adult criteria requiring 1 week and 4 days for mania and hypomania, respectively, may be overly stringent, insofar as a fairly high proportion (25 per cent) of patients below those criteria, so-called bipolar NOS, go on in longitudinal follow-up to develop clear Bipolar I or II disorder and 20 per cent of those with BP-II go on to develop BP-I within 2 years of follow-up.[4] A longer period to recovery is predicted by longer duration of mood disorder, rapid cycling or mixed episode, psychosis, and lower SES.[4]

Children and adolescents who present with a unipolar depressive disorder are at increased risk for developing a bipolar disorder, both in comparison to children without a mood disorder, and to individuals whose mood disorder has its onset in adulthood. Young depressed patients with a family history of bipolar disorder, who present with psychotic symptoms, and/or pharmacologically

induce mania or hypomania are at increased risk for developing paediatric bipolar disorder.[5,18] According to one pharmacoepidemiological study, the younger the depressed patient, the higher the risk for pharmacologically induced mania, although there was no standardized, direct assessment of manic behaviour.[19] Additionally, one study suggests that paediatric bipolar patients with comorbid ADHD tolerate amphetamine as well as non-bipolar children with ADHD.[20]

Sequelae

The most dreaded consequent of paediatric mood disorders is suicide. A unipolar depression conveys a 10–60-fold increased risk of suicide; nearly 80 per cent of adolescent suicide attempters have some form of a mood disorder.[21] Suicide attempts may be even more frequent in paediatric bipolar disorder, with almost one-third of clinical samples showing a lifetime history of a suicide attempt. Studies in adults and adolescents that have assessed for bipolar disorder find that as many as 10–20 per cent of all suicides have some form of bipolar spectrum disorder.[21] Correlates of suicidal behaviour in both unipolar and bipolar disorder include earlier age of onset, history of abuse, comorbid disruptive and substance abuse disorders, hopelessness, mood lability, and chronic and unremitting course.[21]

Other sequelae of untreated depression include educational and occupational under-attainment, interpersonal difficulties, obesity, cardiovascular disease, and alcohol and substance abuse. The effect of depression on body mass index (BMI) appears to be independent of treatment effects.[22]

Aetiology

Both unipolar and bipolar disorders have a strong genetic component. The child of a unipolar depressed parent is at around 2–4 times the risk of the population to develop a depressive disorder; this is even higher in children of parents with earlier onset (<age 20) and recurrent depression.[23] Twin studies indicate that around 50 per cent of the variance in familial transmission of depressive symptoms is explained by heritable factors, and that the liability of depression and to anxiety may be co-transmitted.[6] There is some evidence that adolescent onset depression is more highly heritable than prepubertal depression.[24]

Bipolar disorder has an even stronger genetic component, with a bipolar parent conveying at least an 8-fold increased risk of bipolar spectrum disorder in children; twin studies suggest that heritable factors account for 70–80 per cent of the variance in familial transmission.[23] High-risk studies of both unipolar depression and bipolar disorder show the transmission of a wider phenotype, with increased rates of anxiety disorder being most prominent in both the offspring of depressed and bipolar parents.[5,25]

Genetic linkage studies are beginning to converge on specific regions associated with depression and anxiety, with other regions implicated in bipolar disorder.[23] Linkage studies for early onset, recurrent depression suggest that there may be sex-specific linkage sites.[23]

The serotonin transporter promoter gene has a 44 bp insertion/deletion, with the latter resulting in less vigorous transcription. Several studies have now shown an interaction between stressful life events and the less functional form of the transporter gene with regard to an increased risk for depression in adolescents and young adults.[26] brain-derived neurotrophic factor (BDNF) is a gene that appears to protect the hippocampus system from the neurotoxic effects of stress. A three-way interaction between maltreatment, the less functional 5HTTLPR allele, and the met allele of the BDNF gene has been found with regard to risk for depression in children and adolescents.[27] An association between the val66 form of BDNF and early onset depression has been reported.[28]

Genetic linkage studies have also identified regions of interest for bipolar disorder, although paediatric bipolar disorder per se has not been studied. In aggregate, linkage studies converge on chromosomal regions 6q and 8q.[23,29] Certain phenotypes of bipolar disorder have been reported, such as 'lithium responsiveness', 'alcoholism/suicidal behaviour', and 'psychosis', with distinct areas of familial aggregation and linkage.[17] Geller *et al.*[30] has reported in an association study of paediatric bipolar families that val66 form of BDNF is associated with the disease.

Structural changes in depression have been reported, with the most widely replicated result being changes in the anterior cingulated, an area associated with emotion regulation, in both bipolar and unipolar familial depression.[31]

Paediatric bipolar subjects, compared to healthy controls, show decreased grey matter in the dorsolateral prefrontal cortex (DLPFC), cingulate cortex, and amygdale.[32] Diffusion tensor imaging and magnetic resonance spectroscopy both point to alterations in axonal development and organization in the superior frontal (decreased white matter) and DLPFC regions (decreased levels of *n*-acetyl aspartate), respectively.[33,34]

Neurocognitive factors and emotion regulation

Depression

Depressed children and adolescents, relative to normal controls, show greater attention to, and distraction by sad stimuli, whereas normal controls are more likely to be distracted by happy stimuli.[35] Depressed adolescents are more vulnerable to the effects of rumination and sad mood induction[35] Depressed children and adolescents may show less activation of reward-related circuitry when participating in a reward paradigm.[36] These findings are consistent with earlier research showing that tendency to pessimism and rumination were risk factors for the onset of depressive symptoms, especially when confronted with stressful life events.[35] Functional neuroimaging studies find alterations in amygdala activation to threat and other cognitive tasks, although the direction is not consistent across studies.[37,38]

There is evidence that these tendencies may be present prior to the development of a mood disorder. The young children of mothers with a history of childhood onset depression (COD) show greater evidence of physiological distress (e.g. poor heart rate recovery after disappointment, resort to more passive waiting and less active distraction than normal controls, and show less efficient cognitive processing when confronted with an affectively laden cognitive task.[39,40] Positive reward anticipation, however, moderated the relationship between parent early onset depression and child internalizing symptoms.[41]

The extent to which negative affective bias, and difficulty with active coping is intrinsic versus learned is still unclear. Mothers with early onset depression are less responsive to their children's expression of distress, tend to endorse and promote fewer emotion regulation strategies for their children, whereas maternal accuracy

of recognition of their child's emotional state was protective against child psychopathology.[42–44] Taken together, these findings support a role in helping parents teach their children emotion regulation strategies such as distraction, emphasis on positive reward, and active coping as a means of preventing or treating depression.[45]

Bipolar disorder

There is growing consensus that paediatric bipolar disorder is associated with difficulty with attention, verbal and visuospacial memory, executive function, set-shifting, and recognition of facial expressions.[32] In some studies, these findings are present regardless of current mood state or medication[46] and some of these findings are present in the unaffected, high-risk offspring of adults with bipolar disorder.[47] Bias towards threat, and less activation of areas involved in emotion regulation in the face of frustration have also been reported.[32] Many of these findings are also reported in unipolar depression, and therefore, a direct comparison of these two conditions is needed.

Functional neuroimaging data are consistent with these findings, with greater activation of reward-related circuitry (e.g. caudate and thalamus) and greater activation of inhibitory areas (e.g. DLPFC, anterior cingulated) when performing working memory tasks and viewing negatively valenced pictures compared to healthy controls.[32]

Neuroendocrine/sleep

Neuroendocrine studies suggest that alterations in serotonergic and noradrenergic neurotransmission are associated with early onset unipolar depression.[48] There are no clear findings with regard to cortisol regulation and depression, although hypersecretion of cortisol close to the time of sleep has been reported to be related to adolescent depression.[49] While subjective sleep complaints are common in child and adolescent depression, polysomnographic studies have not consistently shown the decreased REM latency associated with depression that has been reported in adults.[50]

Environmental risk factors

Environmental factors can also influence the onset and expression of paediatric mood disorders, often interacting with a genetic diathesis. Early abuse and neglect is a profound risk factor for depression, especially in interaction with a positive family history for mood disorder and certain genetic polymorphisms.[26,27,51] Family discord, substance abuse, and criminality are associated with depression in children from families at low familial risk for depression.[52] Maternal-child conflict shows a birectional relationship over time with regard to both parental and child mood disorder.[53] A history of abuse is associated with an earlier age of onset and more prolonged and unremitting course in bipolar disorder.[54] Loss of a parent, close friend, or sibling is associated with an increased risk of depression in those children and adolescents with a pre-existing depressive diathesis.[55] Conversely, a positive connection to family, school, and a pro-social peer group, can, in cross sectional studies, protect against depression and other health risk behaviours.[56]

Assessment and monitoring

The properties of different assessment tools of mood disorder were recently reviewed.[57,58] The most common interview-based assessment for the severity of depression is the Children's Depression Rating Scale, Revised (CDRS-R), a 17-item rating scale. Commonly used self-rating measures are the Children's Depression Inventory for children and early adolescents, the Beck Depression Inventory (for adolescents and adults), the Reynolds Adolescent Depression Scale, the Center for Epidemiological Studies, Depression Scale (CES-D), and the Mood and Feelings Questionnaire (MFQ). The CDI shows treatment sensitivity, but does not distinguish well between anxiety and depression, and the CES-D does not have an item about suicidal ideation. The main advantage of the MFQ is that it has a short form for screening, has been validated in both community and clinical sites, has a parent form (so does the CDI), and can be used for both children and adolescents.

Two interview-based methods for monitoring the level of mania are the Young Mania Rating Scale and the Mania Rating Scale from the K-SADS.[4] The advantage of the latter is that it can be taken from one of the most commonly used diagnostic interviews for children and adolescents. A self-report for mania also shows promise, but has not yet been used in clinical trials.[5]

Treatment and prevention

Depression

Practice guidelines recommend that the initial treatment for mild-to-moderate depression be support, education, and one of two forms of psychotherapy (cognitive behaviour therapy [CBT] or interpersonal therapy [IPT]). For patients who eschew psychotherapy, or who live in a region where specific indicated forms of psychotherapy are not available, antidepressant medication is an appropriate approach. Guidelines agree that for more severe depression, combination of antidepressant medication and psychotherapy are ideal, although the data are mixed on this point (reviewed below).[59,60]

Because depression is a chronic and recurrent illness, a long-term approach should be taken to the management of child and adolescent depression. Therefore, after symptomatic relief, treatment should be continued for at least 6–12 months, since there is evidence that without psychotherapy booster sessions or continued medication treatment, there is a substantially increased risk of relapse or recurrence.[59]

There is strong and convergent evidence for the efficacy of CBT for adolescent depression in clinical samples, and for child depression in symptomatic volunteers,[59,61] with a relatively modest effect size ($d = 0.34$) relative to a waitlist condition or a comparison treatment. While some studies in clinical samples showed superiority of CBT to credible alternative treatments, the largest and most comprehensive study of the treatment of adolescent depression found that CBT was no better than placebo with regard to acute clinical response (43 per cent versus 35 per cent).[62] In that study, CBT was more efficacious than placebo in those with higher incomes (>$75 000) and with higher levels of cognitive distortion.[63] While the response rate for combination (of medication and CBT) treatment was not different than for medication alone (71 per cent versus 61 per cent), combination resulted in a more rapid response and greater likelihood of remission (37 per cent versus 20 per cent) than medication alone.[63] However, studies of combination treatment for depression have been inconsistent. The addition of CBT to antidepressant treatment, both in primary care, and to the

management of moderately to severely ill depressed patients failed to improve outcome over antidepressant treatment alone.[64,65] A more recent study of depressed adolescents who did not respond to an adequate initial trial of an antidepressant found that the combination of medication and CBT resulted in a higher rate of improvement among subjects than medication alone.[66]

Interpersonal therapy is a well-established treatment for adult depression, and more recently has been shown to be superior to waitlist control, clinical management, and treatment as usual.[61] This treatment has been demonstrated to be superior to treatment as usual in a community setting, as well.[59] Other forms of treatment that show promise are attachment-based therapy and family psychoeducation, but have not yet been replicated by other groups.[59]

A group CBT approach that has been used for the treatment of depression,[67] has been adapted for the prevention of depression. In adolescents with subsyndromal depression, the group CBT resulted in a lower risk of onset of major depression than in the treatment usual group.[67] This approach was extended to the adolescent offspring of depressed parents. Adolescents, in addition to having parents with a history of depression needed to have had a prior depressive episode or subsyndromal symptoms. In an initial clinical trial and one 4-site replication, the intervention resulted in a 2–5-fold lower risk for new-onset depression.[68] The presence of current depression in the caregiver moderated the effectiveness of the CBT intervention, with children of parents with current depression failing to show an effect from the intervention. Weissman *et al.*[69] recently demonstrated that treatment of maternal depression resulted in symptomatic improvement in their children, particularly with regard to internalizing symptoms. A family psychoeducational approach has been shown to improve communication and support with regard to depression in a family member, although there was no difference in the incidence of depression in this approach versus a comparison educational treatment.[70]

The selective serotonin reuptake inhibitors (SSRIs) form the mainstay of medication management of depression. Fluoxetine is the only medication that is approved by the Food and Drug Administration (FDA) and the Medicines and Health care Regulatory Agency (MHRA) for use in paediatric depression, because it has the strongest evidence of efficacy (http://www.fda.gov and http://www.mhra.gov.uk). Other medications for which there is some evidence of efficacy are citalopram, sertraline, and venlafaxine, although for each of these medications, there is some evidence that these agents are more efficacious for adolescent than for child-onset depression.[71] In contrast, fluoxetine shows similar efficacy for both children and adolescents. Tricyclic antidepressants have been shown to be ineffective for children and adolescents with depression.[72] One possible exception is clomipramine, that has been demonstrated, when given IV to reverse chronic and refractory depression.[73] While there are no controlled trials, buproprion is commonly used for paediatric depression, and in open trials shows evidence of efficacy.[74] One small controlled study suggests that omega-3 fatty acids may be efficacious for the relief of child depression.[75]

Children and adolescents metabolize several of the antidepressants more quickly (e.g. citalopram, sertraline) compared to adults, and so equal or higher doses may be required in order to achieve a similar effect.[76] There has been little work in pharmacogenetics in paediatric populations, although one study has replicated adult findings showing that the less functional form of the serotonin transporter gene is associated with a less vigorous response to an antidepressant.[77]

The use of SSRIs increased steadily over the past decade, but enthusiasm for their use on the part of both families and clinicians has been curtailed by recent reports of an association between antidepressant use and the occurrence of spontaneously-reported suicidal adverse events (i.e. new-onset or worsening suicidal ideation or an attempt), which resulted in the FDA issuing a black box warning about this side effect. The FDA conducted a meta-analysis that showed on average that around 4 per cent of the drug-treated and 2 per cent of those on placebo developed a suicidal adverse event.[78] In the subset of studies where suicidal ideation was measured systematically, there was no difference in suicidality by treatment condition, with a trend towards a protective effect in the medication group. A more recent meta-analysis, using random- rather than fixed-effects modelling and including more studies that were not available at the time of the FDAs analysis also found an increased risk, although the estimates of the risk difference for suicidal ideation and behaviour were 0.7 per cent rather than 2 per cent.[71] In this meta-analysis, the benefits of antidepressants were also assessed, and in the case of depression, around 11 times more individuals showed clinical improvement than developed these suicidal adverse events, suggesting a favourable risk-benefit ratio for the use of antidepressants, given careful clinical monitoring.

For patients who have been treated with psychotherapy or medication, addition of a complementary modality (e.g. psychotherapy or medication) is indicated. Family discord should be addressed by family therapy, and parental depression should be identified and referred for treatment. Failure to respond at that point suggests the need to try a second SSRI, followed by either venlafaxine or bupropion. A recent study comparing depressed adolescents who did not respond to an adequate trial with an SSRI found that a switch to second SSRI was as efficacious as a switch to venlafaxine.[66] Augmentation is indicated if a patient shows a partial but palpable response but still is symptomatic, whereas those who have not responded at all should be tapered and switched to another medication.[79] There have been no clinical trials of augmentation in paediatric depressed subjects, but placebo-controlled trials in adults support the use of augmentation of SSRI treatment with lithium, T3, and bupropion.[80] Also, for depression with a seasonal component, light therapy has been shown to be efficacious in psychiatric clinical trials.[81]

Paediatric bipolar disorder

Because paediatric bipolar disorder is rarer than unipolar depression and because of the previous controversy about diagnosis, there has only recently been increased attention paid to its treatment. Best practice guidelines are based upon downward extension of experience in adults, as well as a handful of clinical trials, but the field is changing very rapidly, and it is expected that these guidelines will change in parallel.[82] Paediatric bipolar disorder has intrinsic in it a paradox: the most functionally impairing aspect of the condition is depression, but treatment of depression may induce mania. Therefore, treatment of paediatric bipolar disorder must be viewed as the prevention of future episodes, with an emphasis on mood stabilization, and not just on the relief of acute symptoms, important as that may be in the short-run. This idea that one needs to take medication in order to stay well, as compared to achieve symptomatic relief, is one that is difficult for children

and adolescents to grasp, and therefore needs to be an important target of ongoing management.

Acute management of mania

Emergent mania represents a true emergency, as it can result in risky behaviour with irreversible consequences. One key to the control of mania is to restore sleep, since sleep deprivation increases mania in a vicious cycle. Most commonly, for the acute control of mania, practitioners use atypical neuroleptics such as risperdone, olanzapine, and quetiapine, which have been shown to be more efficacious than placebo in reducing manic symptoms.[82] Quetiapine has also been shown, in one study, to be superior to divalproex in reducing manic symptoms and achieving both response and remission, whereas lithium and divalproex were shown to have similar efficacy. An open trial comparing the efficacy of three mood stabilizers for achieving clinical response in paediatric bipolar disorder found that divalproex was somewhat more efficacious (ES = 0.58) compared to either lithium or carbamazepine (ESs = 0.38). Oxcarbazepine, a metabolite of carbamazepine, on the other hand, was found to be no better than placebo in achieving stabilization. Both for the management of psychotic symptoms and mania, the combination of a mood stabilizer such as lithium or divalproex and a neuroleptic appears to be more efficacious in producing remission than a mood stabilizer alone.[82]

Acute management of depression

For the treatment-naïve patient, the first step in the management of the depressed bipolar patient is treatment with a mood stabilizer.[5,82] Open trials show that divalproex and lithium are relatively efficacious as mood stabilizers, with carbamazepine being efficacious, but less so than either of the former two agents. In adults, atypical neuroleptics are also being established as mood stabilizers with potency equal to lithium and divalproex, although with very concerning side effects of rapid weight gain. Once a therapeutic blood level of divalproex or lithium has been attained patients often will experience a relief of their depression. Sometimes alteration in the dosage, either an increase or a decrease, can bring further symptomatic relief. If a patient is still experiencing significant depressive symptoms, then an antidepressant can be added, but very carefully. Some data from adult studies suggests that bupropion may result in fewer manic break throughs than other antidepressants but those data do not exist yet in children. Lamotrigine has been shown in adults to provide prophylaxis against future depressive episodes, but is not helpful for the treatment of acute depression; this has not yet been investigated in children, except in open trials.[82]

Medical management

The medications used for mood stabilization all have systemic and potentially very serious side effects. The atypical neuroleptics cause rapid weight gain; therefore weight, a lipid profile, fasting blood sugar, and waistline should be carefully monitored. One preliminary study suggests that concomitant treatment with metformin may attenuate weight gain.[83] Hypoprolactinaemia and galactorrhoea are also consequences of neuroleptic use. Lithium is associated with thyroid disease (usually hypothyroidism), which if undetected can affect mood and treatment reponse. Lithium also can impair kidney function, resulting in an inability to concentrate urine, reduced glomerular fitration rate, and proteinuria. Therefore, renal function should be assessed prior to treatment and annually with a creatinine clearance and a 24 h urine for protein should be obtained at baseline and annually. Divalproex can have toxic effects on the hemopoetic system and the liver, both of which must be carefully monitored. Lamotrigine is rarely associated (0.5 per cent) with Stevens–Johnson syndrome, a disease of mucous membranes that can be potentially life-threatening.

Psychotherapeutic management

Patient and parent education and support are essential, including the importance of medication adherence, keeping regular sleep habits, avoidance of caffeine and other substances, and ability to recognize subtle signs of a shift in mood. Some specific forms of psychotherapy that target family process and emotion regulation have been developed.[84] Family focused treatment (FFT) has been used successful for adult bipolar disorder and results in fewer depressive episodes and better overall functioning. Pilot studies indicate what appear to be similar effects for adolescent bipolar disorder. A family psychoeducational approach also shows promise for improving adherence and reducing the risk of relapse.[85] Two other approaches that have been piloted but not yet tested in randomized clinical trials are the application of CBT to paediatric bipolar disorder, which includes family education, emotion regulation, self-monitoring, and social skills training, and the adaptation of dialectic behaviour therapy (DBT) to adolescent bipolar disorder.[86,87]

Future directions

With regard to both conditions, it is important to try to understand neural circuitry and identify potential intermediate phenotypes, such as emotion dysregulation or impaired executive functioning, which are trait markers for risk for these disorders. The identification of intermediate phenotypes then opens up great opportunities for monitoring treatment response, identifying youth at risk for the disorder, and conducting genetic studies on less complex phenotypes that are more likely to yield definitive results.

While there have been great strides in the treatment of depression, it is difficult to predict who is going to respond to what treatment, and also, who is most likely to experience side effects such as increased suicidal ideation. Pharmacogenetics and monitoring of biomarkers may hold promise for improving matching of patient to treatment. The best approaches to continuation and maintenance have only been addressed for the simplest cases (e.g. uncomplicated episode with patient having a successful response to fluoxetine.[88]

Almost every aspect of treatment in paediatric bipolar disorder requires further study, including the best approach to the management of mania, depression, and mixed state, testing the role of various promising psychosocial approaches, and identifying pharmacogenetic and biomarker predictors of treatment response.

While major depression has been described in high-risk children as young as age 3,[14] it is less clear what are the earliest manifestations of paediatric bipolar disorder. Ongoing longitudinal and high-risk studies will help to clarify the answer to this question in coming years, and may then provide the basis for preventive interventions.

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9.2.8 Obsessive–compulsive disorder and tics in children and adolescents

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Introduction

Although obsessive–compulsive disorder (**OCD**) has long been considered as a disorder of adulthood, the early child psychiatric literature contains famous descriptions of typical cases. At the beginning of the twentieth century, Janet reported on a 5-year-old with classical obsessive–compulsive (**OC**) symptoms, and Freud described in his adult patients obsessional behaviours dating back from childhood, while speculating on the strong constitutional influence in the choice of these symptoms. In the 1950's, Kanner noted the resemblance and sometimes the association between compulsive movements and tics, and Despert described the first large series of obsessive–compulsive children, noting the preponderance of males and the children's perception of the abnormality and undesirability of their behaviours.

Tics have been described since antiquity, but the first systematic reports are those of Itard, in 1825, and Gilles de la Tourette, in 1885. Both noted the association between tic disorders and OC symptoms, and speculated on the hereditary nature of the syndrome.

For the last two decades, there has been a tremendous growth of interest and research on OCD and tic disorders. Significant advances have occurred regarding the phenomenology, epidemiology, genetics, neurophysiology, pathogenesis, and treatment of both disorders. The frequent association of OCD and/or tic disorders with other neuropsychiatric disorders, as well as the increasing evidence coming from in-vivo neuroimaging studies, have led to a fascinating aspect of current neurobiological research—the possible localization of brain circuits mediating the abnormal behaviours. Of all paediatric psychiatric disorders, OCD and tic disorders now appear as model neurobiological disorders to investigate the role of genetic, neurobiological, and environmental mechanisms that interact to produce clinical syndromes of varying severity.

Clinical features

Obsessive–compulsive disorder

Obsessions are persistently recurring thoughts, impulses, or images that are experienced as intrusive, inappropriate, and distressing, and that are not simply excessive worries about realistic problems. Compulsions are repetitive behaviours or mental acts that a person feels driven to perform according to a rigidly applied rule, in order to reduce distress or to prevent some dreaded outcome. Obsessions and compulsions are egodystonic, i.e. there are considered by the subject himself as irrational or unrealistic, and are, at least partly, resisted. Children and adolescents with OCD may hide their symptoms, or will only allow them to appear at home, or in the presence of family members, suggesting partial voluntary control.

The clinical presentation of OCD during childhood and adolescence has been documented in various cultures, with clinical series

reported from the U.S., Japan, India, Israel, Denmark, and Spain. Typically, children and adolescents with OCD experience multiple obsessions and compulsions, whose content may change over time. The most frequent obsessions in young people include fear of dirt or germs, of danger to self or a loved one, symmetry or exactness, somatic, religious and sexual obsessions. The most common compulsions consist of washing rituals, repeating, checking, touching, counting, ordering, and hoarding. Generally, compulsions are carried out to dispel anxiety and/or in response to an obsession (e.g. to ward off fear of harm). However, some obsessions and rituals involve an internal sense that 'it does not feel right' until the thought or action is completed, and certain children with OCD may be unable to specify the dreaded event that the compulsive rituals are intended to prevent, beyond a vague premonition of something bad happening. Simple compulsions, such as repetitive touching or symmetrical ordering, may even lack any discernable ideational component, and may be phenomenologically indistinguishable from complex tics. Several symptom dimensions have been identified in OCD, which could suggest possible aetiologic heterogeneity. Based on the symptom categories of the Children's Yale-Brown Obsessive–Compulsive Scale (CY-BOCS, the most widely used symptomatic measure in paediatric OCD research,[1]) Stewart *et al.*[2] identified four distinct factors, using principal components analysis: (1) symmetry/ordering/repeating/checking; (2) contamination/cleaning/aggressive/somatic; (3) hoarding; and (4) sexual/religious symptoms. These symptom dimensions are congruent with those described in similar studies of adults with OCD, suggesting fairly consistent covariation of OCD symptoms through the developmental course.

Tics

Tics are sudden, rapid, non-rhythmic, stereotyped, repetitive movements (motor tics) or sounds (vocal tics). They may mimic simple or more complicated fragments of normal motor or vocal behaviours, which are misplaced in context. Tics vary greatly in nature, location, number, intensity, forcefulness, and frequency.[4,5] Common simple motor tics are neck jerking, eye blinking, elevation of shoulders, mouth movements. Common simple vocal tics include throat clearing, sniffing, sucking air, grunting, snorting, humming, or barking. Complex motor tics may combine simple tics, and involve facial movements, jumping, gyrating, touching, kicking, grooming behaviours, or echokinesis (repeating someone else's movement). They may appear to be purposive in character, as brushing hair back, or suddenly rotating on one foot to make a 360-degree turn. In a small fraction of cases, the complex motor tics are self-injuring behaviours, which may be potentially dangerous. Complex verbal tics include the repetition of what was just heard (echolalia) or said (palilalia), or socially inappropriate utterances (coprolalia), even disguised through sign language. The specific tic repertoire of an individual typically changes over time with no predictable course, but complex tics are rare in the absence of simple tics. Tics often occur in discrete unpredictable bouts over the course of a day, separated by tic-free intervals. The combination of bouts over different time scales explains why globally tics wax and wane over time.

Tics are suggestible, as indicated by their transient reappearance when they are recalled. They are also suppressible, as they can generally be willfully held back for brief periods of time. Tics are preceded by an inner tension, an urge to move or utter that may build-up during suppression. Suppressing tics requires mental effort and may accentuate inattention; conversely, attentional problems decrease the ability to suppress tics, and are associated with more severe tics. Various premonitory sensory urges have been reported to prompt the tics, together with feelings of inner conflicts over whether and when to yield to these urges. Sensory urges include focal tension, pressure, tickling, cold, warmth, paresthesias, and generalized inner tension or anxiety. They usually arise in the part of the body involved in the subsequent motor act, and completing the tic seems to yield a temporary relief of the urge. Also, various auditory and visual cues, highly selective for each individual, can elicit tics, and some patients are extremely sensitive to these external cues, as in echo phenomena. Excitement and fatigue typically worsen tics, which are often more frequent and forceful when the individual is alone. Activities requiring fine motor skills and attention improve tics. Although much diminished, tics can occur during sleep, unlike many other movement disorders.

Children and adolescents with tic disorders may present a broad array of associated behavioural difficulties, including OC symptoms, disinhibited speech or conduct, impulsivity, distractibility, and motor hyperactivity.[5] The presence of motor and/or phonic tics can be associated with difficulties in self-esteem, self-definition, family life, peer acceptance or relationships, and school performance.

Age of onset

The age at onset of OCD appears bimodal.[6] Prepubertal onset is associated with a male preponderance and an increased risk for tic disorders, including Tourette's disorder. A second peak of onset occurs at or after puberty. Overall, the mean age at onset of OCD in children and adolescents have ranged from 9 years in referred subjects[7] to 12.8 years in a community sample.[8]

The median onset age for simple motor tics is between 4 and 6 years.[9] Phonic or vocal tics usually appear several years after the onset of motor tics, in most cases between 8 and 15 years. Many young children are completely oblivious of their tics, or experience them as wholly involuntary. Premonitory urges typically show up several years after the onset of the tics, on average around 10 years of age. Suppressibility of tics developmentally precedes awareness of premonitory urges, but may get easier as awareness increases.[10]

Sex ratio

In community-based samples of adolescents with OCD, there are approximately equal numbers of males and females, while in most studies of referred children and youth with OCD, males outnumber females by 2:1 or 3:1.[11]

Most studies show that the prevalence of tics is higher among boys than girls, with a ratio of 6–8:1 in clinic-based samples, and about 2:1 in community-based studies.[12] The sex ratio generally increases with tic duration and severity. Thus, in one study, the ratio of boys to girls was 1.6:1 for motor tics present for 1-2 consecutive months, increasing to 7.5:1 when tics were present for 2 non consecutive months, or more than 3 months.[13]

Comorbidity

In referred children and adolescents with OCD, the frequency of a diagnosis of any tic disorder ranges from 17 per cent to 40 per cent, and that of Tourette's disorder from 11 per cent to 15 per cent.[14]

Conversely, one study found that 29 per cent of Tourette's disorder patients displayed OC behaviours.[15] In longitudinal studies, about 50 per cent of children and adolescents with Tourette's disorder develop OC symptoms or OCD by adulthood,[16] whereas, in a follow-up study of children and adolescents initially treated for OCD, nearly 60 per cent were found to have a lifetime history of tics that ranged from simple, mild, and transient tics to Tourette's disorder, for which the rate was 11 per cent.[17] On the basis of personal or family history of tics, a distinction has been proposed between 'tic-related OCD' and 'non-tic-related OCD', under the assumption that the two forms might differ in terms of clinical phenomenology, neurobiological concomitants, and responsiveness to pharmacological interventions.[18] Tic-related OCD appears to have an earlier onset, and to occur more frequently in boys than in girls. The need to touch or rub, blinking and staring rituals, worries over symmetry and exactness, a sense of incompleteness, and intrusive aggressive thoughts and images, are significantly more common in tic-related OCD, whereas contamination worries and cleaning compulsions are more frequent in patients with non-tic-related OCD.

The overall lifetime psychiatric comorbidity in children and adolescents with OCD is about 75 per cent, both in referred and in community cases. The most common conditions comorbid with OCD are affective disorders, with prevalence ranging across studies from 8 per cent to 73 per cent for mood disorders, and from 13 per cent to 70 per cent for anxiety disorders.[19] While occurring less frequently in non-referred subjects, a high rate of disruptive behaviour disorders—attention deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder—has been reported in subjects seen in paediatric OCD clinics. In girls, OCD can be comorbid to anorexia nervosa.

Less than 10 per cent of clinically referred children and adolescents with Tourette's disorder do not have another morbid condition. About 55 per cent also have ADHD, and more than a third have anger control problems.[9] Rage attacks in response to minimal provocation, lasting from a few minutes to an hour and usually followed by remorse, as well as an increased vulnerability for drug abuse, depression, and antisocial behaviour, are primarily observed when comorbid ADHD is present. Globally, comorbidity of Tourette's disorder increases in adolescence, but more markedly for OCD and anxiety disorders in those without ADHD. Individuals with Tourette's disorder have consistently shown difficulties with fine motor control and visual motor integration, as well as impairment in procedural or habit-based learning. Sleep is often disturbed, with increased short lasting motor activity, especially in non-REM sleep, compared to healthy controls.

Classification

Both DSM-IV and ICD-10 define OCD, regardless of age, by obsessions and/or compulsions, which are described, at some point during the course of the disorder, as excessive or unreasonable (criterion B), and are severe enough to cause marked distress or to interfere significantly with the person's normal routine, or usual social activities or relationships. The specific content of the obsessions or compulsions cannot be restricted to another Axis I diagnosis, such as an eating disorder, a mood disorder, or schizophrenia. The DSM-IV adds that the disturbance is not due to the direct physiological effects of a substance or a general medical condition. The ICD-10 allows subclassification of forms with predominant obsessions, predominant compulsions, or mixed symptoms. In DSM-IV, the only difference in diagnostic criteria between children and adults appears in criterion B; although most children and adolescents actually acknowledge the senselessness of their symptoms, the requirement that insight is preserved is waived for children.

In both DSM-IV and ICD-10, tic disorders are divided into four categories, according to duration of the symptoms, and presence of vocal tics in addition to motor tics: Tourette's disorder, chronic motor or vocal tic disorder, transient tic disorder, and tic disorder not otherwise specified (NOS). Transient tic disorder is defined by single or multiple motor and/or vocal tics that occur many times a day, nearly everyday for at least 4 weeks, but for no longer than 12 consecutive months. In chronic motor or vocal tic disorder, either motor or vocal tics, but not both, have been present at some time during the illness. In Tourette's disorder, both multiple motor and one or more vocal tic have to be present, although not necessarily concurrently. Both Tourette's disorder and chronic motor or vocal tic disorder have a duration of more than 1 year, with no tic-free period of more than 3 months. All tic disorders must have onset before age 18 years. In all, the disturbance causes marked distress or significant impairment in social, occupational, or other important areas of functioning, and is not due to the direct physiological effects of a substance (e.g. stimulants), or a general medical condition. The ICD-10 recognizes that there is an immense variation in the severity of tics. At the one extreme of the continuum, the presence of transient tics, at some time during childhood, is near-normal. At the other extreme, Tourette's disorder is an uncommon, chronic, and incapacitating disorder.

Diagnosis and differential diagnosis

In ICD-10, it is stated that OCD cannot be diagnosed if the patient meets Tourette's disorder criteria, while both diagnoses may be given simultaneously in DSM-IV. Unlike tics, compulsions are aimed at neutralizing the anxiety resulting from an obsession, and/or they are performed according to rules that must be applied rigidly. However, both compulsive rituals and complex tics may be preceded by premonitory urges, which persist until the action is completed. In individuals with both Tourette's disorder and OCD, these symptoms are sometimes so closely intertwined that efforts to distinguish them would be futile.

From a developmental perspective, pathological OC behaviours and thoughts differ from normal childhood rituals, mainly by their emotional context and their use of maladaptive versus adaptive cognitive and behavioural strategies.[20] Developmental childhood rituals are part of learning new skills, and accompanied by expressions of positive affect and interest. They are most intense in 4- to 8-year-olds, stress rules about daily life, help the child master anxiety, and enhance the socializing process. In contrast, perseverative behaviours in OCD are not goal-oriented, they are accompanied by a burdened, anxious affect, provoke frustration, are incapacitating and painful, and promote social isolation and regressive behaviour.

OCD must be distinguished from other anxiety disorders and, in some cases, from autism or schizophrenia. In phobias, subjects are preoccupied by their fears only when confronted to the phobogen stimuli, and, in separation anxiety disorder, fear of harm to

parents or loved ones are part of persistent worries and behaviours which are not criticized by the child. Stereotyped movements and ritualistic behaviours are frequent in intellectual disability and autism, but they convey no particular intentionality, and the child does not try to resist them. In schizophrenia, there are erroneous belief systems in several areas, but the subject does not criticize them and does not consider the subsequent behaviours to be abnormal.

Tics should be differentiated from other types of abnormal movements which can occur in numerous congenital or acquired neurological and neuropsychiatric disorders (Sydenham's chorea, encephalitis, Huntington disease, tuberous sclerosis, neuroacanthocytosis, Wilson's disease, head trauma, mental retardation, autism). The term of secondary tics or Tourettism has been applied to these disorders, and the abnormal movements can be choreiform movements, dystonic movements, myoclonic movements, spasms, or stereotypies. Some medications such as central nervous system stimulants (methylphenidate, amphetamine, pemoline, cocaine), antihistaminic and anticholinergic drugs, antiepileptics (carbamazepine, phenytoin), antipsychotics, and opioids may also produce or exacerbate tics.[21] The distinction between tic disorders and other disorders with abnormal movements is based on anamnesis, family history, observation, and neurological examination, which is usually normal in tic disorders. Specific diagnostic tests may be required to confirm neurological or exogenous causes.

Epidemiology

Tics might be one of the most common behavioural problems in childhood, but estimates of the prevalence of tic disorders greatly vary because of differences in the methods used (e.g. parental report versus direct observation), differences in the populations surveyed (e.g. age and sex distribution), and the transient nature of tics. Surveys among school-age children indicate that up to 18 per cent of boys, and 11 per cent of girls manifest frequent 'tics, twitches, mannerisms or habit spasms'. Race and socio-economic status do not seem to influence the frequency of tics. There are virtually no general population studies of transient tic disorder or chronic motor or vocal tic disorder. For Tourette's disorder, most population-based surveys yield prevalence estimates in the range of 5–10 per 10,000, with children being more likely to be identified than adults, and males more than females.[22, 13] In a study amongst all inductees into the Israeli Defence Force over 1 year, the point prevalence of Tourette's disorder was 4.9 per 10 000 males and 3.1 per 10 000 females, and the prevalence of OCD was elevated in those with Tourette's disorder (41.7 per cent vs. 3.4 per cent in others).[23] One longitudinal study assessed the presence of tics and OCD in an epidemiological sample of individuals followed from childhood to adulthood.[24] The prevalence of tics was 17.7 per cent at age 1–10 years, decreasing to 2–3 per cent in adolescence; childhood tics were associated with increased rates of OCD in adolescence; in adolescents with tics, the presence of comorbid OCD predicted persistence of tics into early adulthood.

There has been only one survey on the prevalence of OCD in children (5- to 15-year old), indicating an overall prevalence of OCD at 0.25 per cent, with an exponential increase as a function of age, from 0.026 per cent in 5–7 year olds to 0.63 per cent in 13–15 year olds.[25] In adolescents, epidemiological studies using strict diagnostic criteria and structured clinical interviews have been conducted in several parts of the world, estimating the prevalence of juvenile OCD between 1 and 4 per cent. In the largest study to date (N=5 596 high-school students), the lifetime prevalence of OCD in adolescents was estimated to 1.9 (±0.7) per cent, and none of the identified cases had been previously diagnosed.[8] In a later study,[26] the point prevalence of OCD was 3.6 (±0.7) per cent, decreasing to 1.8 per cent when excluding those individuals with only obsessions; among the OCD cases, there was a significant elevation of tic disorders (Tourette's disorder 5 per cent, chronic multiple tics 10 per cent, transient tics 10 per cent). In two longitudinal studies following cohorts of children in the community up to the age of 18 years, the prevalence for OCD ranged from 1.2 to 4 per cent.[27, 28] Thus, it appears that OCD might be as frequent in adolescents as it is in adults (see Chapter 4.8).

Aetiology

Psychological factors

Psychological theories of OCD have encompassed psychoanalytic as well as more general non-psychodynamic etiological approaches, focusing alternatively on volitional, intellectual, and/or emotional impairment. Freud's famous patient, the Rat Man, has been seen as a paradigm of a psychologically determined illness, illustrating the central role of anal sadistic concerns with control, ambivalence, magical thinking, and the salience of defenses such as reaction formation, intellectualization, isolation, and undoing. Freud also provided fascinating speculations on the similarity between OC phenomena, children's games, and religious rites. Later, Anna Freud stated that 'obsessional outcomes are promoted by a constitutional increase in the intensity of the anal-sadistic tendencies probably as the result of inheritance combined with parental handling'. However, despite the beautifully described dynamics of obsessional symptoms, most illustrative of unconscious processes, the psychoanalysts have also pointed out the extreme difficulty in treating OCD with classical analytic treatment.

Even though psychological factors are insufficient to cause Tourette's disorder, tic behaviours have long been identified as stress-sensitive, and temporally associated with important events in the lives of children. In a prospective study over 2 years, children and adolescents with Tourette's disorder and/or OCD experienced significantly more psychosocial stressors than did healthy controls, and the level of psychosocial stress was a significant predictor of future tic and OC symptoms severity.[29]

Biochemical factors

Although a variety of biological aetiologies have been proposed in OCD since the 19th century, modern neurobiological theories began with the clinical studies showing that clomipramine and other serotonin reuptake inhibitors (**SRIs**) had a unique efficacy in treating the disorder. This inspired a 'serotoninergic hypothesis' of OCD (see Chapter 4.8). In children, the involvement of the serotonin system in the pathophysiology of OCD is supported by one study in which improvement of OC symptoms during clomipramine treatment was closely correlated with pretreatment platelet serotonin concentration,[30] and reports of decreased density of the platelet serotonin transporter in children and adolescents with OCD but not in those with Tourette's disorder.[31] However, the

delayed and incomplete action of serotonergic drugs, suggesting multiple effects on other neurotransmitters as well, and numerous biochemical studies of OCD patients and controls have not yet indicated a single biochemical abnormality as a primary etiological mechanism in OCD.

In Tourette's disorder, multiple neurochemical systems have been implicated by pharmacological and metabolic studies, but a primary disturbance in the dopaminergic system is supported by the tic suppressing effect of dopamine receptor antagonists (see below). Post-mortem studies have shown an increase in the number of presynaptic dopamine transporter sites in the striatum and the frontal cortex of individuals with Tourette's disorder. PET/SPECT studies have demonstrated greater binding to dopamine transporter sites in both the caudate and putamen nuclei,[32] increased dopamine release by psychostimulants in the putamen,[33] and an association between density of dopamine receptors in the caudate and severity of tics.[34] The 'tonic-phasic hypothesis' proposes both a hyperresponsive spike-dependent (phasic) dopaminergic system (possibly related to an alteration in afferent cortical inputs), and a reduction in tonic dopamine levels (possibly secondary to an overactive dopamine transporter system), that would upregulate pre- and postsynaptic dopamine receptors and further increase the phasic-tonic unbalance. There is also some evidence for the role of serotonin in tic disorders, notably a study showing that reduced serotonin transporter binding correlated with vocal tics and OC symptoms.[35]

Genetic factors

In both OCD and Tourette's disorder, twin and family studies provide strong evidence that genetic factors are involved in the vertical transmission of vulnerability within families. The average concordance rate in monozygotic twins is 65 per cent for OCD,[36] and 53 per cent for Tourette's disorder.[37] Family studies have consistently found higher rates of OCD and tic disorders in probands with paediatric OCD, as well as higher rates of tic disorders and OCD in those with tic disorders. Thus, Lenane *et al.*[38] investigating 147 first-degree relatives of children and adolescents with OCD found that 44 per cent of the families had a positive history of tics in at least one first-, second-, or third-degree relative. Conversely, Pauls *et al.*[39] reported that the prevalence rates of OCD and tic disorders were significantly greater among the first-degree relatives of 100 probands with OCD (10.3 per cent and 4.6 per cent, respectively) than among relatives of psychiatrically unaffected subjects (1.9 per cent and 1.0 per cent). These findings suggested that Tourette's disorder and some forms of OCD could be variant expressions of the same underlying genetic factors.

Results from two genome-wide scans have been reported,[40,41] with the strongest linkage peaks being on chromosome 2 for Tourette's disorder ($p=0.00004$), and chromosome 3 for OCD ($p=0.0002$); there were also regions that showed moderate evidence for linkage to both disorders. In early-onset OCD, family-based evidence for association at several serotonin system genes (SCL6A4, HTR1B, HTR2A) and brain-derived neurotropic factor (BDNF) has been reported in some studies, and the association seemed stronger in subjects with tic disorders associated with OCD.[42] There is also preliminary evidence for an association between OCD and two glutamate genes, the glutamate transporter gene (SLC1A1), and a glutamate receptor gene (GRIN2B).[43] A complementary approach involving examining rare cases of cytogenetic abnormalities co-segregating with Tourette's and related disorders has pointed to regions on chromosomes 3p, 7q, 8q, 9p, and 18q.[44]

Thus, as suggested by earlier family studies, OCD and Tourette's disorder might have both shared and distinct susceptibility genes involved in their etiology. It is likely that epigenetic and non genetic factors may also contribute to phenotypal heterogeneity. A range of prenatal and perinatal events have been suggested as risk factors for increased tic severity, including lower birth weight, in utero exposure to caffeine, alcohol or tobacco, and maternal stress.[45] As the basal ganglia are especially sensitive to hypoxia, it is possible that factors associated with transient hypoxia could increase the risk for Tourette's disorder in those with a genetic vulnerability.

Dysfunction of frontal–subcortical circuits

It has been known for a long time that OC symptoms could be associated with neurological disorders of motor control, including Tourette's disorder, Huntington's disease, Parkinson's disease, as well as traumatic or infectious lesions of the basal ganglia.[46] Conversely, in both adults and children with typical OCD, an increased frequency of soft neurological signs has been reported.[47] Since the era of neuroimaging, numerous studies have consistently found that the ventral prefrontal cortical (VPFC) regions, such as orbital prefrontal cortex and anterior cingulate cortex, the striatum, the basal ganglia and the thalamus were basic brain structures involved in the pathophysiology of OCD. These studies have generally identified abnormally high metabolic activity and/or blood flow in the orbital cortex and the head of the striatal caudate nucleus in untreated OCD subjects at rest, compared to various control populations.[48,49] Furthermore, the same two regions, as well as the thalamus to which each projects, have shown further increase in activity during OC symptom provocation. In several functional neuroimaging studies of patients with childhood onset OCD, measures of VPFC and striatal activity correlated positively with OCD symptom severity and treatment response.[50,51] Some studies have also indicated that the anatomy of the caudate, putamen and globus pallidus could differ between paediatric OCD patients and controls,[52] especially in cases of paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS).[53] Although most studies have implicated the VPFC in the pathogenesis of OCD, recent investigation suggests a role for the dorsolateral prefrontal cortex (DLPFC) as well. Thus, one study found a significant increase in N-acetyl-aspartate (NAA), a neuronal marker of activity, in the left DLPFC of unmedicated paediatric OCD patients compared to controls.[54]

Recent MRI studies found that the volume of the caudate nucleus is decreased in both children and adults with Tourette's disorder, whereas the volume of putamen and globus pallidus nuclei are primarily reduced in adults with the disorder.[55] This is consistent with a study comparing monozygotic twins discordant for tic expression, in which caudate nuclei volumes were smaller in the more severely affected co-twin.[56] In addition, subjects with Tourette's disorder were found to have larger volumes in dorsal prefrontal and parieto-occipital regions.[57] Although no association was found between tic severity and the volumes of the basal ganglia, ratings of worst-ever tic severity were associated with larger orbito-frontal and parieto-occipital regions. In one recent study, cortical and subcortical hyperintensities that are considered as a subclinical manifestation of small-vessel disease, were significantly more abundant in children and adolescents with Tourette's

disorder, OCD or ADHD than in healthy controls.[58] These results support a primary disturbance of the cortico-striato-pallidal-thalamo-cortical circuit, especially the projection into or out of the striatum. The small reduction of the caudate (about 5 per cent) may represent a marker for Tourette's disorder, and larger prefrontal cortex would likely result from the ability to suppress tics. Although tics are highly heritable, non genetic factors appear to contribute to these brain differences.

Autoimmune factors

For the last decade, clinical and research interest has grown in an autoimmune model of OCD and/or tic disorders, which could apply to a subgroup of subjects whose disorder begins abruptly during childhood. An association was first reported between acute onset OCD and Sydenham's chorea, a childhood movement disorder associated with rheumatic fever, which is thought to result from an antineuronal antibody-mediated response to group A beta-haemolytic streptococcus (GABHS), directed at portions of the basal ganglia.[59] OCD, or some of its symptoms, have been reported in 70 per cent of Sydenham's chorea cases.[60, 61] Furthermore, in the absence of the neurological symptoms of Sydenham's chorea, post-streptococcal cases of childhood-onset OCD, tics and/or other neuropsychiatric syndromes have been described under the acronym of paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS). Swedo *et al.*[6] defined this novel group of patients using five diagnostic criteria: presence of OCD and/or tic disorder, prepubertal onset, episodic course of symptom severity, abrupt onset or dramatic exacerbations of symptoms temporally associated with GABHS infections (as evidenced by positive throat culture and/or elevated anti-GABHS titers), and association with neurological abnormalities (motoric hyperactivity or adventitious movements, such as choreiform movements or tics). An antigen labelled D8/17, on the surface of peripheral blood mononuclear cells has been shown to be a marker for the genetic tendency to generate abnormal antibodies to GABHS. Two independent groups of researchers have found a greater expression of the D8/17 antigen in the B lymphocytes of patients with childhood-onset OCD or Tourette's disorder compared with healthy controls, indicating that the presence of the D8/17 antigen may serve as a marker of susceptibility for OCD or tics.[62, 63]

Course and prognosis

Several follow-up studies of subjects treated for OCD during childhood or adolescence have looked at the outcome of the disorder in early adulthood.[11] All studies demonstrate the continuity of the diagnosis of OCD from childhood to adulthood: when subjects are still symptomatic, the main diagnosis is almost invariably OCD, although comorbid disorders are frequent, especially mood and/or anxiety disorders. Spontaneous course is most often marked by a waxing and waning severity of the disorder, whereas remissions under treatment can be followed by relapses, even after long periods of time. In the early studies in which subjects had received no or non-specific treatment, the recovery rate was poor (13–30 per cent). By the time patients had access to specific treatment with SRIs and/or cognitive behavioural therapy (CBT), recovery rates increased to 55–65 per cent, although many of the symptom-free subjects at follow-up were still taking medication. A meta-analysis analyzed 16 studies that followed paediatric OCD patients between 1 and 15 years.[64] The overall remission rate (not fulfilling criteria for subthreshold or full OCD) was 40 per cent, with pooled mean persistence rates of 41 per cent for full OCD. Poor prognostic factors included a poor initial treatment response, and comorbid psychiatric illness.

In the majority of cases, tics are transient (present for less than 12 months), or wax and wane in severity with periods of exacerbation of an average duration of 9 weeks. The course of worst-ever tic severity usually falls between 7 and 14 years of age, which also includes the period when tics are most variable (10–12 years). By the end of the second decade, there is usually a steady decline in tic severity. However, adults who are able to suppress tics may be left with distracting urges, and a significant minority (15–30 per cent) continues to have severe tics into adulthood.[65] Despite substantial problems in childhood, the majority of patients with Tourette's disorder grow up to become well socially integrated and economically independent adults. However, as much as 25 per cent have persistent mental health problems. In those, tic severity fluctuates, and psychiatric co-morbidities (ADHD, other disruptive behaviour problems, OCD, mood and anxiety disorders, learning problems) are often the main determinants of global outcome. Poorer prognoses are also associated with comorbid developmental disorders, chronic physical illness, unstable or unsupportive family environment, social difficulties, or exposure to psychoactive drugs such as cocaine.[5]

Treatment

Evidence

The treatment of paediatric OCD has changed dramatically over the past 20 years, with two modalities being empirically shown to ameliorate the core symptoms of the disorder: CBT and pharmacological treatment with SRIs. In Tourette's disorder, D_2 dopamine antagonists have been used with relative success since the 1960s, and CBT techniques are being increasingly scrutinized.

Cognitive behavioural treatment

The cognitive behavioural model of OCD posits that compulsions function to reduce fear, and are subsequently reinforced by fear reduction, which prevents normal habituation and realistic appraisal of the threat value of feared stimuli. Techniques incorporating exposure and ritual prevention are designed to break this cycle by exposing the individual to feared situations, while simultaneously reducing compulsive behaviours.[66] Discussion of obsessive thoughts, and other irrational beliefs, is often part of the exposure exercises but these informal cognitive techniques are used to support exposure rather than to replace it. The CBT of youth with OCD generally involves a three-stage approach, consisting of information gathering, therapist-assisted graded exposure with response prevention, and homework assignments.[67] Anxiety management training plays an adjunctive role. For children with predominantly internalizing symptoms, treatment also includes relaxation and cognitive training. Families need to be involved, to varying extents according to individual situations. CBT is usually implemented with 13 to 20 weekly individual or family sessions, and homework assignments. Partial responders or nonresponders may require more frequent sessions, and out-of-office therapist-assisted training.

Table 9.2.8.1 Controlled studies of pharmacological or psychological treatment in paediatric OCD

Study, Year	N (age), Study duration	Drug (daily dose), Study design	Outcome	% Improvement from baseline on active treatment for OC symptoms
Flament *et al.*, 1985	19 (6–18 yr), 5 wk	CMI (mean 141 mg) Crossover vs. PBO	CMI > PBO at 3–5 wk	22–44 %
Leonard *et al.*, 1989	47 (7–19 yr), 5 wk	CMI (mean 150 mg) Crossover vs. DES	CMI > DES at 3–5 wk	19–44 %
DeVeaugh-Geiss *et al.*, 1992	60 (10–17 yr), 8 wk	CMI (75–200 mg) Parallel vs. PBO	CMI > PBO at 3–8 wk	34–37 %
Riddle *et al.*, 1992	14 (8–15 yr), 8 wk	FLX (20 mg) Crossover vs. PBO	FLX > PBO at 8 wk	33–44 %
March *et al.*, 1998	187 (6–17 yr), 12 wk	SER (mean 167 mg) Parallel vs. PBO	SER > PBO at 3–12 wk	21–28 %
Riddle *et al.*, 2001	120 (8–17 yr), 10 wk	FLV (50–200 mg) Parallel vs. PBO	FLV > PBO at 1–10 wk	21–25 %
Geller *et al.*, 2001	103 (7–17 yr), 13 wk	FLX (mean 40 mg) Paralell vs. PBO	FLX > PBO at 7–13 wk	25–49 %
Liebowitz *et al.*, 2002	43 (8–17 yr), 16 wk	FLX (mean 64 mg) Parallel vs. PBO	FLX > PBO at 16 wk	42 %
Geller *et al.*, 2004	203 (7–17 yr), 10 wk	PAR (mean 30 mg) Parallel vs. PBO	PAR > PBO at 2–10 wk	47–65 %
Barrett *et al.*, 2004; 2005	77 (7–17 yr), 14 wk	Randomized parallel study ICBFT vs. GCBFT vs. control condition	ICBFT and GCBFT > control condition at 14 wk	65 % for ICBFT 60 % for GCBFT

CMI: clomipramine; DES: desipramine; FLV: fluvoxamine; FLX: fluoxetine; PAR: paroxetine; PBO: placebo; SER: sertraline

ICBT: individual cognitive behavioural treatment, GCBT: group cognitive behavioural treatment

A number of open trials, and four controlled studies (see Tables 9.2.8.1 and 9.2.8.2), have documented the beneficial effects of CBT, alone or in combination with pharmacotherapy, for children and adolescents with OCD, with improvement measured on the CY-BOCS scores ranging from 25 per cent to 67 per cent. In the first controlled study by Barrett *et al.*,[68] 88 per cent of youth treated with individual cognitive behavioural family treatment (CBFT), and 76 per cent of those treated with group CBFT showed clinically significant improvement, as compared to no improvement for any patients in the waitlist condition. Treatment gains were maintained at 12- to 18-month follow-up, with a total of 70 per cent of participants in individual therapy, and 84 per cent in

Table 9.2.8.2 Comparative studies of cognitive behavioural treatment and pharmacological treatment in paediatric OCD

Study, Year	N (age), Study duration	Drug (daily dose), Study design	Outcome	% Improvement / Remission for OC symptoms
De Haan *et al.*, 1998	22 (8–18yr), 12 wk	CMI CMI (25–200 mg) vs. BT	BT > CMI	Improvement from baseline BT: 59.9 % CMI: 33.4 %
POTS Team 2004	112 (7–17yr), 12 wk	SER (mean 150 mg) vs. PBO vs. CBT vs. Combi (CBT+SER) 3-site study, Parallel design	Combi > CBT = SER > PBO effect size: 1.4 (Combi), 0.97 (CBT), 0.67 (SER)	Improvement from baseline Combi: 53 %; CBT: 46 %; SER: 30 %; Placebo: 15 % Remission post-treatment (CY-BOCS<10) Combi: 53.6 %; CBT: 39.3 % SER: 21.4 %; PBO: 3.6 %
Asbahr *et al.*, 2005	40 (9–17 yr), 12 wk F/U: 9 mo	SER (mean 137 mg) vs. GCBT	Significant improvement on CY-BOCS with both GCBT and SER Relapse during F/y: 50% (SER), 5 % (GCBT)	NR

BT: behavioural treatment; CBT: cognitive behavioural treatment; CMI: clomipramine; Combi: combination treatment; F/U: follow-up; GCBT: group cognitive behavioural treatment; NR: not reported; PBO: placebo; POTS: paediatric obsessive–compulsive disorder treatment study; SER: sertraline

group therapy diagnosis-free at follow-up, and no significant difference between the two treatment modalities.[69] More evidence for the efficacy of CBT in the treatment of paediatric OCD comes from three studies that have compared CBT to pharmacotherapy and/or their combination (see below).

Behavioural techniques play an important role in the treatment of tics, although generally as adjunctive to medication. In a model of operant conditioning, ticking relieves unpleasant premonitory sensations, which reinforces the maintenance of tics. Habit reversal (HR) is based on awareness training regarding the premonitory urges, followed by training a competing response (a movement that involves the same muscle group as the tic) after the first sensation that a tic is about to occur. The response must be temporally contingent on each occurrence of the urge, but using a muscle group related or unrelated to the tic may not be crucial for tic suppression. Relaxation, self-monitoring, contingency training for positive reinforcement of not ticking, and social support are used as ancillary components of HR. An extension of HR is exposure and response prevention (ERP): after the urge, the patient suppresses the tic voluntarily, which should lead to its extinction. Both techniques have been showed to yield relatively large effect size (1.06 to 1.42) in reducing tic severity at post-treatment[70] and long-term follow-up.[71] A recent review of the literature[72] concluded that the use of HR to treat tics can currently be classified as a 'well established' treatment, and that of ERP as a 'probably efficacious' treatment. Contrary to initial fears, these behavioural techniques have no negative consequences, such as substitution of the targeted tics, or post-suppression rebound or worsening due to increased awareness of premonitory urges.

Psychopharmacological treatment

In the past 25 years, a number of randomized, controlled clinical trials (summarized in Table 9.2.8.1) have been conducted in children and adolescents with OCD demonstrating, as in adults, the selective and unique efficacy of the SRIs in the short-term and long-term treatment of the disorder.[67,73–79] Results have consistently shown that: the antiobsessional action of the SRIs is independent of the presence of depressive symptoms at baseline; their antiobsessional action takes longer to appear than their antidepressant action; the therapeutic response occurs gradually over a few weeks to a few months; final response is most often partial, with a mean reduction of OC symptoms from baseline to post-treatment ranging from 19 per cent to 44 per cent across measures and across studies.[80] Geller *et al.*[81] conducted a meta-analysis of 12 randomized, controlled medication trials in children and adolescents with OCD (total N=1 044), demonstrating that all serotonergic medications were highly significantly superior to placebo, with consistent findings across studies but a modest overall effect (the pooled standard mean difference between active drug and placebo was only 0.46). Clomipramine was statistically superior to the specific serotonin reuptake inhibitors (SSRI), but temporal trends might, at least, partly explain this apparent superiority: the clomipramine trials were conducted earlier in time when no other treatment was available, while the patient population included in subsequent controlled trials have changed over the years with increased availability of pharmacological alternatives. No head-to-head paediatric studies of clomipramine versus an SSRI have been conducted. The recommended daily dosages for SSRIs in the treatment of paediatric OCD are shown on Table 9.2.8.3.

Table 9.2.8.3 Recommended daily dosages of serotonin reuptake inhibitors for the treatment of paediatric OCD

Medication	Starting Dose[a]	Initial Targeted Dose[b,c]	Maximal Dose[b]
Citalopram	10 mg	40 mg	60 mg
Escitalopram	5 mg	20 mg	20 mg
Fluoxetine	10 mg	40 mg	80 mg
Fluvoxamine	25 mg	200 mg	300 mg
Paroxetine	10 mg	40 mg	60 mg
Sertraline	25 mg	100 mg	200 mg
Clomipramine	10 mg	150 mg	250 mg

[a] these doses should be given for about one week, that is about the time necessary to achieve steady state for these drugs, with the exception of fluoxetine. This would ensure that no agitation or increased anxiety is triggered by the medication.

[b] for subjects weighing at least 50 kg; for smaller individuals, a weight-proportional regimen should be used

[c] according to side effects and response

A few studies, summarized in Table 9.2.8.2, have compared pharmacological treatment to CBT or their combination for children and adolescents with OCD.[82–84] In the U.S. 12-week paediatric OCD treatment study (POTS; N=112), the combined treatment with CBT and sertraline had the best rate of clinical remission (53.6 per cent vs 39.3 per cent on CBT alone, 21.4 per cent on sertraline alone, and 3.6 per cent on placebo); the remission rate for the combined treatment was not statistically different from that in the CBT only condition.[83] In Asbahr *et al.*[84] study, both group CBT and sertraline induced a significant improvement in OC symptoms, but after a 9-month post-treatment follow-up period, subjects in the group CBT condition had a significantly lower rate of symptom relapse. A few case reports have also indicated that the addition of CBT to pharmacotherapy can allow successful withdrawal from medication.

The use of clomipramine can entail anticholinergic side effects (dry mouth, dizziness, headache, tremor, fatigue, constipation, sweating, dyspepsia, sexual dysfunction), which may not abate over time and even increase with ascending titration.[85] Clomipramine can cause tachycardia and prolongation of the QT and QTc intervals, and ECG monitoring is recommended.[86] Risks of toxicity also include seizures, and rare cases of sudden death have been reported in children taking tricyclic antidepressants.[87] Although less frequent and less disturbing that the secondary effects of clomipramine, the most commonly described adverse effects of the SSRIs include gastro-intestinal (nausea, constipation, abdominal pain), and central nervous system complaints (headache, tremor, drowsiness, akathisia, insomnia, disinhibition, agitation).[88] The possible induction of mania can also be of concern. Although not commonly reported in clinical trials, the eventual occurrence of sexual side effects should be reviewed with adolescents, since these may impact adherence to treatment. Recent reports of possible growth suppression associated with the SSRIs suggest that monitoring of height may also be advisable.[89] A recent concern, highly visible in the media, has been a possibly increased risk for suicidal thoughts, self-harm and/or harm to others, in youth treated with the SSRIs. However, no individual OCD study has documented a significantly increased risk for suicidal ideation or behaviour on

a SSRI compared to placebo. In pooled analyses of the controlled studies conducted in youth with OCD and other anxiety disorders, behavioural side effects variously labelled as activation, akathisia, disinhibition, impulsivity, and hyperactivity have appeared, but there was no evidence for a significant increase in the relative risk of suicidal thoughts or behaviours.[90] In a recent review of 27 trials of antidepressants in participants younger than 19 years (including six trials for treatment of OCD), there was an increased risk difference of suicidal ideation/suicide attempts across trials and across indications for drug versus placebo, but no completed suicide, and the benefits of antidepressants appeared much greater than the risk.[91] In any case, rigorous clinical monitoring for suicidal ideation and other potential indicators for suicidal behaviour remains advised in youth treated with the SSRIs.

The mainstay of treatment for Tourette's disorder has been traditional antipsychotics, i.e. the potent dopamine (D2) postsynaptic blockers haloperidol and pimozide.[92] The usual starting dose is 0.25 mg/day of haloperidol or 1 mg/day of pimozide. Increments (0.5 mg haloperidol or 1 mg pimozide) may be added at 7 to 14 days intervals, up to 1–4 mg/day for haloperidol and 2–8 mg/day for pimozide. Atypical antipsychotics have also been used for the treatment of tics, and differences in efficacy appear to be related to their relative potency of dopamine blockade. Risperidone has been shown to be superior to placebo,[93,94] and equally effective to pimozide,[95,96] at doses ranging from 1 to 3 mg/day (starting dose, 0.25–0.50 mg). The specific D2 receptor-blocking agents, tiapride and sulpiride, have been commonly used for the treatment of tics in Europe in doses ranging from 15–500 mg/day and 200–1 000 mg/day, respectively, but they are not available in the U.S. The use of traditional antipsychotics is limited by a range of side effects, both in the short term (parkinsonism, dystonia, dyskinesia, and akathisia) and in the long term (tardive dyskinesia). The newer antipsychotics appear to have a lower frequency of neurological side effects in the short term, and a lower relative risk of tardive dyskinesia, but weight gain, hyperlipidemia and diabetes are of growing concern. Among the antipsychotics used in the treatment of tics, pimozide is the most likely to be associated with prolonged QTc interval, although this is a rare occurrence at therapeutic doses. An ECG is recommended before starting treatment, during the dose-adjustment phase, and annually during ongoing treatment. Patients should also be informed that the risk for cardiac conduction abnormalities may increase when pimozide is combined with drugs that inhibit cytochrome P450 3A4 isoenzyme (e.g. macrolide antibiotics, SSRIs, etc.).[97]

Clonidine is an antihypertensive agent (α-2-adrenergic agonist) that has been shown effective for treatment of tic disorders, presumably via acute and chronic downstream effects on dopamine. Clinical trials indicate an average 25–35 per cent reduction in symptoms over 8 to 12 weeks. Clonidine seems especially useful in improving attention problems and ameliorating complex motor tics. Treatment must be started at a low dose (0.05 mg in the morning), and slowly increased to 0.15–0.30 mg per day, given in several doses throughout the day. The major side effects are sedation, hypotension, dizziness, and a decrease of salivatory flow; blood pressure and pulse should be measured at baseline and monitored during dose adjustment, and patients and families should be educated about the potential for rebound increases in blood pressure, tics, and anxiety upon abrupt discontinuation.[98] Guanfacine is another α-adrenergic antihypertensive that has entered into clinical practice. Given the added disability attributable to ADHD in children and adolescents with tic disorders, a treatment combining an α-2 agonist and a stimulant may produce better outcomes than either alone. Recent studies suggest that the acute onset or worsening of tic symptoms among patients receiving stimulants may be simply an expression of the spontaneous time course of tics and comorbid ADHD.[99] Atomoxetine is a selective norepinephrine reuptake inhibitor that reduces significantly ADHD symptoms, and may also improve tics.

Management

As described above, OCD and tic disorders are frequently chronic, and most treatments, notably medication, are suspensive but not curative. Therefore, when defining a treatment plan, clinicians should be aware that they embark on a long-lasting task.

CBT is generally favoured as the initial treatment of choice for OCD, especially in milder cases without significant comorbidity, whereas presence of comorbid depression, anxiety, disruptive behaviour, or insufficient cognitive or emotional ability to cooperate in CBT, are indications for including an SRI in the initial treatment. However, youth who have OCD and comorbid conditions may not be as responsive to SSRIs for OCD, as shown in Geller *et al.*[100] study, in which the response rate was 75 per cent in the non-comorbid OCD group, but significantly lower when OCD was comorbid with ADHD (56 per cent), tic disorder (53 per cent), or oppositional defiant disorder (39 per cent); comorbid OCD may also be more vulnerable to relapse with SSRI discontinuation. Although the SSRIs are indicated for OCD, depression, and anxiety disorders, which make them an ideal first drug teatment when OCD is comorbid with an affective disorder, monitoring for the emergence of manic symptoms is required. The treatment of OCD comorbid with ADHD using stimulants may present a challenge because theoretical concerns exist that stimulants may increase obsessional symptoms. However, it is common clinical practice to combine a SSRI (or CMI) with a psychostimulant.[101]

Similar to adult patients, at least one third of young people with OCD prove refractory to treatment, and many 'responders' exhibit only partial response.[11] For children and adolescents who do not seem to benefit from SRI treatment, the first steps are (i) to reevaluate the diagnosis and associated features, and (ii) to review the adequacy of the dose, duration, and compliance with medication. Then, for many youth with a partial response to pharmacotherapy, further improvement may be obtained by adding a concurrent CBT intervention. Furthermore, if a first SSRI trial fails to produce an adequate response, pharmacological algorithms generally recommend switching to a second SSRI. Considering that these drugs all inhibit the 5-HT transporter, the first drug can be discontinued abruptly, and the second initiated at a dose in the middle of its therapeutic range. Because of its longer half-life, if fluoxetine has been the first treatment, it could be stopped abruptly, but the second SSRI must be titrated slowly. If two or three successive trials of SSRIs have failed, clomipramine is generally considered as the next option. Given that it has a half-life in the same range as most of the other SRIs, no time should be wasted in the substitution, unless a switch from fluoxetine is carried out.

Very few drug augmentation or combination strategies have been tested for youth with treatment-resistant OCD. The addition of risperidone to various SRI agents (clomipramine, sertraline, fluoxetine, paroxetine) has been reported in a series of OCD adolescents

with no comorbid tic disorder, with only modest benefits.[102] The combination of an SSRI with clomipramine takes advantage of the pharmacokinetic and pharmacodynamic interactions of these medications, but it is important to monitor for adverse effects, particularly cardiovascular side effects, and the possible emergence of a serotonin toxic syndrome. Combining two SSRIs is a common clinical practice, despite the risk for drug interaction, since all SSRIs inhibit cytochrome P-450, and combinations may result in increased blood levels of each SSRI.

If short-term treatment with a SSRI often leaves OCD youth with residual symptoms, over an extended period of SSRI treatment, they may experience greater improvement. Three long-term (1–2 years) open studies have documented continued improvement after the acute phase, but at a much slower rate, with fluvoxamine, sertraline, and citalopram. Similarly, in two continuation studies of clomipramine (4–12 months), treatment continued to be effective and well tolerated. There are hardly any data on the doses of SRIs that should be used in treatment prolongation, versus those used in the acute treatment phase. It would thus be prudent to maintain the regimen that produced the maximal improvement in the acute treatment. The most recent guidelines for adult patients recommend a minimum treatment of one to two years, followed by a gradual taper to, first, avoid discontinuation phenomena and, second, monitor patients for a possible deterioration.[103] In youth, it is particularly recommended to choose a period free of stress (e.g. summer vacation) for tapering medication, and to provide alternative psychological support (CBT, education on relapse risk and management) for the period of discontinuation.

Although tics are a common childhood problem, only a small minority of cases find their way to clinics. Given the waxing and waning nature of tic disorders, usual therapeutic practice will initially focus on careful clinical observation, along with educational and supportive interventions, and pharmacological treatments are held in reserve. The decision about whether and how to treat will depend on the primary diagnosis, and the degree of interference with the child's development and functioning. Most simple tics occurring in the absence of severe functioning impairment respond to a simple explanation of the mechanisms. In case of comorbidity with a mood and anxiety disorder, it is not uncommon to see improvement in tic severity after successful treatment of the affective disorder with a SSRI.[104] When tics are responsible of functional impairment, the decision to use medication follows careful assessment and identification of target symptoms, that are interfering in the patient's quality of life. The selection of medication is based on a balance of risks and benefits, and in order not to expose the subject to excessive unwanted side effects, pharmacological treatment should not aim at complete disappearance of tics. For tics of moderate severity, clonidine or guanfacine may be considered as the first line treatment given their safety margin, and an expected 30 per cent decrease in tic severity may be sufficient. For tics in the marked or severe range, however, more potent medications, such as antipsychotics, that decrease tics severity by 35–60 per cent should be considered, despite the increased risk of adverse effects. In addition, patients with tic disorders and their families should be cautioned about both licit and illicit drug use, since sympathomimetic agents ranging from decongestants through speed and cocaine, markedly exacerbate tics.

For children and adolescents with OCD and comorbid tic disorder, the SSRIs alone might have little anti-obsessional effect, and there are reports suggesting that fluvoxamine and fluoxetine may exacerbate or even induce tics in some patients. The adult literature, and a few case reports of children and youth with OCD and comorbid tic disorder suggest that combined treatment with a SRI and a low dose of risperidone or another atypical antipsychotic is a reasonable option.[105] Using data from the POTS study, March *et al.*[106] found that tic disorders appeared to adversely impact the outcome of medication management of paediatric OCD, in contrast to CBT outcomes which were not differentially impacted.

Therapeutically, the finding of possible autoimmune cases of OCD and tic disorders raises the clinical possibility that immunosuppressant treatments might be effective, and a few experimental studies have been conducted. A double-blind, placebo-controlled study in resistant cases of PANDAS supported the efficacy of both plasma exchange and intravenous immunoglobulin compared to a sham condition at one month, and the effects were maintained after 1 year for 82 per cent of subjects with follow-up assessment.[107] In another study antibiotic prophylaxis with penicillin or azithromicin was administered for 12 months to a group of children with PANDAS; compared to the year prior to entry in the study, significant decreases were observed in the number of streptococcal infections, and the number of neuropsychiatric exacerbations with both prophylactic treatments.[108] There is preliminary evidence for the efficacy of CBT in OCD cases of the PANDAS phenotype.[109] However, it is still unknown what percentage of children with OCD may be part of the PANDAS subgroup, and neither immunosuppressant nor antibiotic treatments are to be used for such cases out of the context of board-approved research protocols. Nevertheless, children with abrupt onset or exacerbation of OC and/or tic symptoms require careful consideration of medical illnesses including upper respiratory tract infections during the preceding months, to be promptly treated if present. A throat culture and antistreptolysin O or antistreptococcal DNAase B titers may be considered to assist in diagnosing a GABHS infection.[101]

Even if they are less efficacious alone, other treatment modalities should not be neglected. The effectiveness of psychotherapy per se—apart from behavioural and cognitive interventions—on OCD and tic disorders has not been demonstrated, but the symptoms may have a profound impact on the life of subjects affected, and traditional psychotherapeutic approaches may be useful to help children and adolescents address the intrapsychic conflicts that may affect or result from their illness. Some families become extensively involved in participating in compulsive rituals or reassuring obsessional worries, others become mired in gruelling angry struggles with their symptomatic child. Work with families on how to manage the child's symptoms, cope with the stress and family disruption that often accompanies OCD and tic disorders, and participate effectively in behavioural or pharmacological treatment is crucial. Most cognitive behavioural approaches of paediatric OCD include the involvement of a parent in some therapeutic sessions, and it is noteworthy that the Barrett *et al.*[68] study, which actively involves the family in the child's treatment, reports improvement in the highest range of outcomes among CBT studies. In children with tic disorders, oppositional, defiant, and disruptive behaviours are common, and parenting skill training may be an important adjunctive to treatment. For cases of very incapacitating OCD, there is some empirical evidence that milieu therapy in an inpatient setting may be a useful resource. Finally, the growing availability, in many countries, of family support and

advocacy groups for patients with OCD and tic disorders may be most useful to alleviate the discouragement and incomprehension created by these disorders, and give access to appropriate treatment resources.

Possibilities for prevention

At present, there is no known preventive strategy individually targeted at either OCD or tic disorders. However, early intervention and comprehensive treatment, as long as needed, is certainly the best way to prevent severe incapacitation, and achieve complete recovery in some cases. Even when response to successive treatment efforts is less than optimal, the improvement in function and quality of life may be considerable. In families with one or several cases of OCD or tic disorders, clinicians should be attentive to the onset of similar symptoms in children and siblings, and treat cases of newly occurring disorder early and vigorously. Although this might concern only a fraction of patients, evidence of the onset or exacerbation of OCD or tics associated with streptococcal exposure warrants standard antibiotic treatment and ongoing monitoring for recurrent infection.

Conclusion

Paediatric OCD is the disorder, in child psychiatry, whose clinical picture most closely resembles its adult counterpart. Despite a relative diversity, the symptom pool is remarkably finite, and very similar to that seen in older individuals. Prevalence, comorbidity, and response to behavioural and drug treatment also appear similar across the lifespan. For tic disorders, there is continuity between child and adult presentations, but the disease is much more prone to resolve spontaneously, or to be less disruptive in adulthood. Both OCD and tics occur more often in males than in females, and are likely to be linked to an array of neurobiological abnormalities, many of which remain to be understood.

Invaluable benefits can now be obtained from available behavioural and pharmacological treatments, but complete remission remains uncertain and long-term management may be required. Thus, the treatment of OCD and tics in children and adolescents remains a clinical challenge. It requires careful assessment of the targeted symptoms and, in many cases, comorbidity; attention to the quality of the child's functioning at home and with peers; use of specific CBT interventions, which are not readily available (or accessible) in all communities; patience and caution in the choice and adjustment of medication; and vigilance in watching potential side effects. Given the possible chronicity of OCD and/or tic disorders, and their changing patterns in severity and impact over the childhood and adolescent years, optimal treatment generally requires a long-term ongoing relationship with the child and family.

Current conceptualizations of OCD and tic disorders have been shaped by advances in systems neuroscience and functional in vivo neuroimaging. Continued success in these areas should lead to the targetting of specific brain circuits for more intensive research. This should include testing novel pharmacological agents, tracking treatment response using neuroimaging techniques, and possibly investigating circuit-based therapies using deep-brain stimulation for refractory cases. The identification of the PANDAS subgroup of patients, with an abrupt onset and dramatic exacerbations, certainly brings new insights into the pathophysiology of OCD and tic disorders, and may lead to new assessment and treatment strategies. The increasing evidence for susceptibility genes in OCD and tic disorders will also doubtless point to new therapeutic directions. Furthermore, it is likely that many of the empirical findings used in research on paediatric OCD and tic disorders will be relevant to a better understanding of both normal development, and other disorders of childhood onset.

Further information

Obsessive Compulsive Foundation. Available at: http://www.ocfoundation.org

Tourette Syndrome Association. Available at: http://www.tsa-usa.org/index.html

Tourette syndrome online. Available at: http://www.tourette-syndrome.com/default.htm

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9.2.9 Sleep disorders in children and adolescents

Gregory Stores

Introduction

It was argued in Chapter 4.14.1 that sleep disorders medicine should be viewed as an integral part of psychiatry, whatever the age group of patients, because of the various close connections between sleep disturbance and psychological disorders seen in clinical practice. This is certainly the case regarding child and adolescent psychiatry in view of the high rates of psychiatric disorder of which sleep disturbance is often a part, and also the frequent occurrence of sleep disorders in young people with potentially serious developmental effects of a psychological and sometimes physical nature. The temptation to view children's sleep disorders as merely transitory problems, mainly in infancy, encountered by many parents and of no lasting or serious significance, should be resisted. This may be true for some families but is frequently not the case in others.

The following account summarizes sleep disorders in childhood and adolescence. *Familiarity is assumed with the earlier accounts of sleep disorders in adults (4.14.1), including the introduction to that section which covers basic aspects of sleep and other fundamental issues.*

Sleep and sleep disorders in children compared to adults

In spite of the fact that much has now been discovered about the special characteristics of sleep disorders occurring at an early age, very little of this information has found its way into the training of paediatricians, child and adolescent psychiatrists, psychologists, or other professionals involved in the care of children. This must mean that many treatment and preventive possibilities are missed.

The solution does not lie simply in extending the practice of adult sleep disorders medicine to children. Children are not miniature adults and they need special approaches reflecting their many differences from older patients. These differences extend from basic features of sleep to various aspects of sleep disorders.

Sleep physiology

Profound changes take place during childhood in basic sleep physiology although many are complete by about 6–12 months of age. In general, there is a progression towards differentiation and organization of conventionally defined sleep states, shorter sleep time, less napping, less slow wave sleep (**SWS**), and longer sleep cycles.

Specific aspects of particular clinical importance are as follows:

- Typical sleep duration (including naps) at different ages as shown in Table 9.2.9.1.

Table 9.2.9.1 Average sleep requirements at different ages

Term birth	17 h
1 year	14 h
2 years	13 h
4 years	12 h
10 years	10 h
Adolescence	9 h plus*

*Many adolescents are thought to obtain far less sleep than this.

- The body clock controlling (amongst other processes) the circadian sleep–wake cycle has become established by about 6 months.
- Rapid eye movement (**REM**) sleep is prominent in early infancy, perhaps reflecting its role in brain maturation and early learning, and possibly explaining why sleep is fragile at this stage.
- In comparison, by early childhood **SWS** is especially pronounced. This predisposes children of that age to arousal disorders (e.g. sleepwalking) which arise from **SWS**.
- Between about 5 years and puberty, overnight sleep is especially sound and alertness is maximal during the day. Various conditions causing excessive daytime sleepiness in adults (e.g. narcolepsy) may not have this effect in children because of this increased alertness. However, overnight sleep may become extended.
- In contrast, adolescence is characterized by an increase in daytime sleepiness. The amount of **SWS** decreases, the sleep phase is physiologically delayed and, with the onset of puberty, there is no longer the decrease in physiological sleep requirements seen progressively at earlier ages. The combination of these factors and strong influences to stay up late (especially at weekends but perhaps also during the week) for social and recreational purposes frequently causes unsatisfactory sleep–wake patterns.

Parental influences

The influence of parents is seen throughout children's sleep disorders medicine.

- Especially in the case of young children, parents' perceptions usually determine whether there is a sleep problem. The same sleep pattern or behaviour may be a problem to one family but not another. Factors influencing parental attitudes include their expectations, family and cultural practices (e.g. regarding parents and children sleeping together), and their own emotional state. Sometimes parents can be reassured that what they think is a serious problem about their child's sleep is, in fact, within the normal range. The view taken of the situation might be the result of parental psychiatric illness needing attention in its own right; children of mothers with an affective illness have been shown to have an increased rate and severity of sleep problems although the nature of the connection is debatable.
- Conversely, parents may not seek help for their child's sleep when they ought to do. They may be unaware of the problem, indifferent, or they may mistakenly believe that the child's sleep problem is inevitable and untreatable. This mistaken view is sometimes expressed by parents of children with a learning disability (intellectual disability) whose sleep problems can be particularly severe yet amenable to treatment.
- Parental practices are commonly the reason why a child's sleep problem develops or is maintained. Early child-rearing practices determine sleep–wake patterns which can be delayed or disrupted by over-conscientious night-time feeding in infancy, failure to set limits on bedtime activities, or inconsistency (see later). Sleep disorders of physical origin may be complicated in these ways and exacerbated. It follows that treatment of many sleep disorders relies heavily on correcting parenting practices.
- Sometimes parents are not motivated to improve their child's sleep for reasons that may be difficult to influence. For example, a child's presence in the parental bed may be welcome by one partner as a means of distancing himself or herself from the other at night. Families of handicapped children may lose their extra state financial allowance if their child's sleep problems are successfully treated.
- The child's basic attitude to sleeping is also influenced by its parents. Wider cultural factors are important but, within westernized societies, the child's attitudes to going to sleep and being separated from its parents at night are strongly influenced by their ability to settle the child without being anxious about the separation. Children depend on their parents to provide positive attitudes to sleeping and to avoid negative associations such as disputes, punishment, and rejection.
- Especially in the early years, most children need their parents' help in coping with night-time separation from them, and the potentially frightening experience of the dark or their own thoughts and fantasies. Infants need the comfort of physical contact. Toddlers are helped by bedtime routines and comforting 'transitional objects', and encouragement to become 'self-soothing' so that they can fall asleep without their parents' presence and attention (see later). Parents' ability to provide such help depends on their personality and sensitivity and mental state and perhaps their cognitions about their child's sleep based partly on their own experiences in childhood. Hopefully, older children and adolescents become increasingly independent.

Effects on parenting and the family

The effects of a child's persistent sleep disturbance on family life, including its possible influence on parenting skills, is another important dimension.

- Mothers of children with a learning disability and severe sleep problems are reported to be more irritable, concerned about their own health, and less affectionate towards their children, with less control and increased use of punishment compared with mothers of such children without sleep problems. Similarly, associations have also been suggested between sleeplessness in toddlers in the general population and family problems, including marital discord and possibly physical abuse of the child.
- Family tensions are likely to increase when diagnosis of the child's sleep disorder is delayed or inaccurate, or when effective treatment is not provided.
- Some reports have suggested that successful treatment of the child's sleep problems generally leads to improvement in the

mother's mental state, confidence in her own parenting ability, her relationship with the child, and also the child's behaviour. Wider aspects of family function, including effects on siblings, have received little attention.

Developmental effects of sleep disturbance

- These parental and wider family issues indicate ways in which a child's psychological and social development can be affected by persistent sleep disturbance. In addition, children can be distressed by their experience of sleep disorder phenomena. Examples include night-time fears (which may be intense) alarming hypnagogic imagery, or sleepwalking and sleep (night) terrors which can be embarrassing, especially if they occur away from home. Excessive daytime sleepiness often leads to educational problems and can produce extreme reactions such as the denial, aggression or depression described in narcolepsy, or accidents and substance abuse in adolescence.
- In addition to these largely indirect ways in which a child's sleep disorder may have psychological effects, sleep disturbance can produce direct effects on mood, behaviour, and cognitive function. The developmental consequences might become severe if not arrested at an early age.
- Adolescents appear to be at particular risk of sleep loss and its possible psychological consequences, i.e. depressed mood, anxiety, behaviour problems, alcohol abuse, and even attempted suicide, as well as lower academic performance. The causal relationship between sleep loss and these problems, however, have yet to be fully established. The same is true of the outcome of attempts to correct this sleep loss by various means.
- Even impairment of physical growth is associated with sleep disturbance. Failure to thrive is a recognized possible consequence of early onset obstructive sleep apnoea (**OSA**) and possibly other severe and persistent sleep disturbance, perhaps as a result of reduced slow wave sleep (**SWS**) with which the production of growth hormone is closely linked.
- Other possible physical consequences of sleep disruption includes impaired immunity and endocrine disorders.

Patterns of occurrence of sleep disorders

- Some sleep behaviours which are developmentally usual in children are abnormal in adults and require investigation. Examples are bedwetting and repeated napping. Certain sleep disorders are seen exclusively in children (e.g. sleeplessness caused by infantile colic). Others, such as settling problems and confusion arousals, occur primarily in children (see later).
- Sleeplessness caused mainly by child-rearing practices is particularly common in early childhood. That attributable to the delayed sleep phase syndrome (see later) is considered to be particularly common in adolescence.
- Many of the parasomnias (such as headbanging, sleepwalking, or sleep terrors) are more common in childhood where, generally, they represent a temporary developmental phase without pathological significance. The same behaviours in adults might be more likely to be manifestations of psychological problems requiring exploration.
- Some sleep disorders thought to be confined to adulthood are now recognized in children. While much attention has been paid to OSA in adults, it is now thought that at least 2 per cent of children have this condition to some degree. Restless leg syndrome (**RLS**) and periodic limb movements in sleep (**PLMS**) are now known to occur not uncommonly in children. The RLS may explain some cases of 'growing pains'. PLMS has been implicated as a cause of poor quality sleep resulting (as in other forms of sleep disturbance) with daytime attention deficit hyperactivity disorder (**ADHD**) type of symptoms. Narcolepsy starts by the age of 15 years in at least one-third of cases. Even REM sleep behaviour disorder (once thought to be confined to elderly males), or something similar, has been reported in children and adolescents.

Manifestations of sleep disorders

- The clinical features of basically the same sleep disorder can be very different in children compared with older people. The overall behavioural effects of excessive sleepiness in adults are a reduction of physical and mental activity. In contrast, its effects in young children can be increased activity with irritability, tantrums, or other behavioural difficulties. Some examples of ADHD are thought to be the result of sleep disorders (OSA, PLMS, or circadian sleep–wake rhythm disorder) with improvement in the difficult behaviour following treatment of the sleep disorder.
- OSA illustrates the important differences between children and adults, not only in the clinical manifestations of a particular sleep disorder but also in the underlying cause and treatment needs. Similarly the many manifestations of narcolepsy in childhood may be very far removed from the classical narcolepsy syndrome in adults, at least in its fully developed form. The same sleep disorder may also show different physiological features according to age. Diagnostic criteria (e.g. for OSA and narcolepsy) derived from polysomnographic (PSG) studies in adults do not necessarily apply in children and may well need modification.

Misinterpretation of children's sleep disorders

Chapter 4.14.1 contains an account of the fundamental issue that, especially if clinicians are unfamiliar with the manifestations and consequences of the many sleep disorders now documented in the second edition of the International Classification of Sleep Disorders (ICSD-2), there is a serious risk that these disorders will be misconstrued as something else (or even overlooked completely). The examples given include a number of particular relevance to practice in child psychiatry and paediatrics.[1]

Treatment and prognosis

- Because of the aetiological differences discussed earlier, especially parental involvement, treatment often needs to be very different in children compared to adults. Appropriate behavioural approaches usually entail alterations to parenting practices designed to be acceptable and feasible in each individual family. Other forms of treatment, including chronobiological measures (such as adjustment of sleep schedules from the delayed sleep phase syndrome in adolescence) usually require considerable parental involvement. The same is true of the general sleep

hygiene principles described in Chapter 4.14.2. Explanation and (where appropriate) reassurance for the child and parents is an essential part of any treatment and may be effective in their own right without the need for more specific measures. As in adults, medication has a limited part to play overall.

- An optimistic point of view can be taken of the treatment of most children's sleep disorders because children's sleep is usually more amenable to change than that of adults where the factors underlying the sleep problem may well have become well established and complicated, as in many cases of chronic insomnia. However, treatment needs to be chosen carefully and implemented properly, and parents' confidence in the recommended measures, and their willingness and ability to play their part in treatment, are an important determinant of success or failure. In some instances, it is not possible to implement a treatment programme for the child until parents themselves have been helped (e.g. by treatment for a depressive illness) or problems in the family as a whole have been resolved.

Assessment

The various means by which sleep disorders might generally be detected and assessed are described in Chapter 4.14.1. These subjective and objective approaches need to be modified for use with children because of the involvement of parents, developmental factors, and the differences between children and adults regarding clinical manifestations and diagnostic criteria.

The detection of sleep problems can be improved by routinely asking basic screening questions as part of the history-taking in any child:

- Does the child have difficulty getting to sleep or staying asleep?
- Is there excessive sleepiness during the day?
- Are there episodes of abnormal behaviour or experiences at night?

Positive answers to any of these questions call for a detailed sleep history.

Sleep history and general review

This is the cornerstone of sleep assessment. Unfortunately, history-taking schedules are usually perfunctory in the attention they pay to sleep and its possible disorders. Parents and also the child (if old enough) should be interviewed and the reasons for any disparities considered. Sometimes sibs or teachers can provide important additional information. The main aspects that should be covered are as follows:

- Current sleep problems and their evolution.
- Past treatments and their effects.
- Review of the child's current 24 h sleep–wake cycle (see Table 9.2.9.2) in order to determine in particular
  (a) duration of sleep
  (b) quality of sleep (continuous or disrupted)
  (c) timing of sleep
  (d) features suggestive of specific sleep disorders (e.g. breathing difficulty or jerking limbs).
- Sleep environment and arrangements.
- Development of the child's sleep patterns and problems.

Table 9.2.9.2 Review of child's 24 h sleep–wake pattern (modified according to child's age)

Evening
Time of evening meal
Other evening activities
Going to bed
Preparation for bed, by whom
Time of going to bed
Reluctance to go at required time, parents' reactions
Fears, rituals
Wanting to sleep with someone, other comforts
Time taken to fall asleep, other experiences during that period
When asleep
Wakings, frequency, causes ability to return to sleep
Episodic events, exact nature, timing, frequency
Other behaviours during sleep, e.g. snoring, restlessness, bedwetting
Parents' reaction to night-time events
Waking
Wakes spontaneously or needs to be woken up
Time of final waking
Total duration of sleep period
Longest period of uninterrupted sleep
On waking: preoccupations, mood, feeling of being refreshed, other experiences
Difficulty getting out of bed, time of getting out of bed
Daytime
Sleepiness, naps
Lethargy
Mood
Overactivity
Concentration and performance
Other unusual episodes

- General review of possible sleep symptoms.
- Family history of sleep disorder or other conditions.

A **sleep questionnaire** completed by parents before the interview can provide a useful outline account of these and other aspects.[2]

Additional parts of the overall review of children with a sleep problem that are important in order to identify possible contributory factors are as follows:

- Developmental history including developmental delays, illnesses, or significant events at school or within the family.
- Review of physical health.
- Physical examination.
- Assessment of behaviour and emotional state.
- Family history and circumstances.

Following the initial consultation, a **sleep diary**, kept over a period of 2 or more weeks, can be particularly useful. This provides a more complete and balanced view than that obtained especially from fraught parents likely to give a distorted or unbalanced retrospective account.

Special investigations (see also Chapter 4.14.1)

These depend on the nature of the sleep problem:

- Indications for PSG are essentially the same as for adults.

- The use in children of multiple sleep latency tests (**MSLT**) as an objective measure of sleepiness is hampered by the absence of good normative data at different ages. However, in school-aged children, about 16–18 min to fall asleep is considered normal; less than this might indicate significant daytime sleepiness which is also indicated by falling asleep in three or more of the naps. Nevertheless, in the presence of sleep disorders usually characterized by excessive sleepiness, MSLT results can be normal in late childhood because of the naturally enhanced daytime wakefulness at that age.
- **Actigraphy**, which provides information unobtrusively on basic sleep–wake patterns, is well established for children of all ages.
- **Other possible measures** include toxic screening and the special tests mentioned earlier in Chapter 4.14.1.

Children at special risk of sleep disturbance

The prevalence of children's sleep disorders is not known with any accuracy, even for those which are severe and persistent. A number of methodological problems make it difficult to collect accurate figures and no really vigorous attempt has yet been made to overcome them. It seems that 20–30 per cent of children from infancy to adolescence have sleep problems that are considered significant by them or their parents.

The occurrence of sleep problems exceeds this overall rate considerably in various categories within the general population as a whole, and also in certain clinical subgroups. Determining the exact nature and cause of the sleep problems in these high risk groups (and also in any other affected children), with a view to successful treatment, is important because of the possible adverse effects on the child in a family that have just been discussed. Behavioural sleep problems probably predominate throughout these high risk groups but sleep disorders of a different nature may well be encountered instead or as well as those of behavioural origin.

Children in the general population

- Reference was made earlier to the fact that children in general appear to be particularly prone to different types of sleep disorders at certain ages of development i.e. early infancy, early childhood, and adolescence.
- Adverse psycho-social circumstances are also associated with increased risk of childhood sleep problems. The importance of such factors as the degree of organization in family life, parental concern, child-rearing practices, and the mental health of parents was stressed earlier. High rates of various sleep problems, as well as other psychological difficulties, have been reported in homeless children.

Children with psychiatric disorders

High rates of sleep disturbance have been described in child psychiatric groups in general compared with other children, and in specific psychiatric disorders.

- Various sleep problems, including panic attacks, have been described in **anxious children** in general including those with **panic disorders**. Similarly, many types of sleep problem (including nightmares and other disturbed nocturnal episodes, excessive daytime sleepiness, and bedwetting) have been reported to be particularly frequent in **traumatized children** including those who have suffered burn injury, abuse, or road traffic accidents. Treatment of the sleep disturbance has appeared to improve their emotional state but further research is needed to asses the therapeutic contribution of specific treatment for the sleep disorder as part of the overall care of the traumatized children.
- Difficulty in sleeping is the main complaint in children and in adolescents with severe **depressive disorders** but many complain of excessive sleepiness, possibly because of difficulty getting to sleep and/or poor quality sleep.
- Parental reports of sleep problems in children with **ADHD** are very common. Parental impressions can be distorted but preliminary objective evidence also suggests that persistent sleep disturbance is common and sometimes important as the primary cause (or a significant contributory factor) rather than simply a consequence of ADHD. It was mentioned earlier that ADHD symptoms have sometimes been attributed to definitive sleep disorders in which sleep quality is impaired, with improvement in ADHD symptoms following treatment of the sleep disorder. Preliminary studies of sleep physiology or other objective aspects of sleep in children have also produced evidence of sleep abnormalities. Even where ADHD is attributable to other factors, sleep disruption is likely to worsen a child's behaviour, meriting treatment in its own right wherever possible.
- **Other psychiatric disorders** in which different types of sleep disturbance is reported to be prominent are autism (including circadian sleep–wake rhythm disorders and Asperger's syndrome, tic disorders including Tourette syndrome (sleeplessness and parasomnias), and obsessive–compulsive disorders (poor quality sleep).

  Sleep complaints are also prominent in the chronic fatigue syndrome. As mentioned earlier, disruption by frequent awakenings (not obviously attributable to daytime inactivity) has been described in teenagers with this condition suggesting that daytime symptoms might be at least partly attributable to poor quality sleep. Occasionally Munchausen's syndrome by proxy have come to light in the form of parental complaints of a sleep disturbance. Reports of the sleep of conduct disordered children are in keeping with the expectation that their sleep is disturbed because of their adverse or disorganized home and social circumstances, and general way of life.
- Apart from psychiatric disorders themselves, **medications** used in their treatment may affect sleep. Stimulant medication for ADHD appears to cause sleeping difficulties in some children but some children with ADHD may settle to sleep more readily even if their medication is given later in the day because this improves their bedtime behaviour. See Chapter 4.14.1 for other possible medication effects on sleep.

Children with a learning disability or other neurological disorder

Particularly high rates of sleep disturbance has been consistently reported in children with a **learning disability**. The disturbance is often severe, poorly managed and, therefore, persistent.

- Sleep problems in this group are often behavioural in origin (largely attributed to parenting practices), arising from often

understandable over-permissiveness, inconsistency, or parents' inability to set limits on their child's behaviour because of their own emotional state or excessive demands on their time. Other physical sleep problems include some chronic physical conditions. OSA features prominently in various specific learning difficulty conditions such as Down syndrome, the mucopolysaccharidosis and fragile X syndrome. Epilepsy can also play an important role as well as other co-morbid conditions.

- Sleep problems are also widely reported in children with **neurodegenerative disorders**, e.g. Rett's syndrome and other neurological disorders such as head injury. Again, behavioural factors might be partly the reason, although interference with sleep mechanisms also seems likely, at least in the advanced stages of the disease.

Children with other chronic physical illness

Acute physical illnesses disturb sleep but only for the duration of the illness in most cases. By comparison, chronic illnesses are commonly complicated by long-standing sleep disturbance caused in various ways (see Chapter 4.14.1 for medical causes of sleep disorder some of which apply to paediatric cases).

Main sleep problems: sleeplessness

The second edition of the International Classification of Sleep Disorders was outlined in Chapter 4.14.1. The following selective account of sleep disorders in children and adolescents is organized according to the three main types of sleep complaint: sleeplessness, excessive sleepiness, and the parasomnias. Childhood psychiatric and medical conditions in which sleep disturbance is a prominent feature has already been mentioned. Emphasis is placed on the differential diagnosis of sleep complaints and also on points of particular relevance to psychiatric practice.

The breakdown of problems and disorders according to age should not be interpreted too strictly as there is overlap between the different age groups. In addition to specific treatments mentioned for particular sleep disorders, the promotion of adequate sleep, regular sleep habits, and the other sleep hygiene principles referred to in Chapter 4.14.1 are important. Evidence for the effectiveness of psychological treatments for sleeplessness in children is reviewed in detail elsewhere.[3]

Infants

Ways of preventing or dealing with babies' sleep problems are rarely taught to parents or prospective parents, with the result that many suffer needless sleep loss and distress because the child does not sleep well. It is important to encourage good sleep habits from the start to avoid bad sleep habits later on. There are certain general guidelines for achieving this, admitting that babies vary temperamentally in their response to recommendations and parents vary in their ability to adhere to them. The main basic principles are as follows:

- Establishing a consistent 24 h routine, including a bedtime routine that provides cues that is timed to go to sleep.
- Not prolonging night-time feeding beyond the age (about 6 months) when the baby's body clock has developed enough to confine feeding to daytime.
- Teaching the baby to fall asleep alone so that when he or she wakes in the night (a natural occurrence at all ages) it will be possible to fall asleep again without requiring parental attention ('**self-soothing**').
- Establishing a clear difference in the infant's experience between day and night to help to develop his/her body clock which controls sleep and wakefulness.
- Ensuring the environment is conducive to sleep.

Safety measures to reduce the risk of the infant coming to harm at night from suffocation, or other breathing problems associated with sudden infant death syndrome (**SIDS**), should also be part of parent education about sleep.[4] Main recommendations are: having the infant sleep on his/her back and on a firm mattress that will not obstruct breathing, ensuring that his or her face cannot be covered during the night, ensuring the bedroom is smoke free, and avoiding co-sleeping if either parent has consumed alcohol or has taken medication or other substances with a sedative affect. Also, the baby should not be overheated at night.

Toddlers and pre-school children

About 30 per cent of children of this age present a problem of recurrently not going to bed at the required time, and/or waking repeatedly at night and demanding their parents' attention including coming into their bed. Medical factors must be excluded but the usual explanations are behavioural especially:

- Anxiety about separating from parents at night
- Unhelpful associations with going to bed, e.g. stimulating activities within the bedroom, threats, or recriminations
- Inadequate limit setting on bedtime or night-time behaviour
- Failure to require self-soothing ways of coping with night waking.

Behavioural methods of treating these problems can be very effective, even in severe and long-standing cases, including children with developmental disorders such as learning disability or autism, providing the treatment programme is implemented properly. The main methods used include graded changes and desensitization rather than leaving the child to cry (a quickly effective measure but one which is unacceptable to many parents).

School-age children

Some of the causes of sleeplessness in pre-school children still apply in older children but other factors become more relevant with increasing age:

- **Night-time fears** are common from very early childhood onwards although, in keeping with cognitive development, the content of the fears changes from aspects of the immediate environment (e.g. shadows or noises) through imaginary objects (ghosts, monsters) or the dark, to more realistic and specific fears concerning the child's own health. Such fears are usually transient and require only reassurance and comfort until they cease.

  In some children the fears are so intense and persistent that they reach phobic proportions and need special attention. The cause of the fear should be investigated. The night-time fear might be one aspect of an anxiety state, including post-traumatic stress disorder in which case the child might also suffer from nightmares.

The content of the fear or nightmare might be revealing, suggesting abuse, for example. Other sleep disturbances (e.g. alarming hypnagogic hallucinations) may be the cause of the night-time fears. The child's reluctance to go to bed because he or she is genuinely afraid must be distinguished from pretending to be afraid as a delaying tactic.

Behavioural treatment is said to be very effective in cases of severe night-time fears. The child with night-time fears should be helped by positive associations with bedtime and by not going to bed so early that he or she lies awake in a fearful state.

- Even without night-time fears, a child will be unable to settle to sleep if **bedtime is too early**. Like some adults and even other species, children often have an evening period of intense wakefulness and activity before they begin to relax in preparation for sleep. A child is physiologically unable to sleep if put to bed in this 'forbidden zone'. Instead the sequence of events leading up to bedtime should be arranged so that the child goes to bed when 'sleepy tired'.
- **Worry and anxiety** about daytime matters such as school progress may cause difficulty in getting to sleep or staying asleep. The original source of concern may no longer exist but the difficulty falling asleep may persist because the child has developed the habit of lying awake in bed in an agitated state ('conditioned insomnia'). Sympathetic discussion of the child's worries, attention to the source of concern if possible, and ways of helping the child to relax at night, are generally thought to help. More specific psychiatric measures will be needed if the child has an anxiety or depressive disorder, or if there is evidence of serious problems within the family.
- '**Childhood onset insomnia**' or **'idiopathic insomnia'** refers to a lifelong difficulty sleeping not attributable to environmental, emotional, or medical factors and therefore of constitutional origin. The condition is usually diagnosed retrospectively in adult life.
- **Early morning waking** (i.e. when a child habitually wakes very early, does not return to sleep, and is noisy or demands attention) can be very distressing to parents, and disruptive to the whole family. In pre-school children, early waking may be the result of excessive or otherwise inappropriate napping, but at a later age the problem may be part of the advanced sleep phase syndrome. In this disorder the child's bedtime and sleep onset is so early that his or her sleep requirements have been met well before other members of the family wake in the morning. Gradual resetting of the child's sleep onset time is required.

  In older children and adolescents early morning wakening may be part of an anxiety or depressive disorder. Otherwise, the child may have been woken too early by noise or other environmental factors which intrude into his or her sleep.

Adolescents

High rates of insomnia have been consistently reported in adolescents. The change from the highly efficient sleep of pre-pubertal children to less satisfactory sleep in adolescence was mentioned earlier, including the biological influences in this change. The psychological and social demands and stresses of adolescence further conspire to disrupt sleep patterns. Worries, anxiety, and depression are commonly quoted reasons for not being able to sleep at this age. Nicotine, alcohol, and caffeine-containing drinks, as well as illicit drug use, are additional possible influences.

Difficulty getting off to sleep is often part of the delayed sleep phase syndrome which is reported to be particularly common in adolescence. In this condition (which will be considered further in relation to excessive sleepiness as this is often the major complaint) there is a physiological inability to go to sleep until much later than the required time because of a shift in the sleep phase. The adolescent's reluctance to go to bed earlier (or the bedtime struggles of parents with younger children with this disorder) are often misinterpreted as 'difficult' behaviour. Instead of recriminations and attempts to set limits, the timing of the sleep phase needs to be reset by so-called chronotherapeutic means.

Main sleep problems: excessive daytime sleepiness

Despite the evidence that it is a common problem,[5] sleepiness remains neglected in child and adolescent psychiatry and in paediatrics:

- Part of the explanation is that sleepiness is not usually viewed as a medical problem by parents, teachers, and children themselves, the symptoms being misperceived as laziness or disinterest. Otherwise, they may be interpreted as depression or even limited intelligence.
- Another difficulty is that excessive sleepiness can take various forms including prolonged overnight sleep or inappropriate periods of sleep during the day.
- Extreme degrees of sleepiness will cause a reduction of activity at any age, but lesser degrees in children may produce irritability, over-activity, restlessness, poor concentration, impulsiveness, or aggression. Explanations of such behaviours other than sleep loss or disturbance (such as ADHD) are more likely to be considered, as mentioned earlier.
- A high level of daytime alertness in older pre-pubertal children may be sufficient to offset a tendency to sleep, providing a different clinical picture of sleepiness but not seen at a younger or older age.

Because of these problems of recognition, the prevalence of excessive sleep in children and adolescents is not known. Clearly, it is not rare in view of the range of underlying conditions causing it, many of which are individually quite common.

It is important to establish that the problem really is excessive sleepiness. 'Tiredness' is an ambiguous term: ideally, sleepiness should be distinguished from fatigue or lethargy, without necessarily the need to sleep, for which different explanations are likely including physical illnesses, such as anaemia or endocrine disorders in which other signs are usually present. Occasionally, excessive sleepiness with long periods in bed or at home is simulated in order to escape from a difficult situation. Detection of such cases requires very careful clinical evaluation and assessment and possibly PSG.

Excessive sleepiness is mainly a problem in older children and (especially) adolescents. Many teenagers complain about excessive sleepiness but it has been claimed that very many more than those who seek help are likely to be suffering from chronic sleep deprivation

or '**sleep debt**'. As mentioned earlier, the adverse effects of this are thought to be wide-ranging from underperformance at school, college or work, to road traffic accidents and other mishaps, as well as antisocial behaviour.[6] Sometimes the situation is complicated by the use of stimulants to stay awake, and alcohol or sedative drugs to get to sleep.

The differential diagnosis of excessive sleepiness can be considered in terms of three main categories of cause: insufficient sleep, disturbed sleep, and an increased need for sleep (Table 9.2.9.3).

Insufficient sleep

The combination of late-night social activities or staying up late for study, and having to get up early for school or college reduces the number of hours many adolescents sleep to below that needed for satisfactory daytime functioning. Difficulty getting off to sleep at night and recurrent waking makes the problem worse (or may be sufficient in themselves to reduce or seriously impair sleep). The result is considerable difficulty getting up in the morning, irritability, emotional liability, lethargy, tiredness, or actually falling asleep during the day.

Correction of the problem of late-night social activities requires a change of lifestyle or other measures which may be difficult to achieve without there being strong motivation to do so. The ideal solution is an agreed, co-operative effort on the part of both the young person and parents.

More specific measures will be needed if the habit of going to bed late has developed into a disturbance of the circadian sleep–wake cycle. This may take the form of irregular sleep–wake schedules or, more usually, the **delayed sleep phase syndrome (DSPS)** which deserves special mention because it is common, especially in adolescents, and potentially very disruptive.

The time at which children fall asleep may become delayed during a period of illness, or because of protracted bedtime disputes about going to bed. In adolescents the problem arises from habitually staying up late for social or other reasons, especially at weekends or during holidays. After a time (the length of which varies with the individual) the sleep phase becomes physiologically delayed with the result that it becomes impossible to go to sleep earlier by choice, in spite of feeling tired and having been awake for a long time. Entreaties to go to bed at a sensible time and get up on time for school are likely to be ineffective.

The diagnostic features of DSPS are persistently severe difficulty getting to sleep, uninterrupted sound sleep, great difficulty getting up for school or work, and sleepiness and under-functioning especially during the first part of the day, giving way to alertness in the evening and early hours. The abnormal sleep pattern is maintained by sleeping in very late when able to do so at weekends and during holidays.

Treatment consists of gradually and consistently changing the sleep phase to an appropriate time. This can be achieved by slowly advancing the sleep phase (e.g. by 15 min a day) where the phased delay is about 3 h or less. More severe forms of the disorder require progressive sleep phase delay in 3 h steps ('round the clock'). Additional measures to maintain the improved sleep schedule include early morning exposure to bright light and firm agreement with the adolescent to maintain the new pattern of social activities and sleep. The place of melatonin remains unclear in view of the many uncertainties about its use and potential hazards, including possible adverse affects on reproductive physiology.

Achieving and maintaining an improved sleep–wake schedule by these means may not be easy. The difficulties are compounded if there is a vested interest in maintaining the abnormal sleep pattern, for example to avoid school ('**motivated sleep phase delay**'). The presence of psychological problems, including depression, may well make successful treatment less likely. 'Conditioned insomnia' may appear similar to DSPS but its origins and treatment are different.

Table 9.2.9.3 Differential diagnosis of excessive sleepiness in older children and adolescents

Insufficient sleep
Late-night activities combined with getting up early
Insomnia
Erratic sleep–wake patterns
Delayed sleep-phase syndrome
Disturbed sleep at night
Sleep-related upper airway obstruction
Recreational drugs (caffeine, alcohol, nicotine)
Illicit drugs (including withdrawal)
Medical and psychiatric disorders
Other sleep disorders (frequent parasomnias, periodic limb movements in sleep)
Increased need for sleep
Narcolepsy
Idiopathic central nervous system hypersomnia
Depression
Substance abuse
Neurological disease
Kleine–Levin syndrome (intermittent sleepiness)
Menstruation-related hypersomnia (intermittent sleepiness)

Disturbed nocturnal sleep

Daytime sleepiness, despite apparent normal time asleep at night, suggests that the restorative quality of sleep is impaired. Poor quality sleep can result from frequent awakenings or less obvious arousals including brief subclinical interruptions (or 'fragmentation') of sleep. Sleep may be disturbed by the following:

- Excessive caffeine, alcohol, or nicotine (combinations are particularly hazardous), and illicit drug use and withdrawal.
- Medical and psychiatric disorders in childhood, and some of their treatments.
- Other sleep disorders: frequent parasomnias are likely to be obvious but periodic limb movements in sleep (now considered to be more important in childhood than previously thought) are much more subtle in their effects on sleep continuity.
- Sleep-related respiratory problems, including OSA. This condition merits some emphasis because of its widespread occurrence: at least 2 per cent of children in the general population are affected with a peak onset at 2 to 6 years. The prevalence is much higher than this in various learning disability syndromes such as Down syndrome, as mentioned earlier.

There are important differences between OSA in children and OSA in adults. The typical adult with OSA is an obese middle-aged male who snores very loudly, under-functions during the day and usually responds to continuous positive airway pressure (CPAP) treatment at night.

- In contrast, most children with OSA are not obese, the usual causes are large tonsils and adenoids (the removal of which can be beneficial), the sex ratio is equal and, whereas adults have prolonged obstructive apnoeas, children often have partial airway obstruction with hypoventilation, actual apnoea events being less frequent and shorter. As already mentioned, the result may be over-activity and other disruptive behaviour rather than obvious sleepiness during the day.

Clinical assessment is the cornerstone of recognizing OSA.

- Night-time signs suggesting the diagnosis are combinations of snoring (although only about one in five children who snore most nights have this condition), other noises suggesting breathing difficulties during sleep, paradoxical chest–abdomen respiratory movements, unusual sleeping positions including neck extension, very restless sleep, profuse sweating, nocturnal enuresis, and sudden distressing awakenings during the obstructive event. There is also a higher incidence of other parasomnias but for reasons that are unclear.
- Daytime features include mouth breathing and adenoidal facies, headache and bad mood on wakening, and the behaviour problems already mentioned.
- Physical examination may reveal the anatomical cause of the obstruction (usually enlarged tonsils and adenoids). Radiological studies are required in the more complicated cases. PSG with respiratory measures is needed to assess the severity of the obstruction and the effects on blood gases during sleep which may be greater than suspected from clinical findings.

Treatment is essential to counter or prevent physical and psychological complications. This usually consists of adenotonsillectomy or, much less commonly, other measures such as CPAP depending on the individual case.

Disorders involving an increased tendency to sleep

This occurs where prolonged or otherwise excessive sleep is an intrinsic part of the condition, rather than a consequence.

(a) Narcolepsy

Narcolepsy is a neurological disorder mainly affecting REM sleep physiology. It is not the rarity once supposed; prevalence in the United States has been estimated at 4–9 per 10 000.

Onset has occurred by adolescence in a high number of cases but the diagnosis is often not made for several years. The reasons for this are that symptoms may be subtle in their early stages, concealed, misinterpreted as laziness or psychological disorder such as depression or conversion disorder, or overshadowed by the child's extreme emotional reaction to the condition. Especially because of its many manifestations in childhood, narcolepsy is a good illustration of how a sleep disorder can be misconstrued as another type of clinical condition, especially when familiarity with the field of children's sleep disorders is limited.[7]

The clinical presentation of early narcolepsy is very variable and some time usually elapses before the classic combination of daytime sleep attacks, overnight sleep disruption, cataplexy, hypnagogic hallucinations, and sleep paralysis develop if, indeed, it does at all. In young patients the first sign may consist of no more than prolonged overnight sleep.

It is appropriate to consider narcolepsy in any young person who is excessively sleepy during the day without an obvious explanation, but repeated clinical and PSG assessment may be required at intervals before a definite diagnosis can be made, including its distinction from other forms of sleepiness such as the group of conditions known as **idiopathic CNS hypersomnia**. The demonstration of low CSF levels of the neuropeptide hypocretin (orexin) is now considered diagnostic of narcolepsy. The PSG features of narcolepsy in adults, which are described elsewhere (Chapter 4.14.3) do not necessarily apply in the childhood stage of the condition.

Narcolepsy is a persistent and disturbing condition for which careful treatment with medication together with ancillary measures, as well as much support and advice about education, career, and psycho-social matters, are required. Other aspects of diagnosis and management are discussed in Chapter 4.14.3.

(b) Kleine–Levin syndrome

This also usually begins in the teenage years with periods of excessive sleepiness alternating with periods of normality. The sleepy episodes are associated (in its classical form) with overeating, hypersexuality, and other disturbed behaviours which are often bizarre and out of character. It is also frequently mistaken for a psychological disorder or other medical conditions. The condition should be distinguished from other causes of intermittent sleepiness in young people such as substance abuse, major depressive disorder (in which the sleepiness is much less marked), menstruation-related hypersomnia, and certain neurological disorders.

Main sleep problems: parasomnias

This category of sleep disorders is described in Chapter 4.14.4 in relation to adult psychiatry. The present account emphasizes aspects of the parasomnias of particular importance in childhood and adolescence when, collectively, they are more common than in adult life. Frequently, parasomnias cause parents much concern and they appear to be the subject of considerable diagnostic confusion and delay. As in older patients, different types of parasomnia may co-exist. A detailed account of parasomnias in young patients is available elsewhere.[8]

The following general points about childhood parasomnias have clear implications for clinical practice.

- Precise diagnosis is important as different parasomnias may well need contrasting types of treatment. Accurate diagnosis depends principally on a detailed account of the subjective and objective sequence of events from the onset of the episode to its resolution, as well as the circumstances in which the episode occurs, including its timing. Audio–visual recording (including the use of home video systems) can be very informative. Only occasionally is PSG required, although this can be instructive where clinical evaluation is inconclusive and, sometimes, where there is the possibility that another type of sleep disorder co-exists.
- The more dramatic forms of parasomnia seem to be a particular cause of diagnostic confusion and imprecision, and also quite possibly unnecessary concern about their psychological significance as most are benign.
- Especially when the range and manifestations of sleep disorders is not appreciated, parasomnias (and other sleep disorders) may well be misinterpreted as other physical or psychological conditions.

- A child may have more than one kind of parasomnia or, indeed, more than one sleep disorder (e.g. arousal disorders associated with obstructive sleep apnoea).
- As many childhood primary parasomnias remit spontaneously within a few years, children and parents can often be reassured about the future, although protective measures (e.g. in severe headbanging or sleepwalking) may be required in the meantime.
- Specific treatment, including medication, is needed in only a minority of cases of primary parasomnia but is likely to be required for the underlying disorder in many of the secondary parasomnias.
- Research information on this point is limited, but a primary parasomnia might be symptomatic of a psychological problem if it is very frequent, unusually late in onset or persistent, or associated with a traumatic experience.
- Parasomnias may lead to psychological complications if the child is frightened, embarrassed, or otherwise upset by the experience, or because of the reactions of other people to the episodes.

Primary parasomnias

- Sleep-related **rhythmic movement disorders** such as headbanging, occur in many young children, almost always remitting spontaneously by 3 to 4 years of age. Although alarming to parents, they are usually of no psychological significance (unlike daytime headbanging associated with severe neurodevelopmental disorder). However, protective measures, such as padding the cot-sides, may be needed.
- **Hypnagogic** (sleep onset) and **hypnopompic** (on waking) hallucinations are common and may be frightening to the child.
- Parents are often distressed to witness **confusional arousals, agitated sleepwalking**, or **sleep terrors** which are a form of '**partial arousal disorder**' common in young children (see Chapter 4.14.4 for an account of arousal disorders). The degree of agitation and confused behaviour may be extreme, suggesting that the child is suffering in some way. In fact, in arousal disorders the child remains asleep and unaware of the events. Understandable attempts to arouse the child and provide comfort should be discouraged, as this may cause real distress. Although violence during sleep is described mainly in sleepwalking adults, such behaviour can occur in children.
- The term 'nightmare' is sometimes used misleadingly for any form of dramatic parasomnia. **True nightmares** (frightening dreams) are common. If frequent and associated with intense bedtime fears, they may indicate an anxiety disorder and their content may suggest a cause.
- **Nocturnal enuresis** is very common, affecting about 5 per cent of 7-year-olds at least once a week. Delayed maturation often seems to be the explanation, but physical or psychological factors may be involved, especially where previous bladder control is lost. Behavioural treatment can be very effective.

Secondary parasomnias

Nocturnal epileptic seizures are not uncommon in children and must be distinguished from primary parasomnias because of their different significance, and also the investigation and treatment they require. Seizures which are behavioural in manifestation are the most likely to be misdiagnosed as non-epileptic, for example benign centro-temporal (Rolandic) epilepsy of childhood and nocturnal frontal lobe seizures both of which are closely related to sleep.

Other parasomnias, which are part of medical or psychiatric disorders and which may be encountered in patients of any age, include nocturnal asthmatic attacks with accompanying distress, those associated with OSA or gastro-oesophageal reflux, panic attacks, nocturnal disturbance that is part of the post-traumatic stress disorder, and dissociative states. Simulated parasomnias, shown by PSG to be enacted during wakefulness, can sometimes occur in children.

Further information

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9.2.10 Suicide and attempted suicide in children and adolescents

David Shaffer, Cynthia R. Pfeffer, and Jennifer Gutstein*

Introduction

Suicidal behaviour is a matter of great concern for clinicians who deal with the mental health problems of children and adolescents. The incidence of suicide attempts reaches a peak during the mid-adolescent years, and mortality from suicide, which increases steadily

through the teens, is, in many countries, one of the leading causes of death at that age.

Historical review

Until the late 1950s, knowledge about youth suicide was drawn from unrepresentative case reviews, reviews of the demography of suicide drawn from death certificate data, and speculation about dynamics. The late 1950s saw the first systematic psychological autopsy study among adults that demonstrated the importance of psychiatric disorder as a proximal cause of most suicides.[1] This was followed by similar studies on children and adolescents,[2–5] confirming the association in adolescence. Starting in the mid-1960s, the incidence of suicide in young males began to rise in many countries.[6] The rate of increase eventually stabilized in the late 1980s and, in many countries, is now showing signs of falling.[7] These changes stimulated efforts to develop methods of preventing youth suicide.[8–11].

A good deal is now known about which teenagers commit suicide, less about who attempts it, and very little about the optimal management of suicidal adolescents. The number of randomized controlled trials designed to assess different forms of treatment is exceedingly small, and many suggestions for clinical management are based on anecdotal accounts rather than on findings from well-designed experimental trials.

Clinical features

Completed suicide

Completed suicide occurs most commonly in older adolescents, and, although it can also occur in children as young as 6 years of age, it is excessively rare before puberty.[7] Psychological-autopsy studies have shown that about 90 per cent of adolescent suicides occur in individuals with a pre-existing psychiatric disorder, often present for several years.[3–5] In teenagers, the most common disorders are some form of mood disorder, substance and/or alcohol abuse, often comorbid with a mood disorder in boys over age 15, and anxiety disorders.[3,4] At a trait level, many suicide completers have been noted to be irritable, impulsive, volatile, and prone to outbursts of aggression. However, this pattern of behaviour is by no means universal, and anxious suicides have usually shown no evidence of prior behavioural, academic, or social disturbances.[3]

Although some adolescents—predominantly girls suffering from a major depressive disorder—appear to have thought about suicide for some time before death, most adolescent suicides appear to impulsively follow a recent stress event, such as getting into trouble at school or with a law-enforcement agency; a ruptured relationship with a boy- or girlfriend; or a fight among friends. In many instances, these stress events can be seen as a by-product of their underlying psychiatric disorder.[12]

It also appears that a completed suicide can be precipitated—in a presumably already suicidal youth-by exposure to news of another person's suicide, or by reading about or viewing a suicide portrayed in a romantic light in a book, magazine, or newspaper.[13]

About a third of completed suicides have made a previous known suicide attempt, more commonly girls and those who suffered from a mood disorder.[3] Completed suicide must be distinguished from autoerotic asphyxia, which is rare in teenagers.[3] Suicide pacts, common between middle-aged or elderly married couples and/or other family members, are similarly rare in adolescents, but are not unknown.[3]

Non-lethal suicidal behaviour

(a) Suicidal ideation

Suicidal ideation includes thoughts about wishing to kill oneself, making plans of when and where, and having thoughts about the impact of one's suicide on others. Such thoughts may occur without great significance among young children, who may not appreciate that suicide may result in irreversible death.[14] However, appreciation of the finality of death should not be a factor in judging the seriousness of suicidal ideation. Suicide threats made by young children and adolescents most often involve a threat to jump out of a window, to run into traffic, or to stab himself or herself.

(b) Attempted suicide

The most common profile of a teenaged attempter is a 15- to 17-year-old girl who has taken a small- or medium-sized overdose of an over-the-counter analgesic or medication taken by another family member. The behaviour is usually impulsive and occurs in the context of a dispute and humiliation with family or a boyfriend.[15] The clinical features most strongly associated with suicide attempts are irritability, agitation, threatening, violent, or psychotic behaviour, and a persistent wish to die.[16]

Groups in whom suicide attempts appear to be common include runaways,[3] children who have been exposed to physical and sexual abuse, and homosexual teenagers.[17] However, study-design issues make it unclear whether this is because of a high rate of psychopathology or substance abuse in these groups or because of some factor that specifically predisposes to suicidal behaviour.

A subset of non-fatal suicidal behaviour involving ingestion with a non-lethal intent is sometimes referred to as parasuicide. However, intent is difficult to gauge retrospectively, and not all teenagers are aware of the lethalness of an ingestion, so that this term carries with it a risk of complacency and is probably best avoided in teenagers.

Assessment

Suicide attempts

Assessment of a suicide attempt involves an evaluation of the short-term risk for suicide and attempt repetition, and an assessment of the underlying diagnosis or other promoting factors. If the child or teenager has been referred to as an ideator, it is important to determine whether they are contemplating or have secretly attempted suicide.

Repeated attempts, attempts by unusual methods (other than ingestions or superficial cutting), medically serious attempts, and attempts where the patient has taken active steps to prevent discovery all increase the risk for further attempts or death.[18,19] Children and adolescents systematically overestimate the lethality of different suicidal methods, so that a child with a significant degree of suicidal intent may fail to carry out a lethal act.[20–22]

The mental states leading to suicidal behaviour include anticipatory anxiety, pessimism, or hopelessness, as well as paranoid or other cognitive distortions arising from an underlying psychiatric diagnosis.[23] Inappropriate coping styles (e.g. impulsivity or catastrophizing) in response to external stress may also contribute to the behaviour. Motivating feelings may include the wish to effect a change in interpersonal relationships, to rejoin a dead

relative, to avoid an intolerable situation, to get revenge, or to gain attention.[22]

Classification of associated diagnoses

Suicide

Psychiatric diagnoses commonly associated with a suicide include depression, bipolar disorder, substance abuse, conduct disorder, and overanxious and panic disorders.[3–5] Although the rate of suicide in schizophrenics is high, because of the rarity of the condition, it accounts for very few suicides.

Suicide attempts

Recurring suicidal behaviour has been associated with hypomanic personality traits and cluster B personality disorders.[22] A history of impulsivity, mood lability, with rapid shifts from brief periods of depression, anxiety, and rage to euthymia and/or mania—associated with transient psychotic symptoms, including paranoid ideas and auditory or visual hallucinations-is associated with a risk for further suicide attempts and is compatible with the diagnosis of borderline personality disorder. Many of these symptoms are also features of bipolar mood disorder.

Epidemiology

Completed suicide

(a) Age

In the United States, the age-specific mortality rate from suicide for 10- to 14-year-olds was 1.6 per 100 000 in 1997.[24] This age group accounts for 7 per cent of the population but only 1 per cent of all suicides, and most of these occur in 12- to 14-year-olds.

The comparable figures for 15- to 19-year-olds are about six times higher. The suicide rates at this age in the United States and Canada in 1997 were 9.5 per 100 000 and 12.86 per 100 000 respectively.[7] The proportion of suicides that occur in this age group is about the same as its representation in the general population.

Suicide rates for 15- to 24-year-olds in some other English-speaking countries were 11.0 for males and 2.2 for females in the United Kingdom (1995), 16.0 in Australia (1995), and 26.1 in New Zealand (1997) (all per 100 000 population).[7]

(b) Gender

In the United States and most other countries, male suicides outnumber female suicides among 15- to 24-year-olds by a ratio of 4:1. In China and Cuba, the suicide rate is higher in females than in males.[7]

(c) Cultural and ethnic differences

Rates of suicide vary considerably in different cultural and national groups.[7] Possible reasons include variable access to lethal methods, different degrees of social support, integration, or group adherence, or the influence of religious beliefs or spirituality.[25–27] In some instances, the differences may be a function of geography rather than culture. Contagion within isolated groups may determine differences in rates.

(d) Secular changes

From 1964 to 1995, the suicide rate in the United States and Canada among 15- to 19-year-old males increased almost three-fold, and similar increases were reported in Australia, New Zealand, and the United Kingdom.[7] In most of these countries, there was little change in the female rate or in the rate amongst 10- to 14-year-olds. Fluctuations in the suicide rate appear to be real, rather than due to any methodological artefact (e.g. changes in reporting practices). The most plausible reason for the increase in suicidal behaviour among teenage boys is an increase in alcohol and substance use in the youth population.[3] The reasons offered for the recent decline in suicide rates include lowered substance- and alcohol-use rates among the young and more effective diagnosis and treatment.[28]

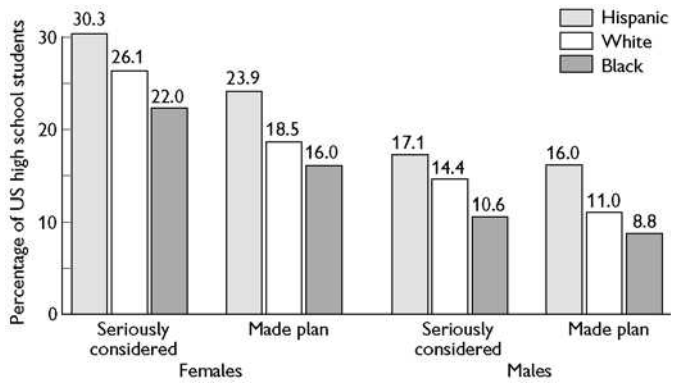

Fig. 9.2.10.1 Youth risk behaviour survey: prevalence of suicidal ideation in teenagers in the previous 12-month period (1997) broken down into gender and ethnicity. (Reproduced from Centers for disease control (1998). Attempted suicide among high-school students–US, 1997. Morbidity and Mortality weekly Report, **47**, 47–9, copyright 1998, centers for Disease Control and Prevention, US.)

Attempted suicide

There is a strong inverse relationship between attempted suicide and age. A large epidemiological survey of four suicide-related behaviours (ideation, plan, gesture, and attempt) in the United States has shown a significantly higher rate of all four behaviours in the youngest age group (15–24 years).[29] This study also compared rates of these four behaviours across two decades (1990–1992 and 2001–2003). It found that rates did not decrease, despite a dramatic increase in pharmacologic treatment.

Suicide attempts in adolescents are at least twice as common in females as males (see Figs 9.2.10.1 and 9.2.10.2). Considerable ethnic variation is seen in the United States, with, for unknown

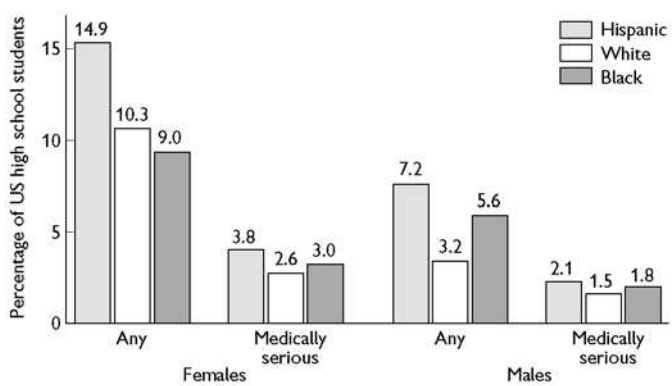

Fig. 9.2.10.2 Youth risk behaviour survey: prevalence of suicide attempts in teenagers in the previous 12-month period (1997) broken down into gender and ethnicity. (Reproduced from Centers for disease control (1998). Attempted suicide among high-school students–US, 1997. Morbidity and Mortality weekly Report, **47**, 47–9, copyright 1998, centers for Disease Control and Prevention, US.)

reasons, Hispanic high-school students having twice the rate of black or white teenagers.[30]

Aetiology

Completed suicide

(a) Psychiatric disorders

The most important risk factor for suicide is a psychiatric disorder.[3] Controlled studies of completed suicide suggest similar risk factors for boys and girls,[3,h31] but with marked differences in their relative importance[3–5] (Table 9.2.10.1). In girls, major depression is the most powerful risk factor, which, in some studies, increases the risk of suicide 12-fold; followed by a previous suicide attempt, which increases the risk approximately three-fold. In boys, a previous suicide attempt is the most potent predictor, increasing the rate over 30-fold. It is followed by depression (12-fold increase), disruptive behaviour (two-fold increase), and substance abuse (increasing the rate by just under two-fold).[3]

(b) Psychosocial stressors

Stressful life events often precede a suicide and/or suicide attempt.[12] They are rarely a sufficient cause in suicide, and their importance seems to lie in their action as a precipitant of stress in young people who are at risk by virtue of their psychiatric condition. Family discord, lack of family warmth, and a disturbed parent-child relationship are commonly associated with types of child and adolescent psychopathology, but these factors do not play a more important role in suicide.[12]

(c) Cognitive factors

Perceptions of hopelessness, negative views about one's own competence, poor self-esteem, a sense of responsibility for negative events, and the immutability of these distorted attributions may contribute to the 'hopelessness' repeatedly found to be associated with suicidality.[18,19]

(d) Biology

Biological factors, specifically dysregulation of the serotonergic system, are common in adult suicides.[32] Dysregulation is manifested by low levels of serotonin metabolites in central nervous system fluids, low concentrations of presynaptic serotonergic receptors, and dense concentrations of postsynaptic receptors. Such serotonin abnormalities have been localized to the ventrolateral prefrontal cortex and brainstem of suicide victims and attempters (in postmortem positron-emission tomographic studies as well as in *in vivo* biological challenges).[33] Serotonin may inhibit extreme fluctuations of mood and reactivity, and the vulnerability to suicide of individuals with these biological abnormalities may be mediated by impulsivity and emotional volatility. As the ventral prefrontal cortex plays a role in behavioural inhibition, it is conceivable that serotonin irregularities in this area make it more difficult for a suicidal individual to control his suicidal impulses.[33] The frequency with which these biological findings occur in adolescent suicide attempters is not yet clear, and studies to demonstrate the precise behavioural correlates of serotonin dysregulation profiles are still lacking. Nordstrom *et al.*[34] have suggested that knowing the biological status of suicide attempters may have a practical value, in that low 5-hydroxyindole acetic acid concentrations in cerebrospinal fluid examined shortly after a suicide attempt may differentiate between suicide attempters who will commit suicide or repeat the attempt within a year and those who will not. The biology of suicidal behaviour is considered more fully in chapter 4.15.3

Table 9.2.10.1 Psychiatric diagnoses in child and adolescent suicides

	Martunnen *et al.*[5]			**Shaffer *et al.*[6]**			**Brent *et al.*[4]**		
Country	Finland			USA			USA		
Area	National			Greater New York			Western Pennsylvania		
Period	1987–1988			1984–1986			1984–1994		
N	53			120			140		
Age	13–19			<20			13–19		
Percentage girls	17			21			15		
Control group	None			Matched community			Matched community		
Diagnostic system	DSM-IIIR			DSM-III			DSM-III		
	Males	**Females**	**All**	**Males**	**Females**	**All**	**Males**	**Females**	**All**
Any diagnosis (%)	93	100	94	90	92	91	82	81	82
Any mood disorder (%)	48	67	51	60	68	61	43	71	47
Substance abuse (%)	27	44	30	42	12	35	35	24	34
Conduct/antisocial/ disruptive disorder (%)	18	11	17	54	36	50	35	10	31
Any anxiety disorder (%)	2	11	4	27	28	27	13	24	14
Schizophrenia (%)	5	11	6	3	4	3	—	—	—
Past suicide attempt	27	67	34	28	50	33	37	62	41

(e) Imitation

Evidence has accumulated indicating that suicide in vulnerable teenagers can be precipitated by exposure to real or fictional accounts of suicide, such as intense media coverage of a real suicide or the fictional representation of a suicide in a popular film or television programme. The risk is especially high in the young, and lasts for approximately 2 weeks.[13] The phenomenon of suicide clusters is also presumed to be related to imitation.

"Cybersuicide," or a tendency for internet sites such as Bebo or MySpace" to encourage suicide pacts or to increase completed or attempted suicide in vulnerable adolescents, has become an increasing concern. This concern is based on multiple case reports from Japan, Wales, and the United States. These have captured substantial media attention. As yet no objective empirical data have been amassed to determine if there is a causal relationship between internet use and youth suicide.

One hypothetical model for how biological and social factors fit together is illustrated in Fig. 9.2.10.3.

In a longitudinal study of a large African-American community in the United States, Juon and Ensminger[35] found risk factors for suicidal behaviour in African-Americans to be very similar to those found in Caucasians (depression, substance use, and a number of family variables).

Course and prognosis

Natural history

Little is known about the natural history of suicidal behaviour, but early-onset suicidal behaviour in prepuberty predicts suicidal behaviour in adolescence[36,37] and an early-onset major depressive disorder is associated with suicidal behaviour in adolescence and adulthood.[38] Attempts to predict, at the time of the first attempt, which adolescents are likely to repeat their suicidal behaviour have been unsuccessful.[22]

Emergency treatment

Because of the need to respond to a suicidal crisis, it is desirable to offer treatment within a service-delivery system that includes inpatient and outpatient settings, acute crisis work, stabilization, extended management, and follow-up care and monitoring.

Outpatient treatment should be used when the child or adolescent is unlikely to act on suicidal impulses, when there is sufficient support at home, and when there is someone who can take action if the adolescent's behaviour or mood deteriorates. Children and adolescents should never be discharged from an emergency or care service without the child's or adolescent's caretaker having been interviewed (see Box 9.2.10.1) to ensure that firearms and/or lethal medications will be made inaccessible to the child. Unless this advice is given, parents will rarely, on their own initiative, take the necessary precautions. Before discharge, the clinician must have a good understanding of the amount of support that will be available for the child or adolescent if he or she is discharged home.

Acute psychiatric inpatient care should be reserved for patients for whom intensive surveillance and intervention are considered essential-as in the presence of active suicidal ideation and intent or when the youngster is unable to commit to not carrying out a suicidal act, when the youth is unpredictable, impulsive, agitated, or psychotic, or if there is a lack of support and supervision in the home. There is no evidence that exposure to other suicidal psychiatric inpatients increases the risk of suicidal behaviour. Determining when a patient is ready for discharge from the hospital or crisis centre will usually include an evaluation of the severity of existing suicidal ideation and intent. Implicit coercions (e.g. telling patients that discharge will be delayed until they can state that they are not suicidal) should be avoided.

Treatment compliance may be improved by offering definite, closely spaced, follow-up appointments, being flexible in arranging appointments if a crisis should arise, and reminding the family and patient by telephone or note about the next appointment. If an appointment is missed, the patient and parent should be contacted. Hopeless and depressed children and adolescents, who may be not be able to commit to a lengthy treatment process, may be better engaged by offering short-term treatment plans with defined intervention goals. While offering confidentiality for some issues,

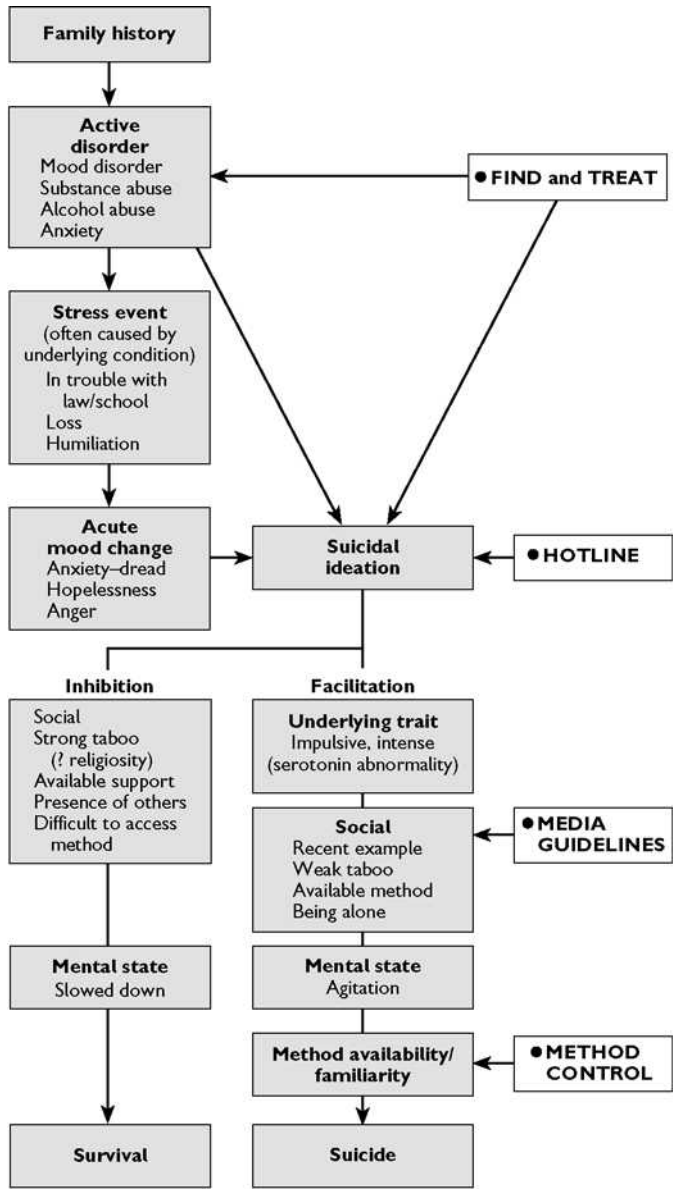

Fig. 9.2.10.3 How do suicides occur and how can they be prevented?

Box 9.2.10.1 Checklist before discharging an adolescent who has attempted suicide

Before discharging a patient from the emergency room or crisis centre, always:

- Check that *firearms* and lethal *medications* have been secured or removed
- Check that there is a *supportive person* at home
- Check that a *follow-up appointment* has been scheduled

it is essential that the clinician communicate to the patient that, if they feel that suicidal thinking or behaviour is imminent, such information will be shared with the parents.

Contracts

A written or verbal 'no-suicide' contract is commonly negotiated at the start of treatment in the hope that it will improve treatment compliance and reduce the likelihood of further suicidal behaviour.[39] In its usual form, the child or adolescent promises not to engage in suicidal behaviour without first informing the parents, therapist, or other responsible adult when he or she has thoughts of suicide or plans to commit suicide. No empirical studies have evaluated the efficacy of a contract, and contracts should be seen as no more than adjuncts to the management of patients with low intent. Even if the patient agrees to such a contract, suicide risk may persist. It should also be appreciated that a 'no-suicide' contract may lessen a patient's communication of stress and dysphoria, decrease the potential for developing a therapeutic alliance, and impair risk management. As mentioned above, coercive communications should be avoided, because these may encourage deceit and defiance.

Specific psychotherapies

Working with suicidal children and adolescents is best done by a clinician who is available, has skill and training in managing suicidal crises, relates to the patient in an honest and consistent way, and can convey a sense of optimism and activity. Given these personal attributes, the therapist may use various models of psychotherapy, although relatively few empirical studies have evaluated their efficacy.

(a) Cognitive behavioural therapy

Cognitive hbehavioural therapy is effective in depressed teenagers,[40] but its value for suicidal adolescents has not been demonstrated.[23] Brent *et al.*[23] modified the approach for depressed adolescents. The treatment comprized 12 to 16 once-weekly sessions, followed by a 6-month booster phase of monthly or bimonthly sessions. It included a psychoeducational manual about mood disorders, training to monitor and modify automatic thoughts, assumptions, and beliefs, training in more assertive and direct methods of communicating, and help in conceptualizing alternative solutions to problems. Meetings with parents were sometimes held to augment the treatment, and psychopharmacology was used adjunctively if depressed adolescents had not improved after 4 to 6 weeks of pharmacotherapy.

Brent's study provides no evidence of the efficacy of cognitive-behavioural therapy for teenagers who had made a suicide attempt who were not included in this study.

(b) Dialectical behavioural therapy

Dialectical behavioural therapy (**DBT**) is the only form of psychotherapy that has been shown in a randomized control trial to reduce suicidality in adults with borderline personality disorder.[41] This treatment is based on a biosocial theory in which suicidal behaviours are considered to be maladaptive solutions to painful negative emotions that also have affect-regulating qualities and elicit help from others.[41]

The treatment involves developing problem-oriented strategies to increase distress tolerance, emotion regulation, interpersonal effectiveness, and the use of both rational and emotional input to make more balanced decisions. It usually involves individual and group sessions over the course of a year, although an untested modification for adolescents (DBT-A) is designed to take 12 weeks.[42] It involves the participation of a relative who is charged to improve the home environment and to teach other relatives how to model and reinforce adaptive behaviours for the adolescents.

(c) Family therapy

As indicated above, family discord, poor communication, disagreements, lack of cohesive values and goals, and irregular routines and activities are common in suicidal children and adolescents who often feel isolated within the family. Family intervention aims to decrease such problems, improve family problem-solving and conflict resolution, and reduce blame directed at the suicidal child or adolescent. Family-based cognitive therapy aims to reframe the family's understanding of their problems, to alter the family's maladaptive problem-solving techniques, and to encourage positive family interactions. Psychoeducational approaches can help parents clarify their understanding of childhood and adolescent suicidal behaviour, identify changes in mental state that may herald a repetition, and reduce the extent of expressed emotion or anger.[31]

Psychopharmacological interventions

In meta-analyses of adult studies, lithium maintenance treatment greatly reduces (8.6-fold) the recurrence of suicide attempts in adults with bipolar or other major affective disorders. Further, when lithium is discontinued there is a seven-fold increase in the rate of suicide attempts and a nine-fold increase in the rates of suicide.[43] Other mood stabilizers, such as valproate and carbamazepine, are also widely used to treat bipolar disorders in children and adolescents; although their efficacy has yet to be empirically demonstrated. Depressed suicidal children and adolescents with a history of bipolar disorder should first be treated with a mood stabilizer before receiving an antidepressant.

Studies in depressed adults have found that the selective serotonin reuptake inhibitor (**SSRI**) antidepressants reduce suicidal ideation, and also reduce the frequency of suicide attempts in non-depressed patients with cluster B personality disorders with a past history of suicide-attempt behaviour.[44] In contrast to the highly lethal potential of tricyclic antidepressants when taken in overdoses, SSRIs have low lethal potential. In a controlled trial of the depot neuroleptic flupenthixol, Montgomery and Montgomery[45] noted a significant reduction in suicide-attempt behaviour in adults who had made numerous previous attempts. Similar studies have yet to be conducted for adolescents.

In the past decade, there has been much controversy over whether the SSRI antidepressants can induce suicidal ideation

and/or behaviour. A number of case reports appeared in 1990 describing patients who had developed suicidal preoccupations after starting treatment with fluoxetine. These reports were not supported by meta-analyses and re-analyses of large SSRI-treatment trials of depressed, bulimic, or anxious patients.[46,47] The conclusion was reached that suicidal ideation is a common feature of depression and that the prevalence in SSRI-treated depressives was no greater than expected.

However, one reanalysis of the data presented in certain of these studies suggested that new ideation was significantly more common in SSRI-treated depressed patients who had not previously reported suicidal ideation. Further, in a naturalistic challenge study, Rothschild and Locke[48] were able to reinduce suicidal ideas in a small series of patients who had first experienced ideation after starting treatment with fluoxetine. These patients had also experienced akathisia as a complication of fluoxetine treatment, and a relationship between suicidality and fluoxetine-induced akathisia has been noted by others.

Several meta-analyses have shed some additional light on this complex issue. A British meta-analysis of 702 clinical trials involving 87,650 adult patients documented a two-fold increase in suicide attempts in patients receiving SSRIs as compared to placebo.[49] An American meta-analysis of pediatric patients used data from 23 trials involving 4582 patients.[50] This study also found an increased risk of suicidality in patients taking SSRIs, after controlling for the risk associated with suffering from depression. Both British and American regulatory agencies now require warnings about suicidal risks associated with SSRIs. (See also chapter 9.5.5)

At this stage, the wisest course of action is for the practitioner to be particularly observant during the early stages of fluoxetine treatment of a depressed adolescent, to systematically enquire about suicidal ideation before and after treatment is started, and to be especially alert to the possibility of suicidality if SSRI treatment is associated with the onset of akathisia.

One must be careful about the risk of inducing suicidal ideation or behaviour through psychopharmacological activation or disinhibition. Clinicians should be cautious about prescribing medications that may reduce self-control, such as the benzodiazepines, and phenobarbitone (phenobarbital). These drugs also have a high lethal potential if taken in overdose. Montgomery[51] noted that benzodiazepines may disinhibit some individuals who then become aggressive and attempt suicide and that there are suggestions of similar effects from the antidepressants, maprotiline and amitriptyline, the amphetamines, and phenobarbitone. Amphetamines or other stimulant medication should only be prescribed when treating suicidal children and adolescents with attention-deficit hyperactivity disorder.

Possibilities for prevention

Community-based suicide prevention

The principal public health approaches to suicide prevention have been as follows:

- crisis hotlines
- method control
- media counselling to minimize imitative suicide
- indirect case-finding by educating potential gatekeepers, teachers, parents, and peers to identify the 'warning signs' of an impending suicide
- direct case-finding among high-school or college students or among the patients of primary practitioners by screening for conditions that place teenagers at risk for suicide
- training professionals to improve the recognition and treatment of mood disorders.

(a) Crisis hotlines

Although crisis hotlines are available almost everywhere in the United States, research so far has been fairly limited and has failed to show that they impact on the incidence of suicide.[52] Possible reasons for this include the fact that actively suicidal individuals (males and individuals with an acute mental disturbance) do not call hotlines because they are acutely disturbed, preoccupied, or intent on not being deflected from their intended course of action. It also seems that the large majority of callers are females, whereas males are at the greatest risk for suicide, that crisis lines are often busy and there may be a long wait before a call is answered so that callers disconnect, and that the advice that individuals receive on calling a hotline may be stereotyped, inappropriate for an individual's needs, and perceived as unhelpful by the caller.

While each of these deficiencies is potentially modifiable, to date there have been no systematic attempts to do so. Research studies in this area have been sparse and are sorely needed.

(b) Method restriction

Method preference varies by gender and by nationality. In the United States, the most common method for committing suicide is by firearm, and it has been suggested that reducing firearm availability will reduce the incidence of suicide. However, in a natural experiment in the United Kingdom, when self-asphyxiation with coal gas became impossible after the introduction of natural gas, the decline in the suicide rate was marked but short-lived. There is, as yet, no good evidence that reducing access to firearms by gun-security laws has a significant impact on suicides attributable to such, although they do impact on accidental and homicidal deaths from firearms.[53]

(c) Media counseling

The United States Centers for Disease Control have issued sensible guidelines for reporters and editors, pointing to the risks of exaggerated or prominent coverage of youth suicide in general, and of the risks in focusing attention on an individual suicide.[11] These sensible guidelines should be known to child clinicians who are engaged in public-health practice, even though there is, as yet, no good evidence that their application is effective in reducing the suicide rate.

(d) Indirect case-finding through education

Controlled studies have failed to show that classes for high-school students about suicide increase students' help-seeking behaviour when they are troubled or depressed.[54] On the other hand, there is evidence that previously suicidal adolescents are perturbed by exposure to such classes.[51] Such educational programmes seem, therefore, to be both an ineffective mode of case-finding and to carry with them an unjustified risk of activating suicidal thoughts. Educational approaches in schools are dsicussed further in chapter 4.15.4

(e) Direct case-finding

If asked in a non-threatening way, adolescents will provide accurate information about their own suicidal thoughts and/or behaviours.[21] Therefore a sensible approach to suicide prevention is to systematically screen 15- to 19-year-olds (the age group at greatest risk) for previous suicide attempts, recent serious suicidal preoccupations, depression, or complications of substance or alcohol use. Youths identified in this way should be referred for evaluation and, if necessary, treatment.

(f) Training primary care physicians and gatekeepers in the recognition and treatment of mood disorders

Preliminary and, as yet, unreplicated studies in Sweden[55] suggest that education of primary practitioners to identify the characteristics of mood disorders better and to treat these effectively produced a significant reduction in suicide and suicide-attempt rates in women. Because the optimal treatment of adolescent depression is not as well understood as that of adult depression, this is an option that may prove to be useful, but further work is needed.

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9.2.11 Children's speech and language difficulties

Judy Clegg

Introduction

Speech and language difficulties have a significant impact on the lives of children and their families. This chapter will give an overview of the types of speech and language difficulties children present with and how these are generally classified and diagnosed. Specific Language Impairment (SLI) and speech and language difficulties associated with child psychiatric disorder, specifically disorders of attention and selective mutism will be a focus. The life course of children with speech and language impairments will be described through childhood, adolescence, and adult life. Current management approaches will be presented and evaluated and strategies for effective communication considered.

Clinical features

Typical speech and language development

It is remarkable how quickly and easily most children progress through the typical stages of speech and language development to become competent communicators by the age of 5 years. Much is known about how children acquire speech and language and when these skills are achieved.[1] Children need to be competent communicators prior to starting school, as learning is dependent on adequate speech and language abilities. At school entry age, children are expected to be able to speak clearly, to understand and use complex grammatical structures, to use language for a range of communicative reasons from requesting to negotiating and predicting, to take part confidently in conversations with both children and adults and to have a knowledge of letter names and sounds and to read some single words. The acquisition of these speech and language skills will enable the child to access the

educational curriculum where learning is dependent on both verbal and written language. If children are not competent in these skills then they will experience significant difficulties in their learning from the start of their school career.

Features of speech and language difficulties

Speech and language development can be affected by hearing impairment, visual impairment, general learning disability, epilepsy, and specific syndromes of learning disability such as Down's syndrome, and Fragile X syndrome. In these examples, speech and language difficulties are usually attributed to and explained by an aetiological cause. However, speech and language difficulties do occur in the absence of an obvious identifiable cause and are therefore considered as a specific impairment, e.g. SLI.

Prevalence rates of speech and language difficulties vary and are dependent on the criteria used to define and classify them. Law *et al.*[2] report prevalence rates in children as high as 24.6 per cent whereas rates for SLI are much lower between 3 and 7 per cent.[3] Importantly, speech and language difficulties can persist over time and often have a negative impact on the child's education and general well-being.

(a) Speech difficulties

A speech difficulty reduces a child's intelligibility and may result in speech sounds being omitted, substituted with another sound or distorted. Speech difficulties can be evident when a child says single words, sentences, and participates in conversation. The physical articulation of speech sounds is affected by physiological and structural abnormalities, such as cleft lip/palate, and neurological impairments leading to dysarthria characterized by weakness and/or in-coordination of the speech musculature system. There is another group of children who have phonological speech difficulties. These children have an intact speech musculature system but have not managed to acquire all the speech sounds of their language and so can only use a limited range, which subsequently limits their intelligibility.

(b) Language difficulties

Language difficulties can involve problems in the development of both comprehension and production.

(i) Vocabulary difficulties

Restricted word knowledge and poor development of the understanding of word meanings result in small vocabularies. Some children have impoverished vocabularies but other children can have specific word finding or retrieval difficulties. Here, the child knows the word he wants to say but is unable to retrieve it accurately and quickly. This is usually evident by 'searching' behaviours where the child may substitute the word for a related word, use a filler word such as 'thingy' or 'stuff', gesture the word instead of saying it or say the first sound of the word but not the rest. For example,

> ICE SKATING: 'I can't do that thing . . . erm . . . you know . . . where you put sharp shoes on . . . I always fall over'.
> PLUM: 'well, I don't really like that one which smells like soil and is purple and juicy'

These problems may not only be due to lexical difficulties but also problems retrieving the right phonological sounds of the word. Cognitive impairments in information processing, specifically short-term and phonological working memory have been associated with problems in vocabulary learning.[4]

(ii) Syntax difficulties

Children often have difficulties in their understanding and use of syntax and as a consequence find it very difficult to not only understand language but also to construct sentences in order to use language to communicate effectively, for example giving a narrative where past events are described and future events predicted. Common problems are learning how to use inflections to mark different tenses and understanding as well as constructing complex sentences such as passives. The child in the following example has lots of syntax difficulties as well as word finding difficulties and it is clear how this affects his ability to convey verbal information. The correct forms the child is attempting are shown in brackets.

'They erm . . . was . . . erm . . . goed to make (made) some vegetable circles (pizzas) and rolls (they rolled) it (the dough) out because that's what you do first and he was reading the menu (recipe) as well and then they is erm . . . erm . . . erm . . . printing (cut) them out and then they put them in the oven because they'll taste crunchy (to cook) and then erm . . . then they took them out of the oven so they be . . . er . . . get . . . cool down (could cool down) and then you would take (ate) them'.

(iii) Social communication difficulties

Children with speech and language difficulties often show associated problems in social communication behaviours, also referred to as pragmatics. These can be both verbal and non-verbal and include difficulties with eye contact, initiation, turn taking, interaction, sharing, requesting, and responding. Higher level social communication abilities can also be affected such as inferring information, giving the listener adequate information and self-monitoring. Ultimately, these can all hinder effective communication between the child and others and also expose the child to negative social experiences, particularly with their peers. For some children, the social communication difficulties may be an intrinsic part of a developmental disorder where speech and language difficulties are evident, for example children with autistic spectrum disorders. In other children, it is important to note that these behaviours can develop as a secondary consequence of poor communication skills due to the speech and language difficulty.

Classification

Speech difficulties can occur in isolation without the presence of language difficulties. Language difficulties can also occur without the presence of speech difficulties but often speech and language difficulties co-occur together. Children can have difficulties with both language comprehension and language production.

Within child psychiatry, both the ICD-10[5] and DSM-IV[6] systems categorize developmental speech and language difficulties. However, there is little robust empirical evidence to support the subtyping of speech and language difficulties. Children are usually classified according to whether the speech and language difficulty is specific, i.e. cognitive development is age appropriate and if there are any co-morbid aetiological or functional explanations. Descriptions of the type of speech and language difficulty involve identifying how the speech and language system is disrupted, describing the levels of impairment, and how this is impacting on the child's communication and their access to learning.

Diagnosis and differential diagnosis

Descriptions of developmental speech and language disorders

Children's language is said to be 'delayed' when their language abilities are behind those expected for their chronological age and 'impaired' or 'disordered' when a language delay does not resolve and the child continues to experience significant and severe problems. Several established diagnoses of developmental speech and language disorders are described below:

(a) Cleft lip and palate

A cleft/lip palate results from the incomplete fusion of the hard or soft palate in the embryonic stages of development. A cleft palate can be accompanied by a cleft lip or either one can occur independently. In the United Kingdom, cleft lip/palate is repaired in the first few months of life. However, some children can be left with fistulas and velopharyngeal incompetency, which significantly affects speech development and intelligibility. Children with cleft lip/palate receive speech and language therapy from birth onwards. At birth the focus of attention is primarily on feeding and then the development of speech and language.

(b) Dysarthria

Dysarthria is a speech disorder due to neurological impairment which affects how the speech musculature system functions. Children with cerebral palsy often have dysarthria, which makes their speech slow, weak, and uncoordinated. There may be a mild, slight slurring of speech to profound dysarthria where a child cannot produce any intelligible sounds or words. Children with moderate and severe dysarthria have shallow breathing which is insufficient to sustain speech and/or a low-pitched voice, nasal speech, and a reduced range of vowels and consonants that can be produced accurately.

(c) Developmental phonological disorder

Unlike cleft lip/palate and dysarthria, phonological speech disorders involve the child's developing speech sound or phonological system. The child's speech is difficult to understand because the child makes speech sound errors which are either due to the speech sound system developing more slowly or in an atypical way and this is not a result of obvious structural, sensory, or neurological impairments. Often, there are systematic patterns of errors in the child's speech, for example the child always replaces the 's' sound with a 'd' sound. Auditory processing and discrimination skills have been implicated in the development and maintenance of this disorder. Over time, phonological disorders often resolve with speech and language therapy input. However, for some children they are severe and do persist into adult life.

(d) Childhood apraxia of speech (CAS)

This developmental speech disorder is characterized by both speech and non-speech behaviours. The speech sound errors are inconsistent and are accompanied with oral movement difficulties in drooling, feeding, and blowing. Reduced early verbal behaviours such as babbling are often evident. CAS often co-occurs with motor apraxia but for some children, only speech and oral movements are affected. There is some debate as to the existence of CAS as there is no obvious cause although both neuromotor planning and the organization of the child's phonological system have been implicated. CAS is often a label given to children where the speech disorder has persisted despite intervention and oral non-speech movements are affected. See Dodd[7] for a detailed review of children's speech disorders.

(e) Fluency disorders

Although classified under speech disorders, stuttering is not an articulatory or phonological difficulty. There are no structural abnormalities and the child usually has a typically developing phonological system. Core stuttering behaviours include part-word or whole-word repetitions, revisions, pauses, blocks, sound prolongations, and obvious struggling behaviours such as jerky head movements. Secondary behaviours result from the stuttering and generally help the individual to avoid stuttering. For example, circumlocution where the speaker substitutes a word he knows he will stutter on for an easier word and environmental control such as avoiding the use of the telephone or talking to certain people. Fluency disorders are often identified in young children before the age of 5 years although many children experience a period of normal non-fluency usually between the ages of 2 and 5 years, which is not severe and resolves spontaneously.

(f) Learning disability

Level of cognitive ability is the strongest predictor of language ability and therefore language development is certainly affected in learning disability. The sequence of language development is similar to that found in typical development but with mild to moderate to severe and profound delay. A child with a profound learning disability may never develop an intent to communicate whereas another child may have established an intent but no verbal language and uses some signs or symbols to communicate instead. For children with mild and moderate learning disability, language abilities plateau with no further improvement, usually in adolescence at a level below the child's chronological age.

It should be noted that specific patterns of speech and language development have been identified in specific syndromes of learning disability. Down's syndrome is characterized by superior vocabulary development to grammatical development and children with William's syndrome often appear as competent communicators but do have significant language learning problems. Speech and fluency problems are common in learning disability and vary according to the aetiology of the learning disability. For example, conductive hearing loss and articulatory speech problems occur where there is cranio-facial involvement.

(g) Acquired childhood aphasias

Acquired aphasias refer to a loss or deterioration in language ability after a period of typical language development. The child acquires language but then loses these language abilities, usually between 3 and 7 years of age. Causes of childhood aphasia include open and closed head injury, cerebrovascular lesions, cerebral infections, cerebral tumours, and epilepsy. Landau Kleffner (first described by Landau and Kleffner in 1957)[8] is an acquired aphasia where language deteriorates after a period of typical language development and the deterioration in language is usually, although not always accompanied with a seizure disorder. Receptive language is severely affected with expressive language problems as well, often word finding difficulties. See Lees[9] and Deonna[10] for a complete review.

Specific language impairment (SLI)

Specific language impairment (SLI) is a term used to describe language impairment (and additional speech impairment) where there is no identifiable medical, neurological, sensory, or functional cause and where cognitive ability measured by non-verbal intelligence (IQ) is within the normal range. Therefore, there is a discrepancy between language and cognitive ability with the exclusion of any obvious causes for the language impairment. Diagnosis of SLI according to exclusionary and discrepancy criteria is dependent on standardized language and cognitive psychometric assessments. However, there is continuing debate regarding which criteria to use to establish a meaningful discrepancy between language and cognition. ICD-10,[5] for example adopt a strict criteria of language skills at least two standard deviations below the level expected for the child's chronological age and language skills at least one standard deviation below the child's level of non-verbal IQ. More liberal criteria advocates a non-verbal IQ of 75 or above with language abilities often only one SD below the mean. Proponents of liberal criteria claim that more stringent criteria may fail to identify children who are at risk of poor long-term outcomes. However, liberal criterion may identify children who simply perform at the lower end of the normal distribution of language ability. It should be recognized that different criteria are used. Although the diagnosis of SLI stipulates good cognitive ability, some specific cognitive deficits in phonological memory, verbal, and visuo-spatial memory and symbolic play are evident and thought to underpin the language impairment.

SLI is considered to affect 3–7 per cent of all children.[3] The use of the exclusionary and discrepancy criteria to define SLI means that as a group, children with SLI are very heterogenous with impairments in many areas of language. Although useful, attempts to subtype SLI[11] have not yet proved clinically robust. However, children with SLI are considered to show disproportionate difficulties in vocabulary and syntax compared to other aspects of language.

(a) Aetiology of SLI

Research in SLI primarily focuses on trying to establishing a cause. SLI is a heritable disorder and much research is underway to try and establish the genetic basis.[12,13] SLI is of particular interest to researchers because of the unusual dissociation between cognitive and language ability and whether this dissociation is explained by innate modular theories of language acquisition or more general cognitive processing deficit theories. Some attempt has been made to identify genetic markers of SLI such as a phonological memory deficit[12] which stems from the research into general cognitive processing deficits as underlying SLI and a specific tense marking deficit[14] or a syntax representational deficit[15] which argues for the disruption of innate modular components of language.

(b) Diagnostic overlaps between SLI and autistic spectrum disorders (ASD)

(i) Pragmatic language impairment

Autism and autistic spectrum disorders (ASD) are discussed extensively in Chapter 9.2.2 of this text. Language and communication difficulties are central to both SLI and ASD. However, the fundamental difference between these disorders is the severity and pervasiveness of the social communication impairment. In SLI, social communication difficulties are considered secondary to the language impairment where children with speech and language difficulties will have problems in developing appropriate social communication skills. In ASD, the social communication impairment is an intrinsic part of the disorder and does not develop as a secondary consequence of a speech and language impairment. Due to the increase in the identification of ASD and the use of the autistic spectrum many more children with milder difficulties are being diagnosed with ASD. This has led to some researchers proposing that there are overlaps between SLI and ASD.

Semantic–pragmatic disorder was first described in the 1980s as a subtype of SLI[16,17] and was a label used to describe children with comprehension problems, echolalia, behaviour difficulties, and difficulty with non-literal language, semantics and pragmatics. At the time, these children were not considered as autistic. However, the increasing use of the autistic spectrum led to debates about whether semantic–pragmatic disorder exists as a separate category of SLI or whether it should be included on the autistic spectrum.[18,19]The crucial issue was whether the social impairment was intrinsic to the language disorder or a secondary consequence of the language disorder. To address this, researchers have attempted to show differences in pragmatic abilities between children with SLI, ASD, and typically developing children. For example, Bishop and Norbury[19] identified a subgroup of SLI children who show a profile of Pragmatic Language Impairment (PLI). These children showed inappropriate behaviours across aspects of social communication including initiating conversations, understanding subtle aspects of language such as humour and sarcasm, adapting their communication to different contexts, understanding and using non-verbal communication, and engaging in conversations about specific interests. Importantly, these children did not show the non-verbal repetitive behaviours typically characteristic of autism. Overall it is argued[19] that there are continuities between autism and specific language impairment but not all children with pragmatic impairments have autism. Therefore, pragmatic language impairment alone should not be used to make diagnoses of autistic spectrum disorders. It is recognized that there are conflicting opinions about the increasing evidence that indicates continuity between disorders that have traditionally been regarded as distinct from one another. However, assessment should consider whether a child's social communication difficulties are being compounded by language difficulties as amelioration of the language difficulties may improve the child's social communication.

(c) Associations between language and behaviour in child psychiatric disorders

Children with primary psychiatric disorders often have a history of developmental problems which can include speech and language delay. Children with primary speech and language disorders are at greater risk of developing behaviour difficulties than children without speech and language disorders. Various mechanisms have been put forward to try and explain this association. These include common antecedents such as low intelligence and deprivation,[20] environmental factors where language stimulation is negatively affected by poor parent–child interactions or the child's inability to attend to the language stimulus,[21] the psychosocial rejections and academic failure experienced by children with communication impairments affecting their self-confidence and self-esteem and therefore their subsequent emotional behaviour development,[22–24] and a neurodevelopmental abnormality or immaturity as a shared

underlying cause.[25,26] Although the simplicity of these mechanisms is appealing, identifying, and differentiating them is certainly complex.

More recently studies have shown that children with primary psychiatric disorders can have undetected speech and language disorders.[27,28] ADHD is one of the most commonly reported psychiatric disorders associated with speech and language difficulties.[29] In ADHD, language difficulties consist of both receptive and expressive problems[30] and pragmatic language difficulties[31,32] such as excessive talking and poor topic maintenance. Although studies have not identified a distinct profile of speech and language difficulties in ADHD, they should be considered in assessment and management.

It has been hypothesized that these undetected speech and language disorders somehow play a role in the development and maintenance of the psychiatric disorder and even that the psychiatric disorder is secondary to the undetected speech and language disorder (see mechanisms above). There is very limited evidence available to specify what the associations are and importantly, it may be that referral practices play a role where the psychiatric problem takes priority and the child is referred to mental health services first. In the United Kingdom SLT and mental health services are usually very separate and it is not common that SLTs work in mental health services. The identification of previously undetected speech and language disorders in the studies above are really the late identification of pre-existing difficulties. Nevertheless, management of childhood psychiatric disorders should consider if speech and language difficulties are a factor in the child's behaviour as they may have an impact on how the child is managed, particularly with respect to participation in verbal therapies and education.

(c) Selective mutism

This childhood disorder is described as the persistent refusal to talk in certain social situations despite being able to talk in other situations. The most common pattern is talking at home but not at school and the refusal to talk cannot be better accounted for by a communication disorder or difficulties in understanding and using spoken language. Pervasive developmental disorder or psychotic disorder should also be excluded. The mutism must last for more than a month (this cannot be the first month of school) and interfere significantly with educational progress, social communication with others, and occupational achievement. In the case of a bilingual child, it is suggested that the mutism should persist for at least 6 months and be present in both the first and second language before diagnosis.[33]

Selective mutism is rare and as a result only a limited number of studies reporting the epidemiology of this disorder are available. Prevalence figures estimate a prevalence of approximately 0.75 to 0.80 per cent [34,35] and it is slightly more common in girls[36] with an onset between the ages of 3 and 5 years.[34] Although there is no clear consensus to explain the cause(s) of the disorder, social phobia, and anxiety are certainly involved. Co-morbidity with behaviour problems, communication difficulties and developmental delay are also found which indicates that a multi-factorial aetiology is the best explanation. Data regarding the long-term outcomes is scarce but there are indications that with early intervention improvements are made but often children are still left feeling uncomfortable in some speaking situations.

(d) Intervention for selective mutism

Intervention approaches include pharmacology, cognitive behaviour therapy, family therapy, psychodynamic therapy, and speech and language therapy. Although some success with fluoxetine has been reported[37] this has not been widely replicated. A behavioural approach considers the disorder as learned behaviour and techniques including contingency management, shaping and stimulus fading, systematic desensitization, and self-modelling are advocated. Family therapy aims to identify whether there are difficulties in family relationships that are contributing to the mutism and attempts to work with the whole family to foster more positive relationships. Psychodynamic approaches involve techniques of play therapy and art therapy to identify the underlying reasons for the mutism and to help the child to express the possible unconscious conflicts he is experiencing. Although children with selective mutism are expected to have good speech and language skills, several studies have reported a high incidence of speech and language difficulties such as articulation and expressive and receptive language difficulties.[38,39] In these circumstances, speech and language therapy is used as a valuable adjunct to the other approaches. Speech and language therapy aims to facilitate the child's communication rather than resolving the underlying causes of the mutism. Therapists work with the child to desensitize him/her to communicating with others by considering the child's communication environment and the communication load of the tasks he is expected to engage in. A hierarchy of stages is followed from easy to hard speech tasks within easy to hard speaking situations. A multi-modal perspective incorporating a combination of the above approaches is advocated. The combination of family involvement with cognitive behavioural, speech and language, psychodynamic and family involvement meet the multi-factorial needs of this disorder. See Cohan *et al.*[40] for a detailed review of the efficacy of the different intervention approaches described. Although social anxiety is the predominating feature of selective mutism, the resulting lack of communication is challenging. Several strategies to facilitate communication with these children are presented and these can easily be incorporated into other intervention approaches:

- Check that there are no speech and language difficulties that may be contributing to the mutism. For some children, although early speech and language difficulties may have resolved the child may still feel under confident in their talking.
- One-to-one settings are most comfortable and try and include the familiar person, (usually the primary carer) who the child communicates regularly with in your interventions to start with. After a while the child may be able to manage this setting without the familiar person. This process may have to be repeated to encourage the child to talk in another setting.
- Encourage and accept non-verbal communication such as head nods, writing, drawing, and gesture as well as verbal communication. Non-verbal communication is easier than talking for most of these children.
- Follow a hierarchy of verbal communication from easy to hard, most children find whispering and talking quietly easier than loud talking.
- Consider the complexity of the task the child is expected to engage in. Questions, which are factual or only require a yes or

no response are much less confrontational than questions, which ask the child about their feelings or opinion.

Further management considerations are detailed in Johnson and Wintgens.[41]

Course and prognosis

Life course and outcomes

An interest in the long-term outcomes of children with speech and language difficulties has emerged fairly recently. Historically, it was considered that primary speech and language difficulties resolved over time with no implications for other areas of development. This is certainly not true and much more is now known about the developmental trajectories through childhood, adolescence, and into adult life. Generally, children with speech and language difficulties continue to show difficulties not only in communication but in cognition, behaviour, educational attainment, and psychosocial functioning. This section will focus on the life course and outcomes of children with profiles of primary speech and language difficulties where there is no cognitive deficit. Cognitive ability is a powerful predictor of development, and therefore the outcomes of children with speech and language difficulties associated with cognitive delay are usually attributed to level of IQ rather than the specific speech and language difficulties themselves.

The impact of a speech and language impairment over the lifespan

Speech and language impairment leads to impoverished communication skills, which certainly impact on other areas of development. The developmental trajectories of children with primary speech and language impairment show impaired receptive and expressive language development in later childhood, adolescence, and even adult life. Cognition, as measured by non-verbal IQ has been shown to fluctuate and even deteriorate over time, particularly in adolescence and later life. Research to date suggests that any deterioration is temporary and resolves but much more needs to be known about this.[42,43] Children with speech and language difficulties are more likely to experience emotional and behavioural problems (see earlier section). During the course of childhood, the risk of developing emotional and behavioural problems seems to increase with obvious negative implications for other areas of development and functioning. There is a possible association between SLD and the development of antisocial behaviour in early adult life. At the age of 19 years participants with SLD in a Canadian community sample did not show high levels of aggression but did have higher rates of arrests and convictions.[26]

The increase in social and behaviour problems may be the result of communication and interaction problems young people experience in conjunction with the increasing social and academic demands placed on them. However, it is hard to disentangle cause and effect when so many variables interact. Socio-economic status (SES), learning ability, and type of educational placement no doubt also make a contribution. Therefore, SES and IQ are probably implicated in the development of social and behavioural difficulties as well as the speech and language impairment. In adult life, severe mental health conditions such as schizophrenia, depression, and personality disorder have been linked with early histories of and persisting severe speech and language impairment.[44]

There are strong associations between speech, language, and literacy development and in fact, speech and language are fundamental in learning to read and write. In order to learn, children need to be competent communicators when they start school. Language is the medium through which children are expected to learn and literacy is dependent on identifying and discriminating speech and letter sounds. Children with persisting speech and language difficulties are at risk for literacy problems and subsequent low academic achievement. Children with speech and language difficulties find it much harder to learn to read and write than children without speech and language difficulties. Educational attainment is dependent on literacy and therefore these children are very disadvantaged. Studies measuring educational attainment using Standard Assessment Tests (SAT) have found that children with speech and language difficulties gain significantly lower SAT scores than controls.[45] In older individuals, attainment at GCSE and A level is also affected. Children with speech and language difficulties are often bullied by their peers and can be targets for victimization.[46] Studies suggest that this is due to their odd communication particularly difficulties with social communication behaviour. Unclear speech is also a significant factor. In adult life, high levels of social maladaption and poor psychosocial functioning were found in adults in their mid 30s with SLI. Employment, relationships, independent living, and health were all significantly and negatively affected.[44] See Clegg[47] for a full review.

Identification of children at risk

There are variations in outcomes and not all children with speech and language impairment are at risk of later negative outcomes. Issues of variability and risk and resilience are not yet fully understood. Risk factors for poor outcomes in later life are the severity of the initial impairment, involvement of receptive language, low IQ, and low SES. Resilience factors are the presence of pure speech difficulties only, high IQ, high SES, and access to specialist support. A critical age hypothesis[48] proposes that any speech and language difficulties that impact on a child's communication after 5 years should be considered as significant and prioritized for intervention.

Speech and language difficulties should not be underestimated in terms of their impact on children's lives. With respect to clinical management, the risk factors should be considered in identifying those children at particular risk. In the United Kingdom, there is specialist educational provision now available that provides post-16 years provision for some young adults to try and reduce negative outcomes, e.g. supporting individuals to gain further qualifications and to enter the work place. Information about long-term outcomes can certainly inform the individuals themselves, their families, and other professionals about prognoses and how best to support individuals to meet their learning and other needs effectively.

Management

Speech and language therapists (SLTs) work in education, health, and social care, voluntary organizations and independent practice. A SLT is an important member of the interdisciplinary team and will lead on the assessment, differential diagnosis, intervention with, and management of individuals with communication and swallowing disorders. In recent years due to the shift to inclusion, many paediatric SLTs now work in schools with educational

professionals as part of the school team. This enables the SLT to contribute to statements of special education needs, formulating independent learning plans (ILP), and delivering the curriculum to make it accessible for children with speech and language difficulties. SLTs also work in community services such as the government Sure Start programme, primary and secondary health care, e.g. acute hospital settings and community clinics, specialist health services, e.g. child development centres and education, e.g. from preschool, mainstream and special schools/resourced provision. The inclusion of SLTs in Child and Adolescent Mental Health Services (CAMHS) is increasing but this is not consistent across the United Kingdom. However, there is now recognition that the SLT can have a valuable role within the CAMHS[49,50] in terms of the identification of any communication difficulties and the subsequent impact on mental health and offering intervention that will help to ameliorate the communication difficulty and facilitate communication. Within CAMHS, a SLT may be employed in a specialist unit, child psychiatry outpatient service, or other. Differential diagnoses are usually made in collaboration with the team and a SLT will use a combination of formal and informal assessment. Formal assessment consists of using measures that compare the child's speech and language abilities with abilities that are expected for the child's chronological age. Various types of assessments are available criterion referenced, standardized, developmental scales, and observational. Intervention may be direct, i.e. individual or as part of a group or indirect, i.e. through working with the family or in the context of the child's classroom and school.

Identification of speech and language difficulties in children

The following checklist may be helpful when working with young school age children in identifying speech and language difficulties and initiating referral to speech and language therapy services.

(a) Speech

1 Is the child's speech difficult to understand?
2 Does the child miss out sounds from words?
3 Is the child less intelligible when speaking in sentences than in single words?
If yes, to one or more of the above then speech difficulties are evident and referral to speech and language therapy is advised.

(b) Comprehension

1 Can the child follow and engage in conversations with both children and adults?
2 Can the child carry out verbal instructions correctly or does he need lots of prompting?
3 Can the child understand a range of concepts such as time and space?
If no, to one or more of the above then comprehension difficulties are evident and referral to speech and language therapy is advised.

(c) Production

1 Does the child have a wide range of vocabulary?
2 Can the child use more complex sentences such as the 'the boy ran for the bus because he was late' rather than the boy was late and ran for the bus?
3 Is the child able to give a narrative of a past and future event?
4 Does the child frequently say non-specific words such as 'stuff' or 'thingy' or hesitate when talking?
If no to 1 to 3 and yes to 4 then production difficulties are evident and referral to speech and language therapy is advised.

(d) Social communication

1 Is the child willing to participate in conversations and does he enjoy this?
2 When the child talks, is it meaningful and relevant to the conversation and/or situation?
3 Does the child use appropriate verbal and non-verbal communication behaviours?
If no to one or more of the above the social communication difficulties are evident and referral to speech and language therapy is advised.

(e) Education and social activities

1 How is the child functioning at school?
2 Is the child able to access and participate in a range of social activities?
3 Are the speech and language difficulties long standing and persisting beyond the age of 5 years?
If the child is struggling to meet the demands of school and presents with persisting speech and language difficulties then this is a cause of concern for later development and outcomes. Referral to speech and language therapy is advised.

General principles for working with children with speech and language difficulties

It may be useful to consider these general principles when working with children with speech and language difficulties

1 Consider your own communication and adapt it to:
- Offer forced choice answers.
- Break up long instructions and sentences into short steps.
- Slowdown delivery and use pauses.
- Use short simple sentences with familiar vocabulary and avoid ambiguous language.
- Use visual strategies such as pictures, real objects, and symbols to support spoken language.
- Remember that children with spoken speech and language difficulties will also have written language difficulties.

2 It is always challenging when you are unable to understand what a child is saying to you. While there is no perfect solution, the following strategies will help:
- Reassure the child that you are interested in what they are trying to tell you.
- Be honest and say that you don't understand but also make it clear which parts you did and did not understand.
- Offer a choice of possible answers to the child, as this will reduce the number of choices available to you to guess from.
- Ask the child to show you something to help or can the child describe it to you or point to it or draw it.
- If these are still not successful, reassure the child that you are interested in what they are trying to tell you and that you will try again later.

Further information

Speake, J. (2004). *How to identify and support children with speech and language difficulties*. LDA, Cambridge, UK.

This is a useful text, which describes children's speech and language difficulties and offers practical strategies for assessment and management.

www.afasic.org.uk

Afasic is a UK charity that offers information and advice to children with speech and language difficulties and their families as well as professionals working in this area.

www.ican.org.uk

ICAN is a UK charity that funds specialist educational provision for children and adolescents with severe speech and language difficulties as well as offering information and advice.

www.rcslt.org

The Royal College of Speech and Language Therapists (RCSLT) is the professional body for practicing speech and language therapists in the UK. Information about speech and language therapy services is available on this website.

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9.2.12 Gender identity disorder in children and adolescents

Richard Green

Variance in psychosexual development

Psychosexual development of sex-typed behaviours spans a broad mix of the elements that comprise 'masculinity' and 'femininity'. The possibility for variation is extensive. Among males, there are boys and men whose stereotypical masculinity may pose problems in mental health and criminality. They are not the focus here. Rather, here it is the marked deviation from the mean towards the 'non-masculine' or 'feminine' extreme. That pattern can also cause clinical concern and constitutes gender identity disorder (**GID**) as manifested in childhood. For females, conventional 'tomboyism' is not the focus here, but rather the extreme that can cause clinical concern and constitutes GID.

Epidemiology

No epidemiological studies exist of GID in children. Prevalence can be estimated only roughly from indirect sources. Two items on the Child Behaviour Checklist[1] are consistent with components of the diagnosis. They are 'behaves like opposite sex' and 'wishes to be of opposite sex'. Among 4- to 5-year old boys, not clinically referred for behavioural problems, about 1 per cent of parents answer in the affirmative that their child 'wishes to be the opposite sex'. For ages 6 to 7 it drops to near zero, but rises to 2 per cent at age 11. For girls, the highest rate was 5 per cent at ages 4 to 5, but less than 3 per cent for other ages. With respect to 'behaves like opposite sex', among the boys the rate was 5 per cent and among girls 11 per cent for all ages. However, these data do not indicate any longitudinal aspect of the reported behaviour, and do not detail the behaviour.[2]

An alternative source of estimation looks to the percentage of adults believed to be homosexually oriented. From this population the percentage of homosexual men and women who typically report childhood cross-gender behaviour is used for the estimate. If the rate of exclusive homosexuality is 3 to 4 per cent for men and 1.5 to 2 per cent for women,[3] with perhaps half of homosexual men and women recalling childhood cross-gender behaviour,[4,5] the estimate of childhood cross-gender behaviour is about 3 per cent for boys and under 1 per cent for girls. However, this estimate suffers from problems of retrospective recall and poor comparability between surveys of adults. Further, the recalled behaviour may not have constituted GID.

A disparate sex ratio is evident in referral rates with GID. Four to five boys to one girl are referred. One reason may be greater parental concern over cross-gender behaviour in boys and the greater stigmatizing peer group response to 'sissiness' than to 'tomboyism'. An alternative explanation is that, as with most atypical patterns of sexuality, there is a higher ratio of males to females reflecting a common intrinsic predisposition among males.

Clinical picture

Children with GID differ from other children, including those who merely are not conventionally masculine or feminine as boys or

girls. Their behaviours are typical of other-sex children. Not only do they express a wish to be the other sex, at least in earlier years before they may learn not to verbalize it, but also their dressing preferences, peer group preferences, toy preferences, game preferences, and perhaps their physical mannerisms are those of the other sex.[6]

The picture of GID in children as described in DSM-IVTR,[7] can manifest, in part, by the repeatedly stated desire to be of the other sex: in boys by a preference for dressing in girls' or women's clothing or simulating female attire from available materials, and in girls an insistence on wearing stereotypically masculine clothing with refusal to wear traditional girls' clothing. In role playing, as in make-believe play or imitating media characters, there is a strong preference by the child for other-sex roles. There is also a strong preference for toys generally identified with the other sex, such as Barbie dolls by boys. The peer group is composed primarily or exclusively of other-sex children. Pictures drawn are generally of other-sex figures. There may be cross-sex physical mannerisms. Concurrently, there is an avoidance of traditionally sex-typed activities. Criteria in ICD-10 are similar.[8]

The diagnosis of GID in girls can be more problematic than in boys. This is because 'tomboyism' is a more common part of paediatric psychosexual development than 'sissyness'. There is, however, a distinction between GID in girls and tomboyism. Typical tomboys do not insist that they want to be boys and will wear girls' clothes from time to time, will have both girls and boys as playmates, and will not work to present themselves as young boys.

Substantial cross-gender behaviours are generally manifest in the third or fourth year. Although they are believed by parents and perhaps professional advisors to be a passing phase, at least with those children, seen clinically they endure into school years. Most children are evaluated at about age 7 or 8, when parents become increasingly concerned that the 'passing phase' is not passing and negative reactions by the peer group are enhanced, causing the child social distress.[6]

Aetiology

Understanding the aetiology of GID considers typical influences on early psychosexual development in male and female children.

Early sex differences

Very early behavioural differences are evident between males and females. There may be recognition of the 'like me', 'not like me' dichotomy of one's sex. When boys and girls aged between 10 and 18 months were shown pictures of faces of infants of the same and other-sex, males looked at faces of males longer and females looked at faces of females longer. This 'like me', 'not like me' dichotomy is also interpretable in the study in which two male and two female 1-year-old children were placed at the four corners of a room and permitted, one at a time, to crawl to any other child. Children more often crawled to a child of the same sex.[9,10]

In early play patterns, boys and girls may differ. When 12-month-old children were observed with their fathers in a waiting room, boys were more likely to handle 'forbidden' objects such as trays and vases.[11] The 1-year-old's toy preferences may also differ, with girls preferring soft toys and dolls and boys preferring transportation toys and robots.[12,13]

Preference for mother or father appears to discriminate boys and girls early. When 2- to 3-year-old children were asked which parent in an adjoining room they would prefer to play a game with, or to build with using blocks, or make a sketch with, both boys and girls preferred their father. At 4 years, girls shifted to mother but boys stayed with father.[14]

When children aged between 2 and 3 years were observed in a free play setting, boys were more aggressive toward peers and showed more rough-and-tumble play. When paired in a test play situation with a boy, girls showed more passive behaviour, i.e. standing or sitting quietly and watching their partner play.[15]

The preference for a same-sex peer group emerges early. When 3.5- to 4.5-year-olds were shown pairs of photographs of boys and girls and asked to select the children with whom they would prefer playing, boys preferred boys and girls preferred girls.[16]

Children become aware of sex role stereotypes early. At 2 years of age, boys believe that boys like to play with cars and help their father, 3-year-olds believe that boys like to build things and that only boys like to play with trains. They also believe that girls like to play with dolls, help mother, and cook dinner. Girls are also seen as more likely to say 'I need help'.[17]

The peer group influences psychosexual development. In mixed-gender peer groups, boys more often receive positive responses for masculine activities than girls receive for feminine activities. Boys seem more responsive to peer pressure, in that they will discontinue feminine activities more rapidly than girls will discontinue masculine activities when they are the target of negative responses from either boys or girls.[18]

These findings suggest that if sex-typed attributes emerge in psychosexual development of typical children shortly after the basic dichotomization of 'like me', 'not like me', or male/female, then the first two components of gender identity are consolidated early: (a) the basic sense of male or female; (b) masculine or feminine gender role. The age at which they consolidate coincides with the emergence of significant cross-gender identity and behaviour as seen in the GID of childhood.

Parental influences

Fathers, when observed with 12-month-old children, were more likely to present their sons with trucks rather than dolls, whereas daughters were given both trucks and dolls equally. However, among those children who were given dolls, boys played with them less.[19]

Mothers of young children have been observed with infant actor/actress babies (Baby X experiments). Some of these stranger infants are cross-dressed or given cross-sex names. The perceived sex of the infant influences the mother's behaviour. Children believed to be boys, whether they were or not, were more likely to be encouraged to physical action. Infants believed to be girls were more likely to be given a doll, whereas male infants were more likely to be presented with a football.[20]

None of these findings of early sex differences have been systematically observed with children followed up years later to determine whether early variation from the more common patterns are associated with later variation in psychosexual development.

In our prospective research of several dozen cross-gender behaving boys and conventionally masculine boys,[21] more mother-son shared time was not found in the group of feminine (prehomosexual) versus masculine boys. There was substantial variability in the extent to which mothers and sons were emotionally close. However, with respect to father-son experiences, feminine boys

shared less time with their fathers in their first years when compared with the contrast group of conventionally masculine boys or with their masculine preheterosexual brothers. There was an inverse relationship between the extent of father-son shared time in the first years and later Kinsey score of sexual orientation. Less father-son time was associated with a higher (more homosexual) score.

Identifying the 'chicken and egg' here is problematic. Possibly, boys with a feminine identification who prefer feminine-type activities are less interesting to their fathers. This would lead to father-son distancing. In many of the families, this was the case. However, the finding that early father absence or father-son alienation was associated with cross-gender behaviour was not invariable. Further, there are families in which the mother and father relate comfortably with children and in which GID manifests itself.

Hormonal influences

Evidence for hormonal influences on psychosexual development derives primarily from studies of the intersexed. Girls with congenital virilizing adrenal hyperplasia who produce an excess of androgen beginning prenatally are more rough-and-tumble in childhood play behaviours and less interested in doll play. They are more likely to be considered tomboys.[22] However, less evidence exists for a deficiency in prenatal androgen for boys with cross-gender behaviours.[23–25] Prenatal sex hormone levels are important theoretically, in that to the extent they influence sex-typed behaviour, such as rough-and-tumble or doll play, they may influence peer group composition. They may influence the labelling of the child as 'sissy' or 'tomboy', and may place the child on an atypical developmental track.

Seminal studies of the intersexed in the 1950s indicated that the sex of assignment in the first 2 to 3 years of life was the critical variable in establishing the basic concept of sexual identity as male or female. This was irrespective of gonadal status, hormonal status, internal reproductive structures, and, to some extent, genital configuration.[26] These studies have been criticized on the ground that with the anatomically intersexed the prenatal endocrine status has not been normal.[27] Thus recent interest has focused on individuals believed to have had normal prenatal development but who shortly after birth were nevertheless reassigned to live in the other-sex role, as well as those with a prenatal abnormality.

In one widely publicized case, one male of a pair of monozygotic twins suffered penectomy through circumcision trauma in the first year of life and was reassigned to live as a girl alongside the boy co-twin at about 23 months of age. Although earlier reports indicated that the reassigned twin was adjusting successfully to life as a girl,[28] more recent follow-up revealed that the individual reverted to living as a male in late adolescence, had undergone phalloplasty, and married a female.[29] The other case involves a male infant who also underwent penectomy from circumcision trauma and was assigned to live as a girl, earlier, in the seventh month. That individual was reported to be living as a woman and is bisexual in orientation.[30] One explanation for the discrepancy in the two reports is that the first child was reassigned as a female later than the time during which basic identity of male or female may be set. Both reports, however, suggest a prenatal influence on sexual orientation.

Children born with cloacal exstrophy also provide evidence for prenatal factors influencing gender identity, irrespective of postnatal socialization. Prenatal sex steroid levels are thought to be normal. However, the genital area of these infants, if chromosomally male is so malformed, that there is little prospect of male genital reconstruction. Therefore, many are socialized as girls. Reports from the US reveal a high rate of rejection of living as girls and transition to living as boys. [31] However, an early report from the UK does not indicate female gender role rejection.[32]

The enzyme deficiency of 5-alpha reductase is another clinical example of competing influences of prenatal sex steroids and postnatal socialization. Without this enzyme testosterone is not converted to dihydrotestosterone, needed prenatally to virilize the genitalia. At birth these chromosomal males with intra-abdominal testes appear to be girls based on their external genitalia. Traditionally, they have been raised as girls. Then, at puberty, they do not feminize but rather their clitoris grows substantially to resemble a phallus and there is no gynecomastia. Most then adopt a male role.[33] Debate continues whether this facility to live as heterosexual men is the product of prenatal testosterone or the extensive body virilization and social pressures to live as men. Long-term study of children where the testes are removed before puberty will provide further information.

Longitudinal aspects of atypical early development

Beginning in the late 1960s, the author conducted a prospective study of several dozen boys with extensive cross-gender identification and behaviour.[6] Most of these boys would today be diagnosed with GID, although at the time the diagnosis had not yet entered into the diagnostic nomenclature. These boys were evaluated periodically and assessments continued until late adolescence or young adulthood for two-thirds. At that time, three-quarters of the boys were homosexual or bisexual. One was gender dysphoric. In contrast, a demographically matched group of boys with conventional boyhood behaviours was heterosexual at outcome.[34] More recent follow-up studies at another program reveal a higher minority percentage of cross-gender children remaining gender dysphoric but with the majority homosexually oriented.[35]

These prospective studies are consistent with retrospective reports by adult transsexual males and homosexual males. Many transsexuals recall extensive cross-gender identification and behaviours in childhood. Often, however, these are not documentable because of the length of time from onset to description and the difficulty of corroboration. Several studies have interviewed adult gay men and lesbian women with respect to gender-typed behaviours in childhood. Typically, more extensive cross-gender behaviours are reported than by groups of heterosexual men and women. These retrospective studies of men and women are consistent cross-culturally.[4,5]

Of theoretical and practical import is the overlap in childhood gender behaviours between retrospective reports given by transsexuals and homosexuals, and in the prospective study of cross-gendered boys. Because transsexualism and homosexuality in the adult male are quite different, the question is: Why should there be such an overlap?

One possibility is that the two groups are relatively similar in earlier years, but that different life circumstances promote more comfort for one group continuing to live as males. Treatment intervention to change cross-gender behaviour may be decisive. Transsexuals were rarely treated as children. Different prevalence

rates may also be key. Whereas the incidence of transsexualism may be one in 10 000 males,[36] the incidence of homosexuality may be 3 or 4 per cent.[3] Thus if there are overlapping behaviours between the two in early years, probability would predict that the vast majority of cross-gendered males will emerge as homosexual, rather than transsexual. However, this does not explain the behavioural overlap between prehomosexual boys and pre-transsexual boys who will later be sexually attracted only to females (the latter living as lesbian women after sex reassignment surgery).

Gender identity and mental disorder

GID of childhood was introduced into the DSM in 1980. Its inclusion derived from the prospective study of cross-gender behaving boys described above, with the present author also being a member of the nomenclature committee. The criteria for a set of behaviours being included in the DSM was that a condition be experienced subjectively as distressing and that it constitute a social disadvantage. GID of childhood met the criteria because of the distress the children experienced in consequence of being either male or female and the peer group stigmatization that flowed from their behaviours.

In the past decade, there has been increasing controversy over whether GID of childhood should remain in the list of disorders. In the same period in which GID of childhood was introduced, homosexuality was removed. As our prospective study revealed that a substantial majority of boys with GID matured into homosexual men, to some critics the inclusion of GID was seen as a backdoor through which homosexuality reentered the list of disorders. A response to this concern is that when the subjective distress of being male or female present in children with GID disappears the person no longer has a disorder, whether a heterosexual or homosexual adult. On the other hand, when the distress of being male or female persists, the diagnosis remains GID as seen in adolescence or adulthood, commonly termed transsexualism.

Inclusion of GID for children in the list of disorders is also seen by some critics as perpetuating sex stereotyping in society, and demanding that children conform to traditional masculine/feminine behaviours. A response to this is that the diagnosis is not made merely for gender non-conformity but only when the child is unhappy being male or female, and where the child's behaviours are so atypical that there is substantial adverse reaction from the peer group.

Initial assessment

In the initial assessment of children with suspected GID, the professional should attempt to engage both parents as well as the child. Frequently there is reluctance by one parent, usually the father, to attend. However, this is a family matter, and the clinician needs to gain impressions of the parent-child relationship, the parent-parent relationship, and the child's behaviour from both parents as well as the child.

Assessment is directed towards understanding whether the behaviours described represent a normal variant of psychosexual development. Does the child overtly express dissatisfaction being the sex to which he or she was born? Is there a marked skewing of gender-typed behaviours towards those of other-sexed children or is there some mix? How long has there been cross-gender behaviour? What have parental reactions been to it initially and more recently? What is the child's response when parents attempt to frustrate the cross-gender toy or clothing preferences, if such attempts have been made? What time availability is there for each parent with the child? What do they do together? Are there other persons, for example grandparents or teachers, who may be reinforcing cross-gender behaviours? What are the parental concerns, both in the short and long term, with respect to the significance of the behaviours? Are there other behavioural or medical problems in addition to the gender identity issue?

Treatment

Typically, three principal targets are set for intervention with GID in children. First, the children are unhappy being the sex to which they were born; second, they are experiencing substantial peer group alienation; third, there is conflict with one or both parents in consequence of their atypical behaviour. A principal intervention strategy is helping the child understand that the world of gender is not necessarily black and white, but that greys exist as well. Boys can understand that not all boys need to be good athletes or rough-and-tumblers, and that boys can be sensitive and creative. Girls do not have to be boys to participate in rough-and-tumble play and sports. Children do not need to conform arbitrarily to all sex-typed attributes to remain in their birth sex role. To the extent this can be internalized, the path to transsexualism may be blocked.

The peer group of children with GID can be expanded to include children of both sexes. Parents may have to make efforts, particularly with cross-gendered boys, to find boys of their son's age who will enjoy non-athletic non-rough-and-tumble companionship, perhaps engaging in board games or computer games together. Similarly, girls with GID who are very athletically motivated may find girls who are also athletically inclined, and not just boys to play with. Children who develop comfort in socializing with both boys and girls may experience enhancement of same-sex identification.

Cross-gendered boys are notably alienated from their fathers and intervention can promote their relationship by finding mutually enjoyable activities. This may serve as a source of same-sex identification in the child, will enhance the quality of the parent-child relationship, and will be a positive outcome irrespective of its influence on later sexual identity.

Very few adult transsexuals had entered into any treatment intervention to address GID during childhood. Children with GID referred for evaluation or treatment may, as a product of that concern by parents, and/or professional intervention, have that route to transsexualism diverted. However, there is no empirical support for intervention directed at emerging sexual orientation. There is no evidence that a specific type of 'treatment' in childhood has any effect on outcome on that dimension of gender identity.[34] Parents should understand that if they are concerned about the ultimate sexual orientation of their child, that is a long time ahead. For the immediate period the child is unhappy who he or she is, is experiencing conflict with the peer group, and may be having difficulties at home with at least one parent. These are concerns that should be addressed.

Cross-gender living by children

In recent years, some children with GID have been permitted to live as children of the other sex. Their parents consider that the

strong preference by their child for the dress, activities, and companionship of the other sex with aversion to conventional sex-typed activities, along with the stated preference for being the other sex, argues for the child expressing its gender needs. Complexities of this decision include integrating it into the child's school and neighborhood environment.

Typically, children with GID experience peer group stigma and domestic conflict in consequence of their gender identity. Reduction of conflicts could enhance self-esteem. This social experiment should provide information on whether the longer-term status of the children differs from the children with GID not permitted cross-gender living, most of whom mature into homosexual adults and a minority into transsexual adults.

Early adolescent gender identity disorder

GID continuing into adolescence merges with GID of adulthood. Management issues address the young teenager's continuing gender dysphoria and the consequent social problems. There may be peer group alienation. Depression may develop. School avoidance may develop. Awareness of sexual attraction to same-sex persons may be an additional source of conflict. Parents may be unaware of their teen's GID.

GID in adolescents presents medical, legal, and ethical dilemmas for clinicians. The somatic changes of puberty are very distressing to these young teenagers. And for those who will ultimately progress to adult GID or transsexualism, these changes may pose substantial obstacles to effective 'passing' in their desired gender role. The latter is especially true for males as the voice deepens, facial hair sprouts, and skeletal proportions masculinize. For females, menses are especially troublesome, though not visible, and breast development, especially when prominent, is very distressing.

Clinical recognition of these issues has led to an innovative program for some young teens with GID. This is a trial period of putting puberty on hold and possibly a later cross-sex hormone induced puberty. In the Netherlands, treatment may consist of administering a gonadotrophin releasing hormone agonist (GnRH analogue) at Tanner Stage 2-3 to block secretion of sex steroids that promote pubertal changes. This could be at age 12–14. Depending on the clinical picture of gender identity, at age 16, cross-sex steroids may be administered, or the analogue withdrawn so that endogenous puberty continues.[37] UK practice disfavors analogue treatment prior to Tanner Stage 4 or 5 after substantial pubertal changes. In the early experience of the Dutch treatment program, no patients who have commenced hormonal treatments prior to age 18 have regretted the decision to live as a person of the other sex. Although there is concern that a couple of years of gonadal steroid suppression could predispose to osteoporosis, this concern remains theoretical.

Legally, there is no age barrier to a minor consenting to a medical intervention in the United Kingdom, providing that there is sufficient understanding of the implications of the treatment.[38] At 16 years, adolescents are presumed competent to consent to medical treatment..

The psychiatric management dilemma here is predicting which gender dysphoric adolescents will mature into adult transsexuals, and which will be able to live in the gender role expected from birth, perhaps as homosexual adult men and women.

Further information

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9.3

Situations affecting child mental health

Contents

9.3.1 The influence of family, school, and the environment

Barbara Maughan

Introduction

Like adult disorders, most child psychiatric problems are now regarded as multifactorially determined: both genetic and environmental factors play a role in their development. This chapter provides an overview of some of the key environmental elements in that equation. Subsequent chapters discuss risks for specific disorders; the focus here is on the more general issues that arise when considering the effect of environmental influences on the onset or persistence of psychopathology in childhood.

Environments and development

As in all aspects of child psychiatry, a developmental perspective is crucial when considering environmental risks. Some developmental periods may be especially sensitive for neurodevelopment, and show heightened effects of environmental insults. In addition, key sources of environmental influence change with age, and the meaning and impact of events will vary with the child's stage of cognitive, emotional, and social development. The family is the central source of early environmental influences, charged as it is in most societies with prime responsibility for the care, nurture, and socialization of the young. As children develop, so their social worlds expand; childcare and school settings take on increased importance, as do relationships with friends and peers. Throughout, each of these proximal contexts is shaped by influences from the wider culture and society. Any comprehensive assessment of a child's environment needs to take each of these types and levels of influence into account.

Nature-nurture interplay

At one time, causal associations between adverse experiences and childhood disorder were assumed to run in just one direction. Today, it is clear that the situation is vastly more complex. Children are not simply passive recipients of experience; they influence, as well as being influenced by, those around them, and they play an active role in constructing and interpreting their social worlds.[1] Even very young infants influence the nature of their interactions with caregivers, and children's capacities for shaping and selecting their experiences increase as they mature. The temperamentally difficult child is likely to evoke more negative responses from parents; when parents themselves are under stress, or find it hard to maintain consistency, troublesome child behaviours can play a key role in fuelling harsh or punitive responses. Delinquent adolescents may seek out delinquent peers, who further encourage their antisocial activities. Associations between environmental factors and disorder often involve complex reciprocal patterns of effects.

Some of the evocative effects of children's behaviour will reflect heritable traits.[2] The advent of behaviour–genetic studies in child psychiatry has provided important insights into environmental as

well as genetic risks. Genetic analyses have shown, for example, that many ostensibly 'environmental' factors include some element of genetic mediation.[3] Parents provide children not only with their environments but also with their genes, so that in biologically related families, nature and nurture are inevitably interwoven. Musical parents will encourage their children to enjoy music, buy them a violin, and may also pass on musical talents. In a similar way, antisocial parents may rear children in hostile and punitive environments, provide models of antisocial behaviour, and also pass on genes that predispose to disruptive behaviours. In all likelihood, genes and environments will often be *correlated* in this way.

Genetically informative studies have also highlighted other key mechanisms in ***gene-environment interplay***.[4] First, environments may *moderate* genetic influences, such that the heritability of some traits may vary systematically with qualities of the environment. Second, genetic factors may contribute to *differential sensitivity to environmental risks*. Research has consistently shown marked individual differences in children's responses to all but the most severe forms of psychosocial adversity. As yet, reasons for these differences are not well understood. They may reflect variations in the severity of exposure; individual differences in resilience or coping strategies, or in environmental sources of protection; or variations in vulnerability. Genetic predispositions clearly constitute one source of such vulnerability, and several examples of gene x environment interactions have now been documented. Finally, pre-clinical studies provide clear evidence that environments can influence *gene expression* through *epigenesis;* as yet, the extent to which processes of this kind apply in humans is unknown.

Risk variables and risk mechanisms

Identifying environmental factors that show links with children's adjustment is only the first step in understanding *how* they function to increase risk for disorder. A variety of different mechanisms has been proposed here. Some may run through the effects of stress on the biological substrate. Exposure to aggression and hostility may influence children's cognitive processing, leading to the development of negative cognitive sets and attributional biases. In a related way, disrupted early attachments are argued to affect the psychological structures needed for later relationship formation. Adverse experiences may lead to direct increases in negative emotionality, disruptive behaviours, and impulsiveness, or to negative interactional styles that impact on social relationships. And finally, stress may affect children's self-concepts, or compromise their coping skills in ways that increase the risks for disorder. Any given environmental risk may be associated with a number of risk mechanisms, and the processes involved in the persistence of disorder may differ from those involved in its onset.

Family influences

Pre- and early post-natal development

Some vulnerability to psychopathology is laid down in foetal development. The potential for adverse effects of maternal substance use on the developing foetus have been known for many years; much recent attention has focussed on associations between prenatal cigarette smoking and risk for externalizing disorders in offspring. In addition, current estimates suggest that as much as 15 per cent of the load of childhood emotional/behavioural problems may be attributable to exposure to maternal anxiety and stress in pregnancy. Though the mechanisms involved here remain to be elucidated, there is speculation that these effects may reflect foetal programming of stress response systems akin to those posited in studies of early life influences on risk for cardiovascular disease.

Post-natally, as children progress from the complete dependence of infancy to increasing independence, they need stable and secure family relationships to provide emotional warmth, responsiveness, and constructive discipline. The influential work of Bowlby[5] and others has shown that a child's need to be attached to others is a basic part of our biological heritage. Infants become increasingly socially responsive over the first 6 months of life. At 6 to 8 months of age they begin to form selective attachments to particular individuals; they seek proximity to these attachment figures if distressed or frightened, and protest if the person they are attached to leaves. In evolutionary terms, these behaviours function to provide protection for the infant, and to reduce anxiety and distress.

Almost all infants—even those neglected or maltreated by their carers—develop attachment relationships of this kind. Their quality varies, however, depending on characteristics of the parent, the child, and the mesh between the two. Infants who have received sensitive and responsive care tend to show ***secure*** attachment patterns; ***insecure*** attachments are more likely to develop when parents themselves are stressed or unsupported, and are unresponsive to their children. Two main types of insecure attachment have been identified: avoidant attachments (associated with rejecting or highly intrusive parental care) and resistant–ambivalent patterns (associated with inconsistent or unresponsive parenting). More recently, a third disorganized category has been described, in which infants show a variety of contradictory behaviours after brief separations, and often appear confused, depressed, or apprehensive. This seems especially associated with parental behaviours that are frightening, unpredictable, or abusive.

Attachment theorists argue that the quality of these early relationships may have long-term implications. Though not entirely resistant to change, infants' attachment patterns do tend to be stable over time. Some of this stability may reflect continuity in the quality of family care. In addition, attachment theory proposes that early attachment experiences are internalized in internal working models of self and others, which function as templates for future relationship formation. Children who have experienced responsive early care come to expect others to be caring and reliable; those who have been ignored or rejected develop less positive expectancies of others, of relationships, and of themselves. Later in development, new relationships may be created in line with these expectancies.

Although many aspects of these models await confirmation, securely attached infants are known to go on to be more sociable and co-operative in their social relationships, and to show more positive affect and self-esteem. Insecurely attached infants show less positive relationships, and are at some increased risk for psychopathology. Taken alone, attachment security in infancy is only a weak predictor of global functioning in early adulthood, suggesting that early attachment experiences work with and through other experiences—including peer relationships, later family experiences, and eventually mature intimate relationships—to contribute to later functioning. In addition, both ICD-10 and DSM-IV recognize two varieties of attachment disorders: non-attachment with emotional withdrawal, typically associated with abuse, and non-attachment with indiscriminate sociability, most usually observed when

children have been exposed to repeated changes of caretaker or institutional care. Although as many as 40 per cent of infants receive insecure attachment classifications, these more severe forms of attachment disorder are rare.

Family relationships and parenting

Many other aspects of family life and relationships, and of parenting styles and behaviours, have been examined for their impact on children's development. Research on families emphasizes the complexity of family relationships; each dyadic relationship is influenced by other relationships in the family, and normative transitions in family life—the birth of a sibling, or mother starting work—reverberate to affect all family members.[6] Relationships with parents and siblings change as children develop, and both these, and specific aspects of parenting, may impact on risks for disorder.

The implications of the most severely compromised parenting, involving abuse or neglect, are examined in Chapter 9.3.3, and family-based risks for individual childhood disorders are discussed in detail in the chapters dealing with each specific condition. In general, these reflect four broad themes:

- discordant, dysfunctional relationships between parents, or in the family system as a whole;
- hostile or rejecting parent-child relationships, or those markedly lacking in warmth;
- harsh or inconsistent discipline;
- ineffective monitoring and supervision.

Within this broad pattern, differential treatment of siblings is known to increase conflict between children, and may have important implications for psychopathology. In addition, outcomes are markedly poorer when children face multiple family-related risks.

Family life can also provide important sources of protective influences for children facing life events and other stressors. Cohesion and warmth within the family, the presence of one good relationship with a parent, close sibling relationships, and the nature of parental monitoring and supervision have all been found to show protective influences of this kind.

Parent and family characteristics

Psychopathology in parents is associated with increased risks of emotional and behavioural problems in children. Recent estimates suggest that as many as 60 per cent of the children of parents with major depression will develop psychiatric problems in childhood or adolescence, and their risks of affective disorder are increased fourfold. Psychosis, alcohol and drug abuse, and personality disorders in parents are also associated with increased risks of disorder in offspring, and parental criminality is a strong risk factor for conduct problems and delinquency.

In most instances, these links will reflect a complex interplay between genetic and environmental effects. Disorder in parents is frequently associated with disturbed marital relationships, and parental psychopathology may also impair parenting capacities. Depressed mothers, for example, are less sensitive and responsive to their infants, and attend less, and respond more negatively, to older children. Alcohol and drug abuse and major mental disorders in parents may impair parenting in more wide-ranging ways. When parents are antisocial, effects may also be mediated through the endorsement of antisocial attitudes and social learning.

Young maternal age is associated with increased risk for child and adolescent conduct problems. In part, these associations are likely to reflect the educational and social disadvantages that predict very early parenthood; in part, the poor social conditions and lack of support faced by many young mothers; and in part, less than optimal parenting styles. Delinquency is also associated with large family size. Once again, the more proximal risks involved are likely to be complex: parental supervision may be less effective in large families, and opportunities to 'learn' from delinquent siblings higher. Beyond this, family size shows few consistent links with childhood disorder. Only children are not at increased psychiatric risk, and they share with other first-borns some small advantages in terms of cognitive development. Birth order also appears to have few implications for behavioural adjustment, although youngest children show some increased rates of school refusal.

Changing family patterns

Recent decades have seen massive changes in the pattern of many children's family lives. The most obvious markers are the dramatic increases in rates of divorce, single parenthood, and step-family formation, along with major increases in maternal employment. In the years immediately after the Second World War, just 6 per cent of British couples divorced within 20 years of marriage. By the mid-1960s that figure had increased four-fold, and divorce rates continued to rise into the 1980s. For most children, parental divorce will be followed by a period in a single-parent household; for a substantial minority, further family transitions will mean that they become part of a step family. In the early years of the 21st century more than 10 per cent of UK families with dependent children were step-families, and approaching a quarter of children lived in single parent households.

Parental divorce

There is now extensive evidence that divorce is associated with negative consequences for children.[7] Psychological and behavioural distress are common, especially in the period immediately following divorce; more severe disturbance is not. Boys in particular are at increased risk for conduct problems. Educational attainments and motivation are often compromised, and subsequent relationships may also be affected. As they approach adulthood, children of divorce move into close relationships earlier than their peers, but also experience higher risks of relationship breakdowns.

Events both before and after the separation seem central in understanding these effects. Longitudinal studies, for example, have shown that children in divorcing families often show disturbed behaviour well before their parents separate. Exposure to the discord and conflict that frequently precede divorce thus seem to be key components of risk. After separation, problematic relationships between parents may continue, and the parents' own distress may compromise their capacity to respond sensitively and consistently to their children's needs. Many families face a sharp decline in economic circumstances after divorce, and for many children their parents' separation may involve house moves, school changes, and other disruptions to their established social networks. Each of this constellation of factors may contribute to subsequent outcomes.

Single parents and step families

Research on the effects of growing up in single-parent and step families illustrates the complexity of family-related influences.[8]

Overall, children in single-parent and step families show higher mean levels of emotional and behavioural problems than those in non-divorced two-parent families; they also have an increased probability of health problems and educational underachievement. But there are also marked differences within each family type and associations between the quality of mother–child relationships and children's adjustment is similar across family settings. In addition, single-parent and reconstituted families often differ from stable two-parent families in a plethora of other ways; in particular, they are much more likely to face economic pressures, poor social support, and higher levels of maternal depression. Once these variations and the degree of negativity in family relationships are taken into account, family type *per se* shows few consistent links with children's adjustment.

Peer influences

Beyond the family, relationships with peers are now recognized to provide a unique and essential contribution to children's social, emotional, and cognitive development.[9] By the end of the pre-school period most children have at least one reciprocated friendship. In childhood and adolescence, peers take on increasing importance; in middle childhood, more than 30 per cent of children's social interactions are with peers, and adolescents are estimated to spend more than twice as much time with peers than they do with parents or other adults. The functions of friendship change with development, expanding to encompass companionship and stimulation, help and sharing, social and emotional support and intimacy.

With friends and peers children acquire skills, attitudes, and experiences that contribute to many aspects of their adaptation. By the same token, children who have poor social skills, or who are rejected or neglected by peers, are at risk of a range of adverse outcomes including poor school performance, school drop-out, and psychiatric disorder. Social rejection may increase children's feelings of loneliness, reduce supports that can buffer against stressors, and also mean that isolated children miss out on important social learning experiences. Since many children with psychiatric disorders also show difficulties in relationships with peers, processes of this kind may well compound their problems. In adolescence, affiliations with behaviourally deviant peers have attracted particular interest as correlates of conduct disorder and delinquency. Here, reciprocal influences have been demonstrated: aggressive disruptive children are more likely to associate with deviant peers, but relationships with peers also show an independent effect on both the onset and persistence of delinquency.

Child care and schooling

By the late 1990s approaching 50 per cent of mothers in the UK returned to full- or part-time work before their infants reached one year of age. This major increase in early maternal employment has prompted extensive research on the impact of alternative childcare on children's development. Recent evidence[10] suggests that multiple features of early care need to be considered in assessing effects. Higher child-care ***quality*** (as indexed by features such as sensitive and responsive care-giving, and cognitive and language stimulation), is associated with improved performance on tests of cognitive, language, and early academic skills, and with more prosocial skills and fewer behaviour problems, By contrast, higher ***quantity*** of child care (as indexed by hours per week in any kind of non-maternal care), is associated with some increased risks of problem behaviours in both the preschool and early school years.

School life then brings its own demands and challenges. Starting and changing schools are significant, sometimes troublesome, events for children; although most young children adapt well, a significant minority show some disturbance when they start school, and both attainment levels and self-perceptions are affected for many young adolescents after the transition from primary to secondary school. Tests and examinations rank high on children's lists of fears, and levels of psychological distress are elevated at times of major examinations. Although fears of this kind are not generally severe, they do show links with clinically significant symptoms. Bullying is a further problem especially associated with the school context. Self-report surveys suggest that over 15 per cent of young children experience some bullying at school, mostly unknown to parents or teachers. Although rates fall with age, up to 5 per cent of adolescents continue to face bullying in secondary school. Persistently victimized children have identifiable characteristics, with histories of anxious insecure behaviours and social isolation often beginning before they started school; bullying then increases their risks of adjustment problems.

Like families, schools differ in their atmosphere and social climate, and these variations show an independent impact on children's academic progress and behaviour. In part, these variations reflect differences in initial pupil intakes. In addition, they show systematic links with organizational characteristics of schools. Schools with more positive outcomes are characterized by purposeful leadership, constructive classroom management techniques, an appropriate academic emphasis, and consistent but not oversevere sanctions. In relation to behavioural outcomes, the composition of pupil groupings may also be influential. Young children are more likely to become aggressive if placed in highly aggressive classes, and risks of delinquency are increased in secondary schools where intakes include large proportions of less able children. For some severely disadvantaged groups, however, schooling may offer an important source of positive experiences. Experimental studies of preschool programmes, for example, have shown important long-term gains in terms of reduced risks of delinquency and unemployment many years after participants left school.

Wider social and environmental influences

Poverty and social disadvantage

Poverty and social disadvantage are most strongly associated with deficits in children's cognitive skills and educational achievements.[11] In the behavioural domain, disruptive behaviours also show links with family poverty. Effects appear to be more marked for boys than for girls, and seem to be stronger in childhood than in adolescence. Intermittent hardship is associated with some increased risk for conduct problems, but the impact is most marked for children in families facing persistent economic stress. Most current evidence suggests that these effects are indirect. Poverty imposes stress on parents, and reduces the supports available to them; these in turn increase the risks of harsh or coercive parenting, and reduce parents' emotional availability to their children's needs. Some studies suggest that relative deprivation—the perception that one is disadvantaged by comparison with others—may be more important than income levels *per se*.

Neighbourhood and community contexts

Rates of childhood disorder vary in different neighbourhoods and communities. Urbanization is frequently associated with increased risks of disorder, and rates may be especially high in chronically disadvantaged inner-city neighbourhoods. In early childhood, many of these effects seem to be indirect; neighbourhood disadvantage increases stress on families, and these in turn largely account for associations with children's difficulties. In severely disadvantaged settings, however, even quite young children may be directly exposed to community violence, and in adolescence, neighbourhood influences may be mediated through associations with delinquent peers.

Multiple stressors

For many children, exposure to these differing types of adversity will covary. Stressed families frequently live in poor neighbourhoods, where schools are under pressure and peer groups deviant. Early epidemiological findings suggested that isolated single risks have relatively little impact on disorder, but that rates rise sharply when risk factors combine. More recently, studies have shown that child, sociocultural, parenting, and peer-related risks each add uniquely to the prediction of behaviour problems. In addition, the total number of risks a child faces explains further variance in outcomes.

Secular trends in disorder and psychosocial risks

Finally, it is important to consider how psychosocial risks may impact on overall levels of disorder. There is now clear evidence that rates of many adolescent disorders—including depression, suicide, alcohol and drug use, and delinquency—have risen since the Second World War.[12] Since it is implausible that changes in the gene pool could occur so rapidly, environmental risk factors must be implicated. Some of these may overlap with risks for individual differences in disorder, but others may be quite distinct. Based on an extensive review of available evidence, Rutter and Smith[12] concluded that a variety of factors are likely to be implicated:

1 increased rates of family breakdown, with their associated effects on the disruption of relationships and exposure to conflict and discord;

2 a change in the meaning of adolescence, with prolonged education and economic dependence on parents occurring alongside increased autonomy in other spheres;

3 a possibly increased disparity between young people's aspirations and the opportunities available to meet them;

4 increased alcohol consumption and illegal drug use;

5 changing social attitudes to acceptable behaviour, possibly enhanced by influences from the mass media.

Other specific factors may affect rates of juvenile crime. In particular, the increasing commercialization of youth culture, providing more goods to steal, may have coincided with diminished surveillance and increased situational opportunities for property crime.

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9.3.2 Child trauma

David Trickey and Dora Black

Children's reactions to traumatic events

This chapter will focus on the impact on children of traumatic events other than child abuse or neglect, which are covered in Chapter 9.3.3. According to the DSM-IV-TR definition of post-traumatic stress disorder (PTSD), traumatic events involve exposure to actual or threatened death or injury, or a threat to physical integrity. The child's response generally involves an intense reaction of fear, horror, or helplessness which may be exhibited through disorganized or agitated behaviour. Terr suggested separating traumatic events into type I traumas which are single sudden events and type II traumas which are long-standing or repeated events.[1]

If the traumatic event includes bereavement, the reactions may be complicated and readers should consult Chapter 9.3.7 to address the bereavement aspects of the event.

Following a traumatic event, children may react in a variety of ways (see Chapters 4.6.1 and 4.6.2 for the adult perspective on reactions to stressful and traumatic events). Many show some of the symptoms of post-traumatic stress disorder—re-experiencing the

event (e.g. through nightmares, flashbacks, intrusive thoughts, re-enactment, or repetitive play of the event), avoidance and numbing (e.g. avoidance of conversations, thoughts, people, places, and activities associated with the traumatic event, inability to remember a part of the event, withdrawal from previously enjoyed activities, feeling different from others, restriction of emotions, sense of foreshortened future), and physiological arousal (e.g. sleep disturbance, irritability, concentration problems, being excessively alert to further danger, and being more jumpy). In young children the nightmares may become general nightmares rather than trauma-specific. Other reactions to trauma in children are:

- becoming tearful and upset or depressed
- becoming clingy to carers or having separation anxiety
- becoming quiet and withdrawn
- becoming aggressive
- feeling guilty
- acquiring low self-esteem
- deliberately self-harming
- acquiring eating problems
- feeling as if they knew it was going to happen
- developing sleep disturbances such as night-terrors or sleepwalking
- dissociating or appearing 'spaced out'
- losing previously acquired developmental abilities or regression
- developing physical symptoms such as stomach aches and headaches
- acquiring difficulties remembering new information
- developing attachment problems
- acquiring new fears
- developing problems with alcohol or drugs.

Such problems may individually or in combination cause substantial difficulties at school and at home. The reactions of some children will diminish over time; however, for some they will persist, causing distress or impairment, warranting diagnosis, and/or intervention. Research predicting which children will be more likely to be distressed following a traumatic event suffers from a number of methodological flaws. However, factors which are often identified as constituting a risk for developing PTSD across a number of studies include: level of exposure, perceived level of threat and peri-traumatic fear, previous psychological problems, family difficulties, co-morbid diagnoses, subsequent life events, and lack of social support.

Diagnosis of PTSD

Both DSM-IV-TR and the ICD-10 diagnostic classification of PTSD are appropriate for use with adults and although children from 8 years old do display similar symptoms to adults[2] there are some developmental differences particularly in younger children.[3] Alternative diagnostic criteria for pre-school children have therefore been developed, which draw on reports by carers and include more behavioural symptoms such as loss of developmental skills, and development of new fears or anxiety.[4]

Other diagnoses

Careful assessment is required to make an accurate differential diagnosis. According to DSM-IV-TR, PTSD can only be diagnosed 1 month after the event, prior to that a diagnosis of acute stress disorder (ASD) may be appropriate. Whereas ICD-10 PTSD can be diagnosed within the first month, and the acute stress reaction is reserved to describe a disturbance that resolves rapidly. If the event is not of sufficient severity to meet the criteria for PTSD and the reaction does not last more than 6 months after the stressor has ceased, then a diagnosis of adjustment disorder may be appropriate. Recovery may take longer for children if their parents continue to suffer from symptoms of PTSD which may constitute a chronic source of stress which may in turn prolong the symptoms of the child. Further information on these diagnoses from an adult perspective can be found in Chapters 4.6.1–4.6.5.

Other diagnosable disorders may result from traumatic events, and may be present singularly or co-morbidly with PTSD; 60 per cent of children with PTSD have a co-morbid mental health diagnosis.[5] According to Fletcher's meta-analysis, common co-morbid diagnoses are: anxiety disorders, depression, alcohol and drug abuse in adolescents, and attention deficit hyperactivity disorder (ADHD).[2]

Assessment

As with other psychiatric disorders, the best assessment can be made by integrating information from a number of sources such as an interview of the parent/carer alone, an interview of the family together, information from school, and information from psychological measures (see below). Careful consideration should be given to which members of the family will be involved in any interviews so as to avoid exposing previously unaffected children to the traumatic details of the event. Sometimes children try to 'protect' their carers from distress by under-reporting their symptoms of trauma, it is therefore also essential to interview the child on their own where possible.[6]

In order to assess what elements of the child's current functioning and distress may be a result of the traumatic event, and those that may pre-date it, it is important to gain as full a picture as possible of their developmental history and their pre-morbid functioning. Reports from teachers and other professionals may be particularly useful in this respect.

On assessment, some account of the traumatic event is necessary so that the clinician can gain an understanding of what exactly was experienced. Furthermore, it is helpful to give the sense that the clinician can bear to hear a story which the child and family may have been avoiding to tell for some time. However, this must be balanced against the child's understandable avoidance of the memory. There is little point gaining a full account of the event during the assessment, if the child becomes so distressed that they do not return for treatment. Pynoos and Eth offer a structure for conducting such an initial assessment which begins with a projective drawing and storytelling. It then proceeds to discussion of the actual event and its impact, followed by closure.[7] If the assessment has included talking about the traumatic event the child and family may become very distressed, and it may be necessary to invest some time in winding down the session, so that the family does not leave overly distressed. This will increase the likelihood of them engaging in the treatment process, which is likely to involve thinking through the event—something which they often do not intuitively want to do.

Assessment includes asking about the motivation for the contact with the service. Unlike many other problems, families with traumatized children may not simply present at the service in order to effect a change in their child's symptoms. They may be asking for advice following a traumatic event to try and prevent problems from appearing later by ensuring that they are 'doing the right thing', or they may be seeking an assessment for the purpose of compensation or other legal purposes.

Psychological instruments

A number of questionnaires and diagnostic interviews are available to assist in the assessment of PTSD. Such instruments cannot replace a clinical interview, but may assist by strengthening clinical opinion and giving a reliable and valid indication of perceived symptom severity or frequency. This can give an indication of the impact of an event, can raise hypotheses which can be further investigated (e.g. screening for possible presence of PTSD which can then be further assessed) and if repeated after treatment can help to measure change. Ohan and colleagues reviewed those measures used in research studies that have adequate psychometric properties.[8] The best diagnostic instrument is the children's PTSD inventory. This is a structured diagnostic interview, the psychometric properties of which are good and have been published in peer-reviewed journals.[9,10] The Children's Revised Impact of Events scale[11] is a useful self-report questionnaire of 13 items appropriate for children aged 8 years and above. It has good screening properties[12] and is freely available from the Children and War Foundation's website (see below). Similarly Foa's Child PTSD Symptom scale (CPSS) is a 24-item self-report questionnaire designed for children aged 8 years and above. It is emerging as having good psychometric properties.[13] The CPSS has the advantage of specifically assessing the symptoms of PTSD from the DSM-IV-TR, and unlike most other self-report measures, it also addresses the important question of the impact of the symptoms on the child's functioning.

Evaluation of treatments

An increasing amount of evidence indicates that trauma-focussed cognitive behavioural therapy (TF-CBT) is the treatment of choice for PTSD.[14] Much of the evidence is from studies of children with PTSD as a result of child sexual abuse. Dalgleish and colleagues summarize the evidence thus; 'The current take-home message from this nascent literature therefore is that CBT appears to have well-established efficacy in treating a range of post-traumatic stress responses following sexual abuse, with preliminary evidence in favour of this form of intervention following other types of trauma'.[15] Consequently, TF-CBT is the only intervention recommended by the United Kingdom's National Institute for Health and Clinical Excellence (NICE) for the treatment of children and young people with PTSD.[6]

Family therapy and psychodynamic psychotherapy as stand alone therapies for children with PTSD are not currently supported by the same level of evidence as TF-CBT. However, there is evidence to illustrate the importance of involving parents or carers in treatment where appropriate.[16] Psychodynamic psychotherapy may have some contribution to the treatment of traumatized children[17] and clinical experience indicates this to be true particularly in children with co-morbid diagnoses or where the traumas are multiple and prolonged (type II) such as in war and civil conflicts, and have led to failures of basic care.

Eye Movement Desensitisation and Reprocessing (EMDR) has proven to be effective with adults and has increasingly been used with children.[18] It shows promise for use with children and young people, but it has not yet established the same level of evidence as TF-CBT.[6]

The available evidence does not currently support the use of medication to treat PTSD in children or young people,[6] although medication may be necessary to treat any co-morbid conditions such as depression, ADHD, or sleep problems.

Management

Trauma-focussed cognitive behavioural therapy (TF-CBT)

'Processing' of an event involves bringing the event to mind and thinking it through often by talking, writing, or playing. This enables the memory to be stored as an ordinary narrative memory which is under the child's control, rather than as the vivid sensory information of the original event which is prone to being involuntarily re-experienced. Furthermore, processing enables a more helpful meaning to be attributed to the event; so whilst it may well be thought of as 'terrifying' and 'unfortunate', it no longer colours the way that *everything* in the child's world, including him or herself is seen.

Many children will process difficult and traumatic experiences naturally, often with the help of those around them such as their carers. Some may take a little while to do so. However, bringing up the memory may initially trigger the fear or distress of the original event, so some children avoid thinking about it, which in turn prevents processing.[19] Similarly the adults around the child may try to protect either themselves or the child from further distress by not discussing the event and so opportunities to enable the child to process it may be missed through avoidance by proxy. Therefore some children will not manage to process the event without professional help.

TF-CBT for traumatized children and families involves enabling the child or young person to bring the traumatic event to mind within the safe environment of therapy. This is akin to *exposure* to the memory, which enables the memory to be processed. *Cognitive restructuring* involves enabling the child to alter their unhelpful negative view of themselves or the world, which is based on the event, to a more helpful and realistic one. Cognitive behavioural therapy for children is further described in Chapter 9.5.3, Stallard provides excellent resources in the form of a workbook,[20] and Cohen offers a comprehensive description of the treatment of trauma and traumatic grief.[21]

Younger children with PTSD may not be able to make use of TF-CBT in the same way that older children can, and their treatment is likely to involve much more family work and ensuring that they learn that their environments are safe through re-assurance and stability.[22]

Early interventions

There continues to be much debate about the value of early intervention (i.e. within the first month). Research to date does not support the use of single session debriefing,[23] however, there may be value in the provision of practical and emotional support in the immediate aftermath, together with some education of what reactions can be expected. Development of a culture where the child is permitted, or encouraged to talk about the event (e.g. within the family or within the school) may protect against the development of PTSD.[24] Early trauma-focussed cognitive behavioural therapy may be offered to older children with the most severe symptoms[6]

which may be completed in groups (e.g.[25]). There may be a role for EMDR in the early stages following a traumatic event, although this has yet to receive empirical support.

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The Children and War Foundation. This is a charity which aims to improve the care of children affected by war and disaster. Two professional groups, the Center for Crisis Psychology in Bergen, Norway and the Institute of Psychiatry in London, UK, have been instrumental in setting up this foundation. Copies of the Children's Revised Impact of Events scale (CRIES), the Depression Self-rating Scale for Children and the Revised Children's Manifest Anxiety scale (RCMAS) are available free from the website in various languages. www.childrenandwar.org

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9.3.3 Child abuse and neglect

David P. H. Jones

Introduction

Child abuse and neglect (child maltreatment) is a combination of a consensus about what comprises unacceptable child rearing/care, together with what children have a right to be free from. This is made explicit in the United Nations Convention on the Rights of the Child,[1] which sets out basic rights and standards for judging children's welfare, including, but not limited to, maltreatment. It incorporates both maltreatment of children within families and that arising from wider social influences, including child labour and sexual exploitation, and children in war zones.[1]

Maltreatment affects the healthy and normal course of development. It causes deviation from an expected trajectory, preventing the developing child's negotiation of sequential tasks and disrupting normal transaction between different facets of development.[2] Therefore maltreatment is the very antithesis of adequate child care and rearing, posing a major public health threat.[3]

Adequate rearing of the young is such a fundamental activity that the state must be concerned with the overall welfare of children within its society; in family settings where they are normally brought up, and in schools, hospitals, and residential settings.

While the Convention provides a framework, several states have developed a children's ombudsman, with wide-ranging powers to oversee the status of children's welfare and to tackle obstacles to it.

There are laws within each society to regulate the care and welfare of children, specifying the consequences if children are maltreated. In England and Wales, the Children Acts 1989, and 2004 address the overall welfare of children, including those deemed in need of extra help and support, and provide a legislative structure for those children who are at risk of, or are actually being, significantly harmed (child maltreatment).

Countries vary in their response to child maltreatment. In the United States, any professional who has reason to suspect that a child is being maltreated is legally required to inform the local child welfare agency (mandatory reporting). Some countries in Europe (e.g. Belgium and Holland) have a system whereby child-maltreatment concerns are dealt with confidentially, through health and social care supportive systems, rather than through primarily legal methods. The United Kingdom lies between these extremes, but relatively closer to the United States model than to the 'confidential doctor' system. Whatever system is in place, it is clear from the scope of the problem of child maltreatment that multidisciplinary working is a core requirement.

A developmental-ecological model is the most useful conceptual framework, which draws together the various factors known to contribute or be associated with the predisposition, occurrence, course and effects of child maltreatment.[3,4] It incorporates individual and interpersonal factors, family influences, immediate neighbourhood ones, together with broader social influences on child rearing and care. However, these layers of increasing social complexity, which surround the individual child, are not static. In addition to transactions between factors, there are important influences historically, and subsequent to any maltreatment, which have an impact on outcome. This inclusive conceptual framework enables genetic and environmental factors to be integrated in a manner that can inform clinical assessment and intervention.

Types of maltreatment

Identification of different types of maltreatment may be necessary for social and legal purposes, but epidemiologically, co-occurrence of varieties of maltreatment is more usual than singularity.[3,5] Official registers often record predominant type or that perceived to be the most serious. This knowledge is one of several methodological problems that affect confidence in research findings. However separate types are retained here, for descriptive purposes, while encouraging the reader to consider likely overlap in individual cases.

Epidemiology

Accurate figures for incidence and prevalence are bedevilled by ascertainment and recording difficulties, including secrecy and shame which are often associated. These influences are illustrated by the wide gulf between incidence and prevalence rates.[3]

Incidence rates increase from reported cases to higher rates obtained from representative community samples. The incidence of significant violence to children varies between 50 to 90 per thousand across cultures in community samples, and dropping to the mid-20s per thousand for cases known to professionals working with children. Cases known to social welfare agencies departments only comprise a minority of these. However, officially reported maltreatment ranges from 2 to 12 per thousand in England, North America and Australia. Neglect is commonest (34 to 59 per cent of cases); physical abuse (15 to 28 per cent); sexual (10 to 28 per cent); and emotional (7 to 34 per cent).

Most prevalence figures for each of physical, emotional and neglect range between 5 and 10 per cent. The equivalent rate for contact sexual abuse is 10 per cent (15 per cent of girls; 5 per cent of boys). Children with a disability are three times more likely to be maltreated.[3]

Life-threatening maltreatment rates have remained relatively constant, currently 0.1 to 2.2/1000 children in industrialized countries, rising to two to three times this in low to mid-income countries. Children are at their most vulnerable during infancy and neonatal periods.

Child sexual abuse

Definition and clinical features

This is defined as sexual activities which involve a child and an adult, or a significantly older child. There are two elements: the sexual activities and the abusive condition.[6] Contact sexual activities include penetrative acts (e.g. penile, digital, or object penetration of the vagina, mouth, or anus) and non-penetrative acts (e.g. touching or sexual kissing of sexual parts of the child's body, or through the child touching sexual parts of the abuser's body). Non-contact sexual activities include exhibitionism, involving the child in making or consuming pornographic material, or encouraging two children to have sex together.

The abusive condition is founded on the premise that children cannot generally give consent to sex, because of their dependent condition. Consent can be difficult to assess in older children or if there is a small age gap between abuser and abused. Considering whether exploitation has occurred can aid this decision: it comprises misuse of authority or age differentials through deceit, unreasonable persuasion, coercion, or overt force.

Half the sexual abuse cases coming to the attention of welfare agencies involve penetration or orogenital contact. The proportion is less in community samples, because reported cases tend to be more serious in nature.

Abuse perpetrated by a caretaking adult normally consists of increasingly severe sexual contact over time, with parallel increases in coercion and threats to the child if the 'secret' is disclosed. As the physical acts and psychological climate worsen, so the child's reluctance to disclose the predicament deepens.

Diagnosis

The most common presentation is through a statement from the child.[7] Unless the child is responded to sympathetically at this point, they may be reluctant to reveal the full nature of their plight. More than half of those who are abused do not disclose the fact, especially if they are male.

Less commonly the child's behaviour can draw attention to abuse, particularly if the child shows sexual behaviour problems, either directed towards themselves or towards other children. However, behaviour and emotional difficulties are normally non-specific, occurring in about two-thirds of children. Older children and adolescents show behaviour difficulties which are unexpected for themselves or their peer group, including substance abuse, suicide attempts, running away from home, or becoming unpredictably

out of control. Not surprisingly, high rates of prior sexual abuse are noted among young people involved in prostitution.

Medical presentations do occur, for instance venereal diseases, evidence of acute assault, or an otherwise unexplained pregnancy.

Prior to investigation, one-third of reported cases are already known to child welfare agencies for other reasons. Children are more likely to disclose their predicament if they have first made a spontaneous statement to someone before being interviewed by professionals.

Child psychiatric services may assist social workers interviewing children and young people with a psychiatric disorder, or very young children. Other specialists should be enlisted for those with communication problems and learning difficulties. The aim of interviewing is to help a child describe their predicament whilst avoiding suggestion.[8] Child psychiatry also has a role to play in providing psychological treatments for symptomatic children and working with disturbed families.

Screening for the possibility of child sexual abuse increases recognition in both adult and child populations, revealing information that can be essential for psychiatric management. Adult services have a role to play in addressing psychiatric problems in family members, including treatment for paraphilias, often in conjunction with the probation service or other specialized provision.

Aetiological and background factors

(a) Characteristics of abused children

Sexual abuse affects children of both sexes and all ages. The most common age when children are abused is between the ages of 7 and 13 years, but up to one-quarter of reported cases comprises the under-fives. Race and socio-economic status are not major risk factors, but there are increased rates of sexual abuse among children living with parents who are emotionally unavailable, psychiatrically disturbed, violent, or who abuse alcohol or drugs.[9] Children from lower socio-economic groups are over-represented in child protection samples, but in adult retrospective surveys there is a weaker link with economic status. Children who have been in substitute care are at higher risk.

Girls are more than twice as likely to be victimized. Boys are less likely to be reported or discovered to have been abused during their childhood. Compared with girls, boys are more likely to be older when first victimized and to be abused by someone from outside the immediate family, and more likely to be abused by women or by offenders who are known to have abused other children. The risk of sexual abuse is almost doubled for children with a disability.[10]

(b) Characteristics of abusers

Most abusers are male, but up to 10 per cent of children are abused by a female, though this figure is higher when the victim is male. Of abusers, 70 to 90 per cent are known to the child, with family members comprising between a third and a half of those who abuse girls, and between 10 and 20 per cent of abusers of boys.

Up to one-third of children are abused by a person who is under 18 years of age. **Young abusers** are, on average, 14 years old, while their victims are 7 years old and usually known to them.[11] The abusers lack social skills and assertiveness, and show impulse-control problems, learning difficulties, and clinical depression. Their home environments are characterized by instability, family violence, and sexual problems in their parents. Parental loss or separation is common among adolescent abusers.

Between 20 and 50 per cent of abusers have a history of childhood sexual abuse themselves. Physical abuse histories are even more common, together with deprivation and periods of substitute care in childhood. These characteristics are common among other offenders for non-sexual abuse offences, and thus do not explain the aetiological pathways through which some young people and adults develop a pathway of sexual attraction or desire to sexually assault a child. Marshall and Barbaree[12] have drawn together psychological, biological, and social factors into an integrated theory of aetiology.

Abusers typically deny sexual abuse allegations. Even measures of penile tumescence in response to childhood imagery are unlikely to discriminate a denying abuser from a falsely accused man. Some psychological features are common among abusers but are unlikely to be definitive, prior to any admission of guilt.[13] The demarcation between intrafamilial and extrafamilial abusers is less sharp than originally thought, and mixed abusers are relatively common.

(c) Family aspects

Up to half of all cases are abused by someone outside the family. In the majority of these extrafamilial cases the abuser is known to the child and in a position of trust, either providing care or supervision, or involved in an educational or recreational activity with the child. Among within-family cases, the original stereotype-of a closed family with a controlling abusive father and mother who is collusive with her husband's abuse of her child-has been demonstrated to be inaccurate. Although such a pattern may be seen, a variety of family styles of functioning occur. However, investigators have found that families containing sexual abuse victims are less cohesive, more disorganized, and permit less healthy expression of emotion than comparison families.[14] These differences may pre-date the onset of sexual abuse or be a consequence of its occurrence.[9] Nonetheless, the observations are important for intervention purposes.

Support from non-abusive adult carers (usually mothers) in terms of belief, protection, and help for children to understand their victimization, is positively linked with the children's response to their experience.[15] This is important for assessment and intervention purposes, because there is a significant link between sexual abuse and markers of parent-child relationship difficulties, such as emotional unavailability, interparental conflict, parental mental health, and substance abuse problems.

(d) Course and prognosis

A wide range of psychological sequelae in childhood and adult life are associated with prior childhood sexual abuse (Table 9.3.3.1).[9,15] However, these are linked with the effects of both the quality of the family environment at the time of abuse, and the nature of subsequent life events.[16] In particular, factors such as family disharmony and violence, existence of other forms of abuse and neglect, and parental mental health difficulties, in addition to subsequent events, such as losses through death or separation, combined with the child's own method of coping with the abuse and ameliorative effects of positive school or social relationships, all contribute to outcome.

About one-third of children are symptom free. Approximately 10 per cent of children show worsening symptoms over time, including depression and post-traumatic symptoms. While effects on personality and social relationships can be disabling during development, other children are relatively unaffected.[16,17]

Table 9.3.3.1 Impairments and problems associated with childhood sexual abuse

	Childhood impairment	Adult impairment
Affective symptoms	Fears PTSD Depression	Anxiety PTSD Depression
Behaviour problems	Conduct disorder Sexualized behaviour Self-destructiveness Hyperactivity	Aggressive conduct Self-destructiveness Alcohol/substance abuse
Cognitive functioning	Educational problems Language difficulties	Educational underachievement
Personality and social adjustment	Self-esteem Attachment Peer relationships	Pregnancy under 19 years Sexual aggression Prostitution Parenting problems Somatization Personality disorder Revictimization Sexual problems

Physical abuse

Definition and clinical features

Physical abuse is the physical assault of a child by any person having custody, care, or charge of that child. It includes hitting, throwing, biting, inducing burns or scalds, poisoning, suffocating, and drowning.[3] In the United States and United Kingdom physical chastisement of children is commonplace, leading to problems of definition. In other parts of Europe and in some Eastern cultures physical chastisement is regarded as unacceptable. Legal definitions in the United States and Western Europe normally link physical acts to observable harm. However, for research and clinical purposes an endangerment-based definition is preferable, because of the widely different sequelae resulting from similar assaults.[18] Failure to prevent injury or suffering is preferably considered a manifestation of neglect. Other definition problems include the frequency or repetitiveness of the acts, their severity, and whether intent to harm should be included. In addition, developmental factors affect the recognition of abuse and possibly its definition also—a smack to the head of an 8-year-old, although unacceptable, will have significantly different consequences from that to an 8-month-old.

The distinction between accidental injury, non-accidental injury, and specific medical diseases is sometimes straightforward (e.g. particular types of fractures, burns, or bruising) but difficult diagnostic dilemmas do occur. It is important to resolve these dilemmas so that the way forward for psychiatric assessment and treatment can be clarified.[19]

The 'battered child syndrome' refers to young children with multiple bruises, skeletal injuries, and head injuries, often accompanied by neglect, malnutrition, and fearfulness, whose parents deny responsibility.[20]

Diagnosis and recognition

Physical abuse is detected through the observation of physical injuries without an alternative non-abusive explanation.[19] Less commonly, a direct account comes from a child or a witness, or through confession by a parent or carer. Usually, the diagnosis is based upon a discrepancy between the physical findings and the history provided. The history may be insufficient or simply improbable. When an explanation is forthcoming, trigger events or developmental challenges are common—for example, persistent crying in infancy, problems of toileting or feeding among toddlers, or issues of discipline in later childhood. In adolescents, conflict surrounding independence may coincide with parental midlife crises. Not all physical abuse can be related to loss of control, however, and the assessor has to consider planned or even sadistic activities, such as scalding, burning, or torture.

There may have been previous episodes of similar or lesser concern, for which adequate explanations were unavailable at the time. Delay in presenting the child for medical attention is not a reliable diagnostic feature; neither is the apparent absence of parental concern nor their unreasonable behaviour at presentation.

Aetiology and background factors

(a) Child characteristics

Physical abuse occurs at all ages, although biological sequelae are more severe in infancy. There is no association with ethnic group, but a strong one with low socio-economic status among the under-fives, becoming weaker throughout childhood and disappearing by adolescence.[21] Children with developmental disabilities have a raised risk.[10] Associations with low birth weight, prematurity, or physical ill health disappear once parental and social variables are controlled for. Boys under 5 years of age are more likely to be abused, whereas girls are at greater risk in childhood overall.

(b) Abuser characteristics

Young maternal age at the time of the child's birth is linked with abuse, but generally the effect of age is overshadowed by low socio-economic status and high social stress.[21] Physical abusers of young children are likely to be female, but male abusers predominate during adolescence. They are more likely to be single parents and to have large numbers of closely spaced children. Their educational level, but not necessarily their intelligence, is lower; they are, however, more likely to be unemployed. Most physical abuse is perpetrated by parents, but others who adopt a caretaking role become increasingly significant in the abuse of older children.

Abusive parents are more likely to have had a childhood history of abuse themselves. However, regarded prospectively, 70 per cent of abused children do not abuse their own children.[22] Non-repeaters are more likely to have enjoyed social support from a partner, had a positive relationship with an adult during childhood, and to have received psychological help during adolescence. In addition, they have a more balanced and coherent perspective about their childhood experiences than those who show intergenerational continuity of parenting problems. The quality of attachment relationships between parents and children shows continuity, rather than the specific type of abuse. Hence, physically abused children have an increased risk of perpetrating both physical and sexual abuse when they become parents themselves.

Frank psychiatric disorder is relatively infrequent among abusers, but studies of physical abuse fatalities underline their importance in a minority of cases.[23] Personality difficulties and disorders are more common, however. Hostile adults with poor impulse control, low self-esteem, antisocial and aggressive personalities,

with accompanying mood disorder are more likely to abuse. These abusers have disrupted social relationships and inadequate coping responses in a wide range of domains. They are frequently socially isolated, alienated, and have disharmonious relationships with neighbours and relatives. For these adults, potentially protective supportive relationships with friends and relatives are inhibited.[3,21]

Abusive parents have maladaptive ideas about their children. They tend to have high expectations for their children's development and behaviour, perceiving it to be deviant when objectively it is not. They are more likely to believe in the appropriateness of strict physical discipline, and to hold negative views and perceptions about their children. They show limited attention to their children, less positive affect, and respond with aversion, anger, or irritation to their children's bids for care or attention, as well as to their positive behaviours, when compared with non-maltreating parents. Physically, abusers show heightened arousal to both child stimuli and non-child-related stressors.[3,21]

(c) Family aspects

Families in which physical abuse occurs are more likely to support mutually abusive coercive communications and interactions than controls. Partner abuse and domestic violence is relatively more common, combined with pervasive hostility and decreased cohesion. Discussion, positive displays of affection, and encouragement of prosocial behaviours are less common than in non-maltreating families.[3,21]

The quality of attachment between child and parent is significantly linked with physical abuse, especially when combined with high levels of social stress, low socio-economic status, and negative parental family attitudes and behaviours. Although infant temperament can be associated with maltreatment it probably only does so if combined with other risk factors, such as parent–child attachment problems, parental attitudes, and family difficulties of the sort described above. Clinicians have long observed that individual children can be perceived negatively by parents, without objective evidence, particularly if the child represents a particular issue or problem for the parent.

Course and prognosis

Some physically abused children have neurological and other physical sequelae as a result of their injuries.[4] Educational difficulties are consistently found on follow-up. The children are less attentive to social cues and less skilful at managing personal problems and more likely to attribute a hostile motivation to their peers, compared with non-abused children, at the age of 5. Their capacity for empathic concern with the everyday problems of their peers becomes blunted. Not surprisingly therefore, chronic oppositional and aggressive behaviour is the most consistently documented childhood outcome. These children range from the socially withdrawn and avoidant, to those who demonstrate fear, anger, and aggression. These features are linked both to the physical abuse and the family context of pervasive aggression and conflict.[3]

The children's attachments to their caretakers are anxious and insecure. Children view themselves negatively, and show increased rates of both depression and anxiety throughout childhood. Long-term exposure results in a constellation of reactions characterized by pervasive denial by the child, an apparent repression or dissociation of memories, relative indifference to pain or distress, episodes of rage directed towards self or others, and an unremitting sadness. Male victims may develop a characteristic hypervigilance.[21]

The major health consequences of physical abuse in childhood have become clarified.[3] The causal connection between physical abuse and later psychological and physical health problems is underlined through clear links between early age of onset, and severity of maltreatment and subsequent severity of psychological and physical ill effects in teenage and adult years. Further, physical abuse cases embedded within violent families and associated with accompanying neglect have relatively worse outcomes, psychosocially and in physical health. Approximately 20 to 30 per cent of physically abused children develop conduct problems in teenage years, starting earlier and displaying more violence than their non-abused counterparts. They are at increased risk of running away from home, and are overrepresented among young homeless children in inner cities. Childhood physical abuse is associated with subsequent substance abuse problems, self-destructive behaviour and suicidality, depression, teenage pregnancy and poor physical health outcomes. Genetic factors mediate the association between physical abuse and later antisocial behaviour and, probably, affective disorder too.[3]

Child neglect

Definition and clinical features

Neglect refers to the underprovision of the child's basic needs, both physical and psychological. Most cases comprise omissions of care by parents and others in the parental role. However, institutional neglect also occurs, mainly in the form of collective caretaking failure—for example, residential children's homes in the United Kingdom, orphanages and nurseries in Eastern Europe, and neglect of care by educational establishments.[3]

Definition problems include whether neglect should include the apparent impact on the child and/or the degree to which it was intended.[3] There are cultural variations in what might be perceived as neglect. The practice of putting young children into separate bedrooms, while considered normal practice in much of Western Europe, would be considered frankly neglectful in some Eastern cultures.

Notwithstanding these definition problems, four main types can be identified: physical, supervisory, cognitive, and emotional neglect. Neglect can occur first during the prenatal period, for example through maternal substance abuse, and may be observed throughout childhood. **Physical** neglect includes inadequate nutrition, clothing, shelter, but also exclusion and abandonment. This is the most common form of neglect reported to welfare agencies in North America, Australia and Western Europe. **Supervisory** involves inadequate parental overview, relative to the child's needs, for developmental needs of the child, but also employing unsafe alternative carers, and failure to use available health care. **Emotional** neglect includes insufficient parental affection, and inattention to the child's cues, which has been termed 'psychological unavailability'.[24] **Cognitive** is insufficient parental responsiveness, attention and speech, but also denying access to education opportunities.

Diagnosis and presentation

Although most reported cases involve younger children, neglect occurs at all ages. Many cases are followed for years before being

finally identified by professionals. Non-organic failure to thrive can precipitate earlier recognition. Otherwise neighbours, relatives, or school teachers report the child's plight to protection agencies, by which time the effects are severe and neglectful caretaking entrenched. Recognition may also come about through the child's presentation with developmental delay, language problems, school non-attendance, inadequate medical or dental care, or with significant psychological difficulties. Conclusions about neglect need to be linked with the individual's developmental needs. Additionally neglect must be distinguished from the effects of poverty. Conclusions are assisted by using multiple sources of information; from the children themselves, caregivers, reviewing longitudinal case records, direct observation and standardized measures.[3]

Aetiological and background factors

(a) Characteristics of neglectful parents

Neglectful parents are likely to be poor, have multiple difficulties, and display what has been described as the apathy-futility syndrome. Parents show immature personality characteristics, with low self-esteem, impulsivity, and an inability to plan or demonstrate choice in such important areas as adult partners, having children, or employment. Neglectful parents frequently hold inaccurate or unrealistic expectations about their children's development or behaviour. Neglect may derive from parental psychiatric illness such as schizophrenia, depression, or drug or alcohol abuse.

(b) Characteristics of neglected children

Neglected infants have anxious, disorganized attachments with their caretakers. Later in childhood they are more aggressive than comparison children, though less so than physically abused children. Neglected toddlers show non-compliance and become easily frustrated, later developing low self-esteem and self-assertiveness and showing less flexibility or self-control. Both in preschool and school they lack persistence and enthusiasm, and become socially isolated.

(c) Family aspects

Child neglect is normally embedded within broader family insularity, lack of cognitive stimulation, affection or emotional nurturing between its members, and significant household disorganization. Neglectful parents are likely to be unresponsive to both their infants and older children, showing a paucity of prosocial positive behaviours, less interactions and stimulation, and more negative behaviours than controls. Even though there is a strong link with poverty, parents who neglect children stand out among their equally materially impoverished neighbours.

Course and prognosis

The seeds for the neglected child's long-term difficulties with social interaction, relationships, and educational progress can be observed in infancy. Neglected children tend towards passiveness and helplessness under stress. They show significant developmental delays, especially language problems, attention-seeking behaviour, and superficial displays of affection, as well as conduct problems, persistent defiance, and hostile behaviour. Studies of children who as infants were subjected to psychologically unavailable caretaking reveal persisting difficulties with anger, non-compliance, low frustration tolerance, little enthusiasm or persistence for tasks, poor impulse control, relative rigidity, and lack of creativity. Similar negative developmental outcomes have been reported to occur following non-organic failure to thrive in infancy, especially where combined with physical neglect, leading to long-term cognitive delay and poor educational attainment. Children who as infants experienced psychologically unavailable parenting do even worse than physically abused children, showing a greater number of emotional problems, inattention, social withdrawal, and unpopularity with other children.[24]

Psychological maltreatment

Definition and clinical features

Emotional abuse (better termed 'psychological maltreatment') refers to those interactions with children that have the potential to damage the child psychologically, given his or her particular developmental needs.[3] Four broad groupings of acts are described: the need for psychological safety and security; for acceptance and positive regard; for age appropriate autonomy, and sufficient opportunities to explore environment and extra familial relationships. Included within **psychological safety** are exposure to domestic violence, threats of injury, suicide or abandonment, and discipline through intimidation. Within **acceptance and self-esteem** are verbal and non-verbal negativity, active rejection, ridiculing, inappropriate expectations and undermining. **Age appropriate autonomy** includes both inappropriate responsibility giving and prohibiting age appropriate socialization and placing a child in a reversed parental role. **Restriction** includes restrictive confinement and isolation.

Psychological maltreatment may be direct toward the child, or operate indirectly, for example through the child witnessing domestic violence, or observing parental involvement in antisocial activities. It may occur in institutional settings as well as within families. The overlap with neglect is evident from the list of acts of omission and commission listed above.

Diagnosis and recognition

Recognition may occur when other kinds of maltreatment are discovered, or when domestic violence is revealed. It may also occur when a child is noted to be living with, and/or providing care for a parent with mental or physical illness, personality disorder or substance abuse. Sometimes recognition follows a child's referral to developmental or mental health clinic, or through the reported observations of neighbours or professionals (e.g. teachers, police). Diagnosis requires detailed history, with examples, direct observations of parent-child interactions, and interviews with older children. Standardized data gathering schemas may assist diagnosis.

Aetiological and background factors

(a) Characteristics of abused children

Reports of emotional abuse in children become more frequent throughout childhood into adolescence. Reported cases are more likely to be linked with lower socio-economic status. There is no particular link with racial or ethnic groups. Psychological maltreatment is frequently integral to other forms of maltreatment and so distinguishing different aetiological factors and consequences is complex.

(b) Characteristics of abusers

Although not systematically studied, this probably varies according to the mixture of subtypes present, and whether any other kinds of abuse or neglect coexist.

(c) Course and prognosis

Psychological maltreatment in infancy has a very poor outlook (see discussion of neglect). Much less is known about the outcome of different mixtures of psychological maltreatment identified during childhood and adolescence. There are indications that the degree and extent of psychological maltreatment is a better predictor of case outcome than the extent of any coexisting physical or sexual abuse, thus underlining its importance to the developing person's mental health.

Fabricated or induced illness (Munchausen syndrome by proxy)

Definition and key elements

Fabricated or induced illness (FII) is where a parent or carer feigns an impression or induces a state of ill health in a child whom they are looking after. The key elements are parental falsification or deceit, and a triangular interaction between parent, child, and health professional, in which the doctor is misled by the parent, some parental need is met, and the child harmed (directly or indirectly). The harm occurs through: verbal fabrication of symptoms/signs; falsification of reports or specimens; or through inducing ill health (either actively or by withholding essential substances).[25]

Diagnosis

The presentation can be in any bodily system, but common forms are fabricated epilepsy, non-accidental poisoning, apparent life-threatening events in infancy (either directly induced suffocation, or fabricated), or multi-system disorders (e.g. gastrointestinal and renal problems).

The diagnosis of fabrication is almost always undertaken by paediatricians, whose awareness of the possibility of fabricated signs or symptoms is now much greater and leads to an earlier diagnosis than when first described by Meadow.[26]

There are several elements to the phenomenon:

1 the harm caused to the child through fabrication;

2 the impact on the child's development, both physically and emotionally;

3 the psychological status of the fabricator.

Psychological services are especially involved in (2) and (3)—assessing the child's developmental status, and considering the mental state of the fabricator and assessing family dynamics. Differentially, factitious illness by proxy needs to be distinguished from parental overanxiety or exaggeration, or frank malingering, though sometimes FII contains elements of all these.[25]

Epidemiology

The annual incidence among children in the United Kingdom has been calculated to be 0.5 per 100 000, but for those under 1 year it is 2.8 per 100 000.

Aetiological and background factors

(a) Characteristics of abused children

The majority of children are under 5 years of age, with boys and girls equally represented. Affected infants are likely to have feeding problems; withdrawal and hyperactivity are seen in school-age children, whereas adolescents may develop somatization themselves. Up to three-quarters of the children show evidence of other fabrications, or of physical abuse and neglect.

(b) Abuser characteristics

Most fabricators are female—79 per cent of whom have a somatization disorder themselves and half have a personality disorder, particularly so among fabricators who induce illness. Most abusers deny responsibility, at least initially.

(c) Family characteristics

Unusually, families are often intact, though 40 per cent have serious marital problems. Child-parent attachment difficulties are common, and other siblings in the family may be affected. Typically, fathers are not involved in family life.

Course and prognosis

Affected children may be damaged by the abuse itself, while mortality is between 5 and 10 per cent. About 20 per cent are reabused, though not necessarily in the same way as the original FII. Emotional harm, conduct problems, and educational difficulties occur in half the children on follow-up.

Prevention of maltreatment

Reducing the incidence and recurrence of child maltreatment are crucial initiatives, because of the resultant ill effects[27] and difficulty in instituting effective treatments, quite apart from the humanitarian prerogative. Family support—and education concerning parenting, child development, and the management of problems—has a positive impact on parental attitudes and knowledge as well as on observed behaviour. Significant effects on children's behaviour, cognitive outcome, and child maltreatment rates are less clear. Preventive efforts have more impact upon physical abuse than on neglect.[28]

Brief interventions are beneficial for low-risk parents, whereas more intensive approaches are needed for higher risk groups. High-risk groups include deprived, impoverished parents, young mothers, and those with a personal history of childhood abuse themselves. However, for a prevention approach to be effective it must be personalized to the needs of the individual families and include outreach components for the most negative and hard to access parents.[28] Including males, whether resident or occasional visitors, is crucial to maintaining and sustaining improvements in parenting and child care. Equally, effective programmes are more likely to be valued by the parents themselves, underlining the importance of matching the skills of staff and the contents of programmes with the families' specific needs.[29,30] Similarly, programmes must be culturally sensitive if they are to be effective and accepted by parents. Interventions are most effective when they impact upon a broad network of influences and relationships, ranging from those of the immediate family to broader neighbourhood and social influences on children's welfare.[3] Furthermore, primary, secondary and tertiary prevention approaches need to be integrated and carefully planned within each area.[31]

Sexual abuse prevention is probably ineffective when aimed at enhancing children's capacity to protect themselves.[28] On the other hand, programmes which include parents (and increase their capacity to keep their children safe) and incorporate antibullying tactics are much more likely to be effective, although this probably stems from increasing disclosure of early sexually abusive actions rather than primary prevention.[28] Additionally, broader social

initiatives to reduce child sexual abuse within schools and institutions are essential to a comprehensive area strategy.[27,31]

Intervention and psychological treatment

Treatments

Psychological treatments have been developed for different types of maltreatment, notwithstanding contemporary appreciation that co-occurrence of types of maltreatment is common place. Effective treatments for physical and sexual abuse are reasonably well established.

Interventions with empirical support are principally behavioural, and cognitive behavioural ones.[3] These are normally structured and emphasize skill building to overcome emotional distress and behavioural disturbance in children and parents. Trauma-focussed, cognitive behavioural therapies are significantly superior to family and general therapeutic treatments for both physical and sexual abuse. These treatments combine psycho-education, exposure therapy, cognitive procedures and restructuring as well as behavioural management. Children are also assisted with emotional recognition and regulation and attention to maladaptive ideas. Other empirically supported interventions include child parent psychotherapy (blending psychodynamic and cognitive behavioural components).

Psychological treatments in neglect have concentrated on improving parenting skills and sensitivity through direct encouragement of positive interactions in feeding, play, general care, combined with individual therapy for parents themselves.[3] Parallel psychiatric treatment of parental mental health problems, such as depression or substance abuse is also important, although there is debate about whether such treatments should precede or be delivered subsequent to treatments focussed on parenting. There has been some support for therapeutic day treatment programmes and multisystemic therapy for neglect.

Psychiatric interventions for psychological maltreatment have not been subject to empirical evaluation thus far. In the meantime it seems reasonable to focus on improving sensitivity and responsiveness within parent child relationships (through direct work and feedback), together with family based work, and individual work with parents who have been subject to deprived or abusive backgrounds themselves.

Treatments for FII are sparse, but combinations of treatments for neglect and psychological maltreatment, combined with behavioural management of somatization appears promising.[3]

Management

(a) Guiding principles[3,32]

The first priority is to establish the **child's safety** and/or freedom from neglect. This may require separation of child and abuser. Those cases involving neglect or psychological maltreatment may be managed through verifiable agreement, and providing services designed to promote the child's welfare within the family. Interdisciplinary planning and coordination will be essential in order to achieve this.

Interventions must be focused primarily on the **child's welfare**, rather than other objectives such as adult treatment or family preservation. Interventions should also focus on the child's **physical and emotional safety**, and be **developmentally focused**, while simultaneously tackling **parenting problems**. A developmental focus involves adapting treatments to a child's age and developmental status, and taking into account any developmental impairment. Additionally, it is essential that treatment approaches comprehensively address those developmental processes affected by maltreatment, i.e. affect regulation, attachment, the evolving self system, and peer relationships.[5]

Approaches to parenting problems range from interventions with parents individually, those focused specifically on parent child dyads, through to more broadly based social support services, including day attendance at family centres designed to improve parenting care, the use of family support workers, harnessing neighbourhood supports and other parents prepared to assist new parents at risk, and parenting classes. Services to improve family conflict resolution and parental management of hostility and aggression may also be needed for a comprehensive approach to tackling parenting problems. Parents with mental health problems need to be psychiatrically evaluated to see whether treatment could assist overall case management. Sexually abusive behaviour or physical violence perpetrated by adults may be amenable to psychiatric intervention. Persistently dangerous abusers need to be identified and child safety assured.

Further, interventions should specifically address the **child's experience** of maltreatment and any moral or legal dimensions to this. Interventions should not focus solely on maltreatment itself, but equally address any **general mental health issues**. They should also aim to **prevent future difficulties** as well as addressing current problems. Supportive and **nonabusive parents and carers** should be part of the intervention and incorporated into treatment plans. Obtaining **parental acknowledgement and recognition** of their part in maltreatment is an important part of successful interventions. Parental acknowledgement may be achieved through education and discussion, and through treatment approaches designed to alter hostile or neglectful views and attributions held by parents. First choice interventions should be those with the highest level of **empirical support**. Generally, interventions should involve school and **local networks** of child professionals in a systemic fashion. A systemic perspective is helpful with respect to all families. Sensitive working across disciplinary boundaries requires knowledge of **local** family justice system and child protection **working practices**.

(b) Case planning

When deciding what to do in the individual case, treatment may have to address several aspects of abuse and neglect, because co-occurrence of maltreatment type is common. Safety is of paramount consideration. There is little value in starting treatment if the child remains unsafe. This will require multidisciplinary or multiagency co-operation in order to develop a comprehensive plan for child and family. Even where intensive treatments are unavailable, psycho-educational approaches are helpful for all abused children. In reality, most maltreated children receive no systematic therapeutic intervention.

The majority of treatment approaches combine intervention for the child with adult treatment and often family based work. Supportive work with non-abusive carers is of proven value. In planning this, however, it must be remembered that failure to protect a child is harmful and dangerous for the child's future too. In addition, in one family there may be more than one maltreating carer, e.g. abuse by one adult carer and neglect by another.

Effectiveness of intervention is gauged through several dimensions. For example, child safety, improved carer availability and sensitivity for the child, whether the child has overcome effects of trauma, psychological symptom reduction, improved peer relationships, speech and language catch up and educational progress. Improvements in family functioning which are sensitive to interventions include; reduced conflict and violence, improved communication and emotional expression, changes to disturbed attachment patterns and improved child to parent attachment, and greater warmth in parent/child relationships. In addition, effectiveness may be gauged through target mental health objectives for individual carers, e.g. reduction in parental depression or improvements in anger management.

Developmental considerations will also contribute to evaluation of effectiveness. For example, a shorter timescale is appropriate for younger children in view of their developmental needs; and greater than average improvement in parenting may be needed for children with impairments or disabilities.

Risk management is a central issue for mental health practitioners when providing interventions in maltreatment cases, and underlines the need for systemic awareness and well-developed interdisciplinary practice. Risk factors for occurrence are reviewed elsewhere.[3] Recurrence risk has been the focus of systematic review[33], and narrative overview, using an eco-developmental perspective and identifying risk elevating as well as lowering factors.[34] Nonetheless, multiplicity of relevant factors as well as complexity of transactions in individual cases render actuarial approaches to risk management an unrealistic aspiration at this point. In its place, an approach that has been characterized as structured professional judgment appears the most appropriate.[35] Key risk elevating factors for recurrence include: domestic violence, child neglect, cases where there had been previous maltreatment, and parents with mental health disorders. A structured approach to risk management entails an approach to data gathering, diagnostic formulation and subsequent decision making that rests explicitly on available evidence about risk factors for recurrence.[34]

(c) Ethical and legal considerations

Decisions as to whether to share sensitive information with other professionals will be guided by child welfare and safety considerations, which are paramount, and override adult consent withheld. Sometimes children request confidentiality, in situations where their safety is potentially compromized. Normally, explaining to children why information must be shared, and involving them enables their trust to be maintained even where there is initial disagreement.

Generally, patient information will need to be shared during multidisciplinary planning meetings, but agreement can usually be made to respect confidentiality, except to the degree necessary to assure child safety. Many cases will involve family justice systems, which normally require overview of progress. Practitioners will need to provide reports for planning meetings and family courts, in order to contribute to safe care.

(d) Long term

Mental health effects may present at different points in the life course, particularly at key times of developmental change, e.g. at a first romantic relationship, or when becoming a parent. If possible, such possible future difficulties should be noted in a person's medical record, to facilitate future intervention. As part of an area's comprehensive prevention approach, new parents with a history of childhood maltreatment should be offered extra support, as they are a vulnerable group for parenting problems (although it is important to stress that discontinuity of parenting problems is more common than continuity).

Further information

Web sites

California Evidence-based Clearinghouse for Child Welfare. http://www.cachildwelfareclearinghouse.org/

Every Child Matters. http://www.everychildmatters.gov.uk/

International Society for the Prevention of Child Abuse and Neglect. http://www.ispcan.org/

National Society for the Prevention of Cruelty to Children. http://www.nspcc.org.uk/Inform/informhub_Wda4993.html

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9.3.4 The relationship between physical and mental health in children and adolescents

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Introduction

The link between physical and psychological disorder in children and adolescents is well established. Children with chronic illness are at increased risk of emotional and behavioural disorders. In addition, repeated presentations with physical symptoms may represent underlying psychological distress or psychiatric disorder.

Because of the inextricable links between young people and the family in which they live, it is inappropriate to consider symptoms in an index child in isolation. The effects of symptomatology on family functioning, parent, and sibling relationships should be considered. This may have important aetiological and prognostic significance.

Associations between physical and psychological symptoms

There are various ways in which physical and psychological disorders are related; these are summarized in Table 9.3.4.1.

In this chapter we shall consider the following:

- The psychiatric consequences of physical illness
- Helping the dying child and his or her family
- The effects of psychiatric disorder on the course and outcome of physical illness
- Aspects of assessment and treatment intervention

Table 9.3.4.1 Associations between physical and psychological symptoms

Nature of association	Examples
Psychiatric consequences of physical illness and treatment	*Organic*: acute confusional state, psychosis induced by brain disorder *Functional*: adjustment disorder after diagnosis of diabetes, specific needle phobia in young child with cancer receiving chemotherapy
Effects of psychiatric disorder on physical illness	Depression delaying the mobilization of a child following partial limb amputation after severe meningococcal disease, oppositional-defiant disorder affecting treatment adherence in diabetes
Physical complications of psychiatric problems e.g. deliberate self-harm, substance abuse	Liver failure following paracetamol overdose
Psychiatric disorders or psychological distress presenting with physical symptoms	Aches and pains in school age children, reduced physical well-being in adolescent depression, somatoform pain disorder, dissociative disorder

- Somatization and somatoform disorders, with a particular focus on recurrent abdominal pain, dissociative/conversion disorder, and chronic fatigue syndrome

Psychiatric aspects of chronic physical illness

Chronic physical illness and the risk of psychiatric disorder

Chronic physical illness in children, defined as disorders that last at least 1 year and are associated with persistent or recurrent handicap, affects about 4 per cent of children in Western countries.[1] This encompasses a broad spectrum of disorders including more common problems such as eczema, asthma, diabetes, epilepsy, and less prevalent conditions such as cystic fibrosis and cancer. Many children successfully adapt to living with a chronic illness, but it can be associated with a number of different types of stresses for children and their families.

The stress of chronic illness may operate at several levels. In addition to the presence of the illness itself, diagnostic and treatment procedures may be painful or have undesirable side-effects—changes in physical appearance such as alopecia, scars, and obesity may lead to difficulties in peer relationships. The demands of treatment such as dietary restrictions in diabetes may be difficult. The illness, together with hospital attendance for treatment, may lead to a considerable interruption to schooling as well as a reduced ability to participate in leisure activities and socialize with peers.

Although the majority of children and families successfully adapt to these stresses, children with chronic physical illness have a slightly increased risk for the development of associated psychiatric disorders. Specific factors related to the child and the illness have been shown to contribute to the likelihood of developing psychiatric disturbance and to influence the nature of the psychiatric disorder that develops (Table 9.3.4.2).[2]

(a) Nature of the physical disorder

Much of the increased prevalence of psychiatric disorder in children with chronic physical illness is accounted for by those with disorders affecting the brain, especially when epilepsy is involved.[3] They have a three-fold increased risk of psychiatric disorder over general population rates. The risk in young people with a chronic physical illness that does not involve the brain is considerably lower and only slightly increased over general population expectations.[3] The excess of psychopathology in children with brain anomalies may be attributable to the direct effects of organic pathology on behaviour, or may be mediated by the greater physical disability that frequently accompanies brain damage. Associated intellectual impairment may also be an important contributory factor.

Table 9.3.4.2 Factors related to the risk of psychiatric disorder and the form of its presentation

Nature of physical disorder (whether brain involvement)
Stage of illness (whether acute stresses involved)
Severity of illness
Degree of life threat
Psychosocial risk and protective factors in family
Age (developmental stage)
Effects of illness and treatment procedures

Whilst this dichotomy between disorders involving and not involving the brain is useful, there is little specificity in the behavioural pattern that may be attributable to intracerebral pathology. As a possible exception, children with brain dysfunction such as epilepsy or cerebral palsy may be more likely to exhibit externalizing disorders such as hyperactivity.[3] Psychiatric disorders in this group of children may be persistent, with 70 per cent still experiencing difficulties at 4-year follow-up. Overactivity, restlessness, and inattention are the best predictors of persistence.

For conditions not affecting the brain, the development of psychiatric disorder seems most likely to be linked with the accumulation of generic stress factors and family changes common to living with a chronic illness. These include life stresses such as hospitalization and daily difficulties such as specific dietary requirements and disruption of family routines.[4] A broad spectrum of psychiatric presentations are associated and these are not specific to the nature of the underlying disease processes. Children with non-neurological physical illnesses are more prone to developing emotional symptoms and eating anomalies as opposed to antisocial behaviour. Eating anomalies may arise from an emphasis on diet and a concern about poor appetite in the families of many children with chronic illnesses. Maternal anxiety may focus on feeding, especially in preschool children. The specificity of the relationship with emotional disorders is of interest. Physical illness in the child can generate family and social stresses and changes that are known risk factors for the development of emotional disorders in children. This includes mood disorders in parents and overinvolved and overprotective parenting.[2]

(b) Stage of the illness

Disorder at the time of initial diagnosis is not uncommon and is frequently short lived. In one study, 36 per cent of 8- to 13-year-olds with newly diagnosed insulin-dependent diabetes mellitus developed an adjustment disorder (most commonly dominated by depressive symptoms) within the first 3 months of diagnosis; 50 per cent had recovered within 2 months.[5] Similarly, in patients with chronic renal failure, psychological problems were reported in 60 per cent of children at the time of starting dialysis. One year later, after stabilization of their physical condition, the prevalence of disturbance was reduced to 21 per cent.[6] It is very likely therefore that in many children with chronic physical illness, psychiatric disorders are most frequently transitory adjustment disorders to stressful times in the illness.

(c) Severity of illness/degree of life threat

More severe physical disorders and those constituting a greater degree of life threat are associated with a higher risk of psychiatric disturbance. In children with end-stage chronic renal failure, those with more severe disorders (on hospital haemodialysis) have been found to have more psychiatric disorder than those not yet requiring dialysis.[7] More severely affected diabetic children and adolescents with a history of hospitalization for ketoacidosis in the previous year are more likely to exhibit psychiatric disorder than a control group of outpatients also with insulin-dependent diabetes mellitus.[8] Post-traumatic stress disorder (which by definition requires acknowledgement of perceived life threat), and high levels of post-traumatic stress symptoms have been found in children and parents up to a year after admission to Paediatric Intensive Care Units[9]; (a much higher proportion than following admission to general paediatric wards), and up to 10 years after treatment for childhood cancer.[10]

The link between illness severity and risk of psychosocial impairment may vary with the setting in which it is examined. Less severe physical impairment has been shown to be associated with a higher risk of behavioural problems in the school setting.[7] Teachers may be less aware of the presence of an underlying physical disorder in this group who have less visible physical signs, and may make less allowance for these children than for those with a more overt disorder.

(d) Psychosocial risk and intrafamilial protective factors

When a physically ill child develops psychological symptoms, these are frequently attributed by families and professionals to the presence of the illness and its stresses. It is important not to neglect consideration of other predisposing factors (i) within the child, for example genetic vulnerability, temperamental characteristics, (ii) in the family such as marital disharmony, lack of open communication, maternal mental illness affecting parenting, and (iii) within the broader social environment such as bullying at school and poor peer relationships. These factors contribute to child psychopathology in ill as well as in healthy children. Conversely, protective factors such as secure parent–child attachments, increased family social support in response to the physical diagnosis, as well as sensitive paediatric management of hospitalizations and stressful medical procedures may reduce the risk of developing psychiatric disorder.

(e) Age (developmental stage)

Manifestations of psychological distress in ill children vary with each developmental stage. Preschool children have fewer cognitive resources to cope with discomfort and stressful medical procedures and are likely to rely on maternal support and distraction to cope with illness. Between 4 and 7 years of age, children may believe that illness has been caused by something bad they have done and that they should be punished.[4] Clinginess to parents, fearfulness, sleep difficulty, and oppositional–defiant behaviour are seen in preschool children. The need for repeated painful procedures, for example with cancer chemotherapy, can lead to the development of specific needle phobia.

For school-age children, school life is a key aspect of their adjustment to illness. Return to school after cancer chemotherapy can be associated with the development of school phobia, loneliness, and social isolation. School absence and having to catch up with school work, teasing, or even bullying, especially of children who look different, may also occur and contribute to lowered self-esteem and the risk of affective disturbance. Cognitive development in adolescence allows a greater understanding about the implications of chronic illness and the realities of death; depression occurs more frequently in this age group. Adolescents may begin to challenge and experiment with their treatment; they may fail to come to outpatient appointments or attend erratically. There may also be a decline in compliance with medical advice and adherence to treatment regimens.[4] For example, diabetics may not follow dietary advice or pay reduced attention to their insulin regimen and monitoring of blood sugars leading to poorer diabetic control. Adherence may be influenced by family factors; poorer metabolic control is associated with less family cohesion and a parenting style that is perceived as critical and negative.[11] Adolescents aged 13–18 years with diabetes and co-morbid internalizing disorders, and discharged from hospital, have been found to be at greater risk of readmission up to 2 years later. This relationship was not found for younger children, suggesting that greater parental control of diabetes management (as is usual for younger children) may ameliorate the potential for psychiatric disorder to affect treatment adherence.[12]

The way in which psychiatric disorder presents may influence its perceived significance to health professionals and the likelihood of psychiatric referral. Presentations with behavioural disturbances such as screaming, struggling, panicking, or a failure to comply with treatment are more likely to precipitate referral than internalizing disorders such as depression.

Effects on parents and siblings

Whilst most families successfully adjust to the presence of a child with chronic illness in the family, this may act as a risk factor for psychological disorder. The incidence of marital break-up is not increased, but there are reports of increased marital distress. Interparental conflict may not be directly expressed but instead diverted to excessive worry and focus on the illness, which can be very stressful for the child.[13] In parallel with the heightened short-term psychological difficulties found in ill children immediately following diagnosis, a similar temporal pattern of disorder has been reported for parents and siblings. Most research has focused on mothers, who often undertake the practicalities of caring for a sick child. They may need to stop work themselves, leading to increased social isolation and a reduction in extra-familial support.[14] Fathers and mothers often cope differently with the diagnosis; mothers tend to react by emotional release, whereas fathers are more likely to withdraw and concentrate on practicalities.[14] Higher rates of maternal psychiatric treatment and negative affect have been found in families with a chronically ill child. The risk of maternal depression is greater for mothers of children with chronic as compared with newly diagnosed epilepsy; the burden of illness may impede parenting capacity and contribute to the development or maintenance of psychopathology in the children.[13] Siblings may resent both the extra attention an ill brother or sister is receiving, and repeated separations from parents during periods of hospitalization. Their psychological adjustment is related to the degree of functional impairment[15] and recent physical health of their ill sibling, the extent to which family life is disrupted by the illness, and the psychosocial support available. The need for improved communication with healthy siblings has been identified.

One disorder which highlights the complexities of interaction between living with a chronic illness and its effect on family members is AIDS. Vertical transmission from an infected mother to her unborn child has decreased in the last 10 years but there has been an increase in the number of adolescents with the virus due to survival of children with perinatally acquired HIV into adolescence in addition to adolescents acquiring the virus through other means. For many children with HIV, infection is also present in other family members, often the mother. Families have to cope with the disease itself and its treatment, the stresses of chronic illness which include an uncertain prognosis and the possibility of death as well as having to negotiate the stigma and social isolation that frequently accompany the diagnosis. Caregivers who are HIV positive themselves report poorer physical and emotional health compared with non-infected caregivers; this is associated with greater psychosocial impairment in the children—a higher risk of internalizing

problems such as anxiety and depression, more externalizing problems e.g. oppositional behaviour and poorer academic functioning. Disclosure of the diagnosis to affected children is often avoided; reasons include parental unease discussing their own HIV infection, fear of stigma, beliefs that the child is not emotionally ready to cope with the information and parents' own distress.[16] Children (aged 6–16) who are not told their diagnosis have been reported as having more internalizing problems than those informed.

Management

In the absence of rigorous treatment research in this area, the most important tenet of the psychological care of children with physical illness is based on good clinical practice, with clear and consistent communication between paediatricians, child psychiatrists and their multi-disciplinary teams. This allows early detection and intervention for psychological disorder.

Child psychiatrists frequently work closely with paediatric colleagues to assist in identifying young people at risk for psychiatric disorder, to provide assessment and treatment when indicated, and to give support and advice with regard to diagnosis and management. Many paediatric units have regular weekly psychosocial ward rounds where professionals both from within the hospital and from the community—representing paediatrics, child and adolescent psychiatry teams, social work, and education—can meet to discuss the progress of the child from each perspective.

A full psychiatric assessment involving the child and the family will be carried out in referred cases. This needs to be preceded by a careful explanation to families about the reasons why a psychiatric consultation has been sought.

Important information about premorbid concerns and the child's level of functioning may be obtained from schools, social workers, and other professionals involved with the family.

Specific psychiatric diagnoses should be treated appropriately. Children may develop acute confusional disorders associated with intracerebral infection or febrile illness. Manipulation of the ward environment to ensure: clear differentiation between night and day, that familiar toys are nearby, close family members are in attendance, and developmentally appropriate explanations are given to the child about where they are and what is happening, may help considerably. If behaviour is too difficult for staff to safely manage and is interfering with treatment, sedative medication may be needed and should be discussed with paediatricians.

Children with adjustment disorders may be helped by psychological interventions. Management may include ways of decreasing existing stresses or helping individuals to adjust to them. Possible interventions include supportive counselling, individual therapy using cognitive behavioural principles, and family therapy.

When there is a chronically ill child in the family, parents often find it difficult to maintain the usual boundaries. For example, disciplining an ill child may be associated with parental guilt; this can lead to increasing anxiety for children who exhibit increasingly oppositional behaviour in an effort to test the boundary limits. Discussion regarding parenting techniques in the context of these feelings may be helpful. Parents also tend to increase their protective responses to ill children and show more overinvolved parenting. If excessive it may impede the child's development, but to a modest degree it may be helpful and advantageous.

Systematic desensitization together with relaxation and distraction techniques may be used to treat a specific needle phobia. This needs to be carried out in collaboration with ward staff taking account of associated psychopathology, for example, oppositional behaviour, a generalized anxiety state, or an adjustment reaction. Treatment of the associated problems can often obviate the need for direct phobic treatment. When indicated, the latter's success is likely to be dependent on external changes that reduce anticipatory anxiety. These might include minimizing the time the child needs to wait for treatment and ensuring that more experienced members of the medical team are responsible for cannula insertion.

Generalized symptoms of anxiety are not uncommon in parents and children and may be manifested in different ways, for example, a young child may resume bed-wetting, a school-age child may become intensely distressed by being away from his parents, adolescents may experience difficulties sleeping, and anxious parents may become agitated with ward staff. Regular explanations from staff about the child's condition and treatment may help to alleviate this anxiety. Communication difficulties within the family may contribute to anxiety and be helped by family meetings where difficulties can be shared. Relaxation and distraction techniques together with cognitive behavioural interventions may also be of benefit. If symptoms are intense and interfering with physical treatment, anxiolytic medication may be indicated.

Antidepressant medication may be considered for children and adolescents with a depressive episode. This should be discussed with the medical team to minimize drug interactions and side-effects that may exacerbate the physical condition of the patient.

Treating children with severe illness who may be receiving distressing and painful treatment can arouse intense emotions in the most experienced of paediatric staff. Regular meetings with mental health professionals may help them to process some of these feelings and prevent them impeding patient care.

Prognosis of psychiatric disorder in children with chronic physical illness

Many of the psychological difficulties experienced by chronically ill children are short lived and do not continue into adult life. Overall, studies indicate that psychiatric outcome is not severely compromised in the majority of adult survivors. Persistence of disorder is related to the severity of childhood psychological symptoms (the more severe being more likely to last), persistence of physical symptoms into adulthood,[17] and to the presence of physical disorder affecting the brain (Table 9.3.4.3).

The form of psychiatric symptomatology in childhood and adulthood may also be different; for example, cystic fibrosis sufferers, aged 8–15, have been found to report more eating related symptoms whereas symptoms of anxiety and depression are more prevalent in the adult group. With regard to cystic fibrosis, a consistent association between disease severity in adulthood and psychiatric disorder has not been shown. However, increased disease severity in childhood is associated with lower educational attainment;

Table 9.3.4.3 Factors associated with persistence of psychosocial dysfunction into adulthood

Severity of childhood symptoms
Persistence of *physical* symptoms into adulthood
Physical disorder involving the brain

in adulthood, employment is associated with both higher academic achievement and less depressive symptoms.[18]

Many studies suggest that by adulthood, most survivors of childhood cancer are indistinguishable from the general population with regard to psychosocial outcome. However, more detailed analysis suggests that factors such as age at diagnosis, site of the tumour, and nature of treatment (e.g. cranial irradiation) may influence cognitive and psychological outcome. For children and adolescents up to age 18 diagnosed with brain tumours, cognitive deficits and psychosocial problems increased with age and time since diagnosis.[19] As survival has increased, adults are exposed to the chronic toxic effects of treatment such as endocrine abnormalities, cardiac or pulmonary problems, and infertility. Follow-up of childhood cancer survivors, to a mean age of 28, revealed that current physical functioning, including pain, was associated with suicidality even after accounting for treatment and depression variables. Younger age at diagnosis, longer time since diagnosis, and cranial irradiation were also important risk factors.[20] Survivors of acute lymphoblastic leukaemia and Wilms' tumour did not show increased psychopathology as adults but had more difficulties with interpersonal functioning and day-to-day coping.[21]

Individuals with intracerebral pathology maintain high levels of disorder in adulthood, especially with regard to behaviour and social isolation. By contrast, patients with congenital heart disease surgically corrected in childhood, are not at increased risk of psychiatric disorder as adults.

Although young adults with end-stage chronic renal failure report more episodes of psychiatric disturbance than healthy matched controls before 17 years, they do not necessarily have increased psychopathology in late adolescence and adulthood. In common with survivors of other chronic childhood disorders,[17] the majority of adult renal patients are reported as functioning well socially, but they are more likely than age-matched controls to be living with their parents, to have less school qualifications, higher rates of unemployment, and fewer intimate relationships outside the family.

Care of the dying child

Children at different developmental stages differ in their understanding of death. They gradually acquire components of the death concept; between 9 and 11 years of age, most children have reached a full understanding, acknowledging that it is permanent, inevitable, and universal. However, experience of serious illness and death interacts with the stage of understanding, so that children aged 5 or younger may have a more mature understanding and exhibit symptoms of anxiety about death. There is evidence that even young children with terminal illness are aware that they are dying, although they may not tell anyone that they know.[22]

Parents (and professionals) often find it difficult to talk about death with children. This is likely to interfere with coping for the whole family. Families with an open pattern of communication do better psychologically.[22] Mental health professionals may have a role in facilitating this discourse, promoting parents' confidence, and competence in communicating with their children. This will help the whole family to begin the process of mourning.

Children need information, reassurance, an opportunity to express their feelings, and adults with whom they can do so. As children lack the vocabulary of adults they may often exhibit their distress by behavioural changes, for example, bed-wetting, difficulty sleeping, and school refusal. Children and their siblings faced with death need clear, simple, and truthful explanations. They should not be pushed to talk, nor frightened with excessive medical detail.

Dying and grieving lead to a whole range of distressing feelings. This is part of a normal process, and mental health professionals can help their colleagues and families to acknowledge that this upset is acceptable.

Bereaved children frequently model their grief experience on what they perceive as being acceptable in the family, and an overt denial of upset by parents may lead to psychological difficulties in the child. The issue of whether to involve siblings after the death of the child in funerals or graveside visits often arises. If children are prepared for what to expect, involvement can be helpful in enabling them to acknowledge that a change has taken place and other people are feeling as sad as they are.[22]

Mothers are involved in nursing and caring for their dying children. They report an excess of depression, problems of helplessness, and a fear of being unable to cope with the child dying. Parents may feel that they can never fully recover from the loss of a child. Fathers tend to report more difficulties with feeling left out of the ill child's life and then with worry about their spouse being too preoccupied with the dead child. The effects of a child's death on family life can be traced even years after the death.[22] Formal follow-up after bereavement may help to identify those families and individuals experiencing psychological reactions that may benefit from more intensive support.

The effects of psychiatric disorder on the course and outcome of physical illness

Psychological disorder, as well as being a consequence of both acute and chronic physical ill-health, may also have an impact on the course of physical illness.

An increasingly recognized disorder in this respect is post-traumatic stress disorder. Sudden physical trauma, such as burns and road traffic accidents, are examples of antecedents. Victims of road traffic accidents between 5 and 18 years of age, particularly those who experience high levels of distress immediately after the accident, are at greatest risk of exhibiting post-traumatic stress symptoms 3 months later.[23] Such responses may be contributed to not only by the accident itself but also by the medical procedures that take place on arrival in hospital. Surgical collars, intravenous infusions, and monitoring equipment can be associated with intense fear.[23] Children with acute severe sepsis such as meningococcal disease admitted to paediatric intensive care units are also at risk of developing similar symptoms.[24] In turn, these reactions can have an effect on the child's ability to co-operate with future hospital attendance, medical, and surgical interventions. Stress reactions may be ameliorated, to some extent, by the provision of age-appropriate information about what has happened and what is going to happen.

The diagnosis and treatment of such disorders may be impeded by the fact that follow-up for young people may not be at the admitting hospital. Burns units and paediatric intensive care facilities are often at tertiary centres some distance from the patient's home. General practitioners and local paediatricians have a role to play in assessing how the family is coping, specifically regarding

symptoms of post-traumatic stress. Child psychiatry involvement may be appropriate if psychological treatment is required both during admission and at follow-up. Cognitive behavioural interventions with individual children and families may be used to alleviate symptoms.

Affective symptoms, particularly depression and anxiety, are not uncommon following an acute medical admission and may interfere with physical treatment. For example, an adolescent admitted to paediatric intensive care with meningococcal disease requiring a partial limb amputation could develop a depressive disorder. Symptoms of despair and hopelessness coupled with a lack of interest and energy may impede the physiotherapy programme, delay mobilization, and hospital discharge. Paediatric staff need to be alert to such potential sequelae and to have child psychiatric colleagues readily available for assessment and treatment.

Exacerbation of chronic illnesses such as asthma can be precipitated by emotional disturbance; adolescents aged 11–17 with anxiety or depressive disorders reported more asthma symptoms in the previous 2 weeks than young people without these affective diagnoses.[25] Adjunctive psychological treatments such as family therapy have been shown to lead to an objective improvement in airways disease,[26] compliance, and reduced hospital admissions.[27]

Somatization and somatoform disorders

Disorders presenting with functional physical symptoms and somatization

'Functional' somatic symptoms with no obvious organic explanation are frequent in childhood. Children have a limited vocabulary for expressing their emotions and often communicate their distress by means of physical symptoms. Somatization refers to this process. In some cases these symptoms become persistent with associated functional impairment; this may lead to consultation. The definitions of somatization disorder (one of the somatoform disorders) used in ICD-10 and DSM-IV are too stringent for children (in that diagnosis requires multiple physical symptoms over years). Other disorders (namely somatoform pain disorder, dissociative/conversion disorder, and neurasthenia) are seen in children and adolescents. The risk factors for somatization in this population are shown in Table 9.3.4.4.

Table 9.3.4.4 Risk factors for somatization in children and adolescents

Individual:	Personal experience of physical illness Enhanced focus on physical sensations Somatic attributions Conscientious, vulnerable, sensitive, anxious personalities with particular concerns about peer relationships High achievement orientation
Family:	Physical health problems Psychiatric problems Parental somatization Emotional overinvolvement Limitations in the ability to communicate about emotional issues
Environment:	Life stresses e.g. school, teasing or bullying, academic pressure

Aches and pains and somatoform pain disorder

Aches and pains (often abdominal pains and headaches) are a common manifestation in young children. Between 2 and 10 per cent of children in the general population have problems in this area. Mothers assess the child's symptomatology with specific regard to whether the child is 'pretending', 'upset', or 'ill' and generally respond appropriately. They recognize that children may experience symptoms as a result of stress or use them to avoid something they find difficult.

Abdominal pain commonly leads to a general practitioner consultation and may account for 10 per cent of new appointments with paediatricians. In only a few of these cases is serious organic pathology found. Lack of identifiable organic pathology does not imply a psychogenic aetiology. The latter is rather supported by evidence that psychological events influence the symptoms.

Children who somatize tend to have a family history of physical ill-health and parental illness. In some cases there are also psychosocial difficulties in the family. There is an association with stressful life events. Co-morbid internalizing disorders (depression and anxiety) are commonly present.[28]

In adolescence, headaches become a prominent symptom, peaking in prevalence at 12 years of age. As with abdominal pain in younger children, they are frequently preceded by physical or psychological precipitants, such as academic or social stresses in school or difficulties at home. Headaches lead to absence from school but are not associated with underachievement. A family history of migraine is common.

As defined in ICD-10, in persistent somatoform pain disorder, severe distressing pain occurs in association with emotional conflict or psychosocial problems that are sufficient to allow the conclusion to be drawn that they are the main causative influences. The result is usually a marked increase in support and attention, either personal or medical.

In trying to best manage severely affected children, close collaboration between paediatricians and child psychiatrists is helpful. The lack of demonstrable organic pathology should be communicated and professionals should help the family to make the link between physical symptoms and psychological precipitants with the help of a written diary if necessary. It is important to reduce the attention given to the physical symptoms in order to decrease the resulting functional handicap. Early return to school together with the resumption of normal activities should be encouraged. The short-term prognosis for presentations to medical services is good, with 75 per cent of children recovering within several months.

Dissociative/conversion disorder

Children and adolescents present as if having a physical disorder affecting voluntary motor or sensory functioning, although none can be found; the symptoms correspond to the patient's idea of physical disorder, which may not coincide with physiological or anatomical principles. Aetiologically, the disorder is believed to arise largely unconsciously and to represent an escape from an unbearable personal conflict. It usually manifests in adolescence and is more common in girls. The most common presentation is neurological with disturbance of motor function such as weakness of legs, paralysis of a limb, or bizarre gait. Multiple symptoms often occur.

Premorbid psychopathology in the child and family are often absent, although perfectionistic and conscientious traits with concerns about academic performance and a child and family focus on

high achievement have been noted. Overconcern with physical health and illness often characterize these families; frequently there is a family history of physical health problems. Families often present as being close, but communication, particularly regarding emotions, may be limited.

It is assumed children develop conversion disorders as an unconscious means of escaping a situation with which they cannot cope. This includes intolerably high academic expectations (often the child's own), unresolved family conflict, and, in a minority of cases, sexual abuse. The disorder is often precipitated by minor physical illness and may also occur in children with identified organic pathology, for instance the development of pseudoseizures in an individual with epilepsy.

The majority of these patients are managed by paediatricians. After investigations have excluded organic pathology and a psychogenic contribution is suspected, this needs to be communicated to the family. The shift from physical to psychological factors may be difficult for the family to accept; information may need to be conveyed slowly 'at a pace the family can cope with'. A collaborative approach between paediatricians and child psychiatrists is important. A persistent focus on physical aetiology may be unhelpful but in management it is more useful to focus on the handicap caused by specific symptoms rather than on their cause by introducing a programme of rehabilitation and physiotherapy directed at these features, including school attendance. Psychotherapeutic work, both individually and with the family, may help the family understand the factors maintaining the child's symptoms and explore any identified stressors or conflicts. Time can also be spent helping families to consider alternative strategies they may use to cope with future conflicts.

Chronic fatigue syndrome (neurasthenia in ICD-10)

This is operationally defined as disabling physical fatigue of over 6 months' duration, unexplained by primary physical or psychiatric causes. There are often other unexplained somatic symptoms and a strong belief by the patient and their family that the aetiology is physical.[29] It might be considered as one of the somatoform disorders, as similarities are shared with regard to aetiology and management.

There is no firm evidence that chronic fatigue syndrome results from a specific viral infection but a physical illness is often the precipitating factor. There is frequently a family history of physical illness and a preoccupation with physical symptoms. Parents invariably attribute the symptoms to an organic aetiology. Children tend to be described as high achieving and perfectionistic, as well as sensitive, vulnerable and anxiety-prone; onset can be temporally related to transitions at school, for example transfer to secondary school. Depressive mood changes are common and on assessment depressive disorder is found in one-third of cases.[29]

Chronic fatigue syndrome can be extremely disabling. A self-perpetuating cycle is set up whereby fatigue and the resultant inactivity lead to loss of muscle bulk and deterioration in physical fitness. Activity becomes increasingly difficult and is avoided, leading to a further deterioration in physical ability. An essential focus of treatment is to disrupt this cycle.

Management is often multi-disciplinary including paediatricians, physiotherapists, school teachers, and child psychiatrists. A clear explanation of the results of physical investigations and the fact that no serious organic pathology has been found is important. Focusing on improving symptoms, as opposed to debating aetiology, is most helpful. Helping the family to shift from a purely physical model to one that includes psychological factors in maintaining symptoms may be difficult and needs to be negotiated slowly. In particular, enabling them to see the disorder as an interaction between physical, social, and emotional factors can be useful.

Treatment ingredients usually include a graded exercise programme, a progressive return to school, and work with the family to facilitate engagement and address factors that may be impeding recovery. Antidepressants may be useful for co-morbid depressive disorder. There is an evidence base for both cognitive behaviour therapy (CBT) and graded exercise in adults and one study which demonstrates the effectiveness of CBT in adolescents.[30]

Treatment of functional symptoms and somatoform disorders in children and adolescents: research evidence

There are few satisfactory controlled studies on the effects of treatment for childhood somatization. Most work on children with somatoform disorders has been based on small groups with severe problems, and the management advice outlined above is derived from the conclusions of experienced clinicians and open-treatment case reports. However, there is some evidence from controlled studies indicating the efficacy of a cognitive behavioural family intervention for recurrent abdominal pains in children.[31] For hospitalized children with severe, recurrent abdominal pain, parental attribution of symptoms to psychological factors facilitated resolution.[32] The superiority of relaxation training over placebo in reducing migraine attacks has been shown.

Outcome of functional somatic symptoms and somatoform disorders

Of adults with a childhood history of abdominal pains, 50 per cent have recurrent symptoms in adult life despite a pain-free period in adolescence; they also have an increased prevalence of psychiatric disorders (particularly anxiety disorders).[33] Childhood conversion disorder is generally associated with a good outcome; recovery is usually complete by 3 months. When children (9–16 years) were followed up 4 years after a conversion disorder from which 85 per cent had recovered, 35 per cent had a mood or anxiety disorder; affective disorder was higher (100 per cent) in the minority who had failed to recover as compared with 23 per cent in those who were better.[34] In young people with chronic fatigue syndrome, recovery or marked improvement in symptoms can be expected in 50–75 per cent of cases in the short to medium term. School non-attendance may be over a year and time to full recovery 3 years or more. There are indications of an increased likelihood for the development of psychiatric disorder after recovery.[35]

Concluding remarks

Somatic and psychological symptoms are intimately linked. Changes in physical health can affect psychiatric outcome. Conversely, emotional distress may affect adherence to treatment, and it is sometimes expressed through physical symptoms. Awareness of this interplay is important and should be mirrored by a close working relationship and close communication between paediatricians and child psychiatrists.

Further information

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9.3.5 The effects on child and adult mental health of adoption and foster care

June Thoburn

Introduction: mapping the terrain

Adoption and foster care are important 'solutions' to identified problems or risks, but potentially they are also contributors to problem behaviours or emotional difficulties. In their problem-solving role, they are seen as potential solutions, not only to actual

or future mental health problems of children, but also to the adverse effects of involuntary childlessness.

This chapter concentrates on the impact of adoption and foster care on the children placed, but their role in problem solution or problem generation for adults is also touched on. Adoption is more often than not a satisfactory way of meeting the need to become parents for those childless couples who succeed in having a child placed with them (a tiny minority of the involuntary childless). It is very rarely a solution to the problems of a parent who gives up a child for adoption whether voluntarily or involuntarily. Studies of adults who relinquished children indicate that the reaction to the loss of their child may be associated with moderate distress or may lead to a long-term grief reaction, which in turn will potentially harm children subsequently born to that parent. One must also note that some parents who lose a child to adoption or foster care are themselves children, sometimes not yet in their teens, whose needs are often overlooked in the interests of providing for the infant.

Fostering and adoption started as very similar processes, diverged in Europe and North America in the first half of this century, and are now much closer together again. The 'total severance' model of legal adoption—the type that most people in Europe, the United States, and Australasia immediately recognize—has a short history. In the United Kingdom it was not until the passing of the 1958 Adoption Act that secrecy became the norm. The 'sealing' of birth information started in the United States around 1948 but it was not until 1991 that Alabama 'sealed' its adoption records.[1] This experiment of totally closed adoption was short-lived, and many countries have introduced legislation to allow adult adoptees and/or birth relatives to access identifying information that allows them to seek each other out.[2] 'Open' adoptions, in which some degree of contact between the adopters, the birth parents, and the children is maintained after placement, are increasingly common.

As countries have become richer, the need to place children for adoption has diminished and the number of infants placed at the request of their parents has fallen well short of the 'demand' of those wishing to start a family through adoption. In consequence, it has been possible to encourage potential adopters to 'stretch' their notions of parenthood, and to place older children, those with disabilities, and those with behavioural or emotional problems with adoptive parents as well as with foster parents. The main remaining difference between adoption and foster care is that the majority of children placed in foster homes live there for comparatively short periods before returning to their families of origin. They are best seen as supplementary rather than substitute parents, although in all 'first world' countries long-term or 'permanent' foster care is an important option for a minority of those entering public care, especially in those countries (the majority) who rarely use adoption as a route out of care.

Adoption and foster care will impact on the mental health of the children in different ways, which may be considered along six main dimensions (see Box 9.3.5.1). The dimensions interact differently for different children. An infant placed from an Asian country might be adopted by childless relatives in Europe and have had a positive early experience of parenting, or might have experienced very adverse early nurturing and be adopted by strangers of a different ethnic origin. The child placed at six may have had good care from one or both parents until some traumatic event led to the need for an adoptive placement, or the child may have been seriously maltreated and had several placements before finally joining a substitute family.

The nature of the evidence on the impact of adoption and fostering on mental health

The actual or potential problems most obviously associated with child placement are those resulting from separation and loss. Brodzinsky *et al.*[3] have made significant contributions to our understanding of the psychology of adoption. Put simply:

> . . . for later-placed children, the loss of family or surrogate family connections is overt, often acute, and sometimes traumatic. In contrast, for children placed as infants, loss is, of necessity, more covert, emerging slowly as the youngster begins to understand the magnitude of what has happened. . . . In addition, there may be loss of a clear sense of genealogical connections and, in the case of transracial and inter-country adoption, loss of cultural, ethnic, and racial ties.

The impact of loss will also vary with the child's temperament, and the work of Rutter,[4] and of others who have written on 'resilience', are important sources. A 'born worrier' will go through life wondering what there was about him or her that was not worth keeping, and no amount of positive parenting will make this angst go away; a resilient child will shrug away the past and make the best of even not particularly good parenting by the substitute parents.

It is important, before considering the research findings, to take note of the limitations of our knowledge on the long-term outcomes of foster care and adoption. Turning first to the characteristics of the children, studies of family placement often include both infants and older children, those with emotional difficulties and those without. Some studies of foster care include children placed temporarily alongside others placed permanently, and in some US studies the term 'foster care' includes all children in out-of-home placements (for family placements the term 'foster family care' is used).

At the other end of the process a broad range of 'outcome' measures is used[5] and 'success' rates vary depending on the measures used and the length of time between placement and reported outcome. The well-being of the young adult (using a range of standardized instruments) is the most reliable outcome measure but more often 'output' measures are used. (Was the child placed? Was legal adoption completed? Was a satisfactory reunion with the birth parents achieved during childhood or as an adult?). Measures of satisfaction of the different members of the adoptive family are also used. Unsurprisingly, therefore, reported 'success' rates have varied between below 50 per cent and around 95 per cent.

Box 9.3.5.1 Dimensions of family placement

- The age of the child at placement.
- The degree of disturbance of the child prior to placement.
- The nature of attachments with birth family members and short-term foster carers.
- (For those in foster care) the duration of placements, the frequency with which they occur, and whether the child returns to the same foster carers on each occasion.
- (In the case of adoption) whether the child is adopted within the family (step-parent or relative adoptions), by foster carers to whom the child is already attached, or by parents not known previously (stranger adoption).
- Whether or not the child is adopted or fostered by parents of the same cultural and ethnic background or country of origin.

The placement process that researchers seek to evaluate is extremely complex. When, as with adoption or permanent fostering, the aim is for the child's life chances to be improved by their becoming fully a part of the new family, it becomes impossible to unpick the very many variables that will have had an impact on the mental health of the young person between placement at 6 weeks and maturity at around 26. (There is some evidence that adopted people move towards emotional maturity at a slower pace—not surprisingly with at least two extra hurdles to surmount: that of separation and loss, and that of making sense of their adoptive identity). In longitudinal studies, if numbers are large enough, it is possible to control for the major variables such as age at placement, disability, and emotional or behavioural problems at the time of placement. However, the many aspects of parenting, and the nature of any therapeutic input may all have had an impact on the placement. The researchers may seek the opinions of parents and children as to what they found helpful, but clear causal relationships between outcomes and variables such as parenting styles, models of social work practice, and therapy cannot be claimed.

In summary, whilst researchers have, for many years, sought to bring academic rigour to their studies, family placement remains an 'untidy' subject. The more complex the placement circumstances and the longer the timescale, the more difficult it is to attribute success to any one factor, type of placement, or model of intervention.

A review of the research evidence on outcomes

The above section explains why, although there are some random controlled trials of treatment approaches and of short-term foster care models, the literature contains more research syntheses of the different aspects of family placement[5–10] than 'classical' systematic reviews. The findings from the large volume of quantitative and qualitative research will be summarized under the broad headings of time-limited foster care placements and placements made with the intention that the child will become a full part of the adoptive or foster family. The emphasis will be on the second group, which will be further subdivided into placements of infants and placements of older children.

Time-limited placements

In general terms, short-term foster care is used along with other services in an attempt to improve family functioning so that the child may benefit from increased stability in the family home or as a short-term crisis intervention measure. The aims of short-term fostering can be summarized as: temporary care; emergency care; assessment; treatment and 'bridging'—to independence or between placements following placement break down.[7]

Generally short-term placements used as part of family support are successful in that few placements actually break down and most parents express satisfaction with the service. This is especially so if the placement follows careful preparation for the child, the birth parents, and the foster parents and if those who need a series of placements return to the same foster family. Several UK researchers have found that a 'keep them out of care at all costs' attitude tends to prevail in child welfare agencies, thus leading to too many ill-planned and ill-matched emergency placements, which in turn lead to placement break down and to unnecessary moves in care.

Testa and Rolock[11] conclude broadly positively from an overview of treatment foster care research in the United States, and Fisher and Chamberlain[12] report better outcomes for very troubled children in multi-systemic treatment foster care than for a 'service as usual' group. (These approaches involve placement with specially recruited, trained, and financially rewarded foster carers on a time-limited basis. Intensive multi-agency support is provided to the parents, foster carers, and children.) Though placement stability remains a problem, behavioural improvements are reported and these schemes are well rated by most of the young people and their foster carers. Some researchers report a problem of 'overstaying', but this should perhaps be reframed as a success, in that some young people settle in so well that, against the odds, the task-centred foster family becomes a 'secure base' and the foster parents continue to provide support to the young people as they move into adult life.

Associations have been found in some studies between positive child outcomes and practitioners who facilitate good contact between the birth parents, foster carers, and the child; provide support to the foster carers and the birth parents; and take a multi-agency approach to treatment of the child and parents before, during, and after placement.

Adoption and long-term foster-family placement

Whilst, in the United Kingdom and North America, adoption is considered to be the major placement option for most young children who cannot remain with their birth parents, opinion is divided (often along country lines) as to the importance of long-term foster family care as a placement of choice. Practice also varies in different countries in respect of placement with relatives. In most countries it is the exception rather than the rule for relatives to adopt (foster care, guardianship, or informal arrangements being preferred) whereas in the United States legal adoption by relatives tends to be encouraged.[13]

(a) Outcomes for children placed as infants

The largest volume of research on the long-term outcome of adoption concerns children placed 'voluntarily' as infants. However, inevitably, the practice referred to in these studies is already dated by the time the long-term outcomes can be measured some 20 years or so after the child was placed. Although some may have been born to mothers who had poor antenatal care, few of these early-placed children will have experienced neglect or maltreatment. However, with the growth of inter-country adoptions, studies of infants placed more recently are more likely to include substantial numbers of children who have experienced adverse conditions during their early months. It is likely that disruption rates will be higher than they have been in the past.

(b) The impact of placement in the short-term

An important source of detailed information on short-term outcomes of infant placements is the longitudinal study by Rutter *et al.*[14] which compares young Romanian children placed with British families with a cohort of English infants placed in 'stranger' adoptive families. Reactions to placement of the English infants who had generally good postnatal care are predictable in the light of knowledge about child development, attachment, separation, and loss. Those placed quickly settle with no obvious signs of stress; those with adverse early experiences including institutionalization (most of the Romanian infants) also appear to settle well if placed in their early months. Those placed when older than 6 months are more likely to show stress reactions at the loss of a carer to whom they are beginning to be attached, or to show

adverse reactions resulting from early maltreatment, neglect, or institutionalization.

(c) Signs of stress during childhood

The more robust studies of the mental health of adopted infants in their middle years and early adolescence are those that prospectively follow them as they grow-up. The conclusion drawn from these studies is that children placed with substitute families as infants tend to do better at each stage than non-adopted peers living in the generally adverse environments in which the children were likely to have lived had they remained with their birth parents.

All studies have found that, even for those with poor antenatal and birth history or who experience adverse circumstances in their early months, subsequent physical and cognitive development is generally good. However, children adopted as infants appear to be at a slightly higher risk of experiencing problems in their social, emotional, and behavioural development compared with other children raised in similar socio-economic circumstances. This is particularly the case with adopted boys. Information from longitudinal studies is supplemented by studies of clinical populations, such as those whose parents seek psychiatric help for them. Rates of maladjustment appear to be higher around the age of 11, and decrease as the children move into later adolescence. Some studies report that adopted children are more vulnerable on some measures of behavioural and emotional development than others, including an inability to settle, restlessness, a tendency to lie or fantasize, and difficulties in getting on with their peers and teachers. Low self-esteem and feelings of insecurity are also more likely to be present amongst children in their middle years and adolescence.

(d) Long-term outcomes

There is a lack of recently published quantitative studies of the well-being of adults adopted as infants. Summaries of the research[3,6,7] report that few of those placed as infants (around 5 per cent) will leave their adoptive families before the age of 18, in circumstances of conflict, which can be described as 'adoption break downs'. Qualitative studies have reported that around 80 per cent of both adopters and adoptees express broad satisfaction with the growing-up experience. Howe[15] uses in-depth interviews with the parents of adult adoptees to analyse the mental health problems that have persisted into adulthood and reports that, when more serious problems do emerge, the issue of adoptive identity often underlies a range of presenting symptoms. Also, amongst the over 80 per cent of adults who are generally satisfied with the experience of growing-up adopted are some who continuously or episodically have a sense of unease around questions of identity and the reasons why their birth parents 'gave them up' for adoption.[2] The most authoritative recent research on long-term outcomes is the Swedish cohort study of Lindblad *et al.*[16] These authors compared population data on nearly 6000 inter-country adopted adults (mostly placed when under the age of 5) with their non-adopted peers. They note that whilst there were more similarities than differences, the adopted children were more likely than peers brought up in similar circumstances to have psychiatric problems, including substance abuse, and there was a higher suicide rate.

In summary, when well-being and mental ill-health are the outcome measure used, adults who were adopted as infants tend to be healthier, have higher IQ scores, lower rates of criminal behaviour, and fewer psychiatric symptoms than non-adopted peers from similar backgrounds to those into which they were born and to be broadly similar to those brought up by birth parents living in similar circumstances to the adopters. However, the larger scale studies that allow for the control of the many intervening variables tend to lack detail on the children's experiences of family life and of any therapeutic interventions. It is therefore unclear whether any differences can be associated with adoption *per se* or with the more advantaged home circumstances of the adopted children.

(e) Children placed when past infancy

Researchers and clinicians tend to agree that beyond 6 months of age, the risks of moving children increase, and the older the child at placement, the more likely it is that there will be difficulties in the child's behaviour, which increase the risk of placement break down. Some delays in placement are caused by incompetence or poor practice. However, the main reason for delay in placement (sometimes referred to as 'drift') is contested legal proceedings. In most countries it is only possible in extreme circumstances to place a child for adoption without the consent of the birth parents, although adoption by long-term foster carers they have lived with for some years sometimes occurs. In the United Kingdom, United States, and Canada it is not uncommon for parental consent to be dispensed with by court order, but human rights legislation and the attempts at reunification mean that few children are placed from care before the age of 6 months. International adoptions tend to be delayed because of the search within the country of origin for an in country placement, or because of legal formalities.

(f) Medium-term outcomes

Many of the children placed when older bring problems with them into placement, to which may be added those discussed earlier, which are specifically associated with being adopted or fostered. For those placed from overseas, the difficulties are those commonly associated with institutionalization and privation of affection and consistent care. For a large proportion of those placed from care, the problems are those associated with maltreatment or neglect, including attachments with parent figures that may have been anxious, ambivalent, or avoidant, followed by the loss of those attachment figures. They may also have been separated from siblings and experienced multiple changes of carer.

Rushton and Dance[17] provide detailed accounts of the behaviour of 133 English children placed when over the age of 5. Eight years after placement, 19 per cent of the children had left their placements and only just over half of the 99 continuing placements were in the 'continuing/happy' group. Behaviours their parents had difficulty managing included over-activity, aggression, and destructiveness.

(g) Longer-term outcomes for late-placed children

Whilst some children placed in positive environments that provide committed and loving parenting and stability will recover from the adverse effects of early significant harm, developmental recovery cannot be anticipated in all cases. From a longitudinal study of over 1100 'hard-to-place' children placed in adoptive or permanent foster families not previously known to them, Thoburn[18] found that one in five of the placements had disrupted between 2 and 6 years after placement. There was a strong and statistically significant association between disruption and the age at placement (see Fig. 9.3.5.1). Of those aged between 7 and 8 years at placement, one in five experienced placement break down; this proportion rose to almost one in two for those placed between the ages of 11 and 12. The graph is less stable for teenagers, in part because numbers are smaller and statistics less reliable, and in part because families are

Fig 9.3.5.1 Age at placement and percentage of placements disrupting. (Reproduced from J. Thoburn, Evaluating placement: survey findings and conclusions. In *Permanent family placement: a decade of experience* (eds. J. Fratter, J. Rowe, and J. Thoburn), pp. 34–57, copyright 1991, Brirish Agencies for Adoption & Fostering (BAAF), London.)

more likely to 'hang on in' if they know the young person can be helped to leave home 'respectably' in a year or so. More recently, Rushton and Dance[17] found that the placements that disrupted did so between 6 months and 7 years after placement (a mean of 34 months). Rushton and Dance[17] followed up 90 children placed for adoption when aged at least three for an average of 7 years. Seventeen per cent of placements had disrupted (mostly before adoption was finalized), and for a third of those still in placement, there were many problems, which were often getting worse. Festinger followed up for 4 years 516 American children placed from care at a mean age of 2 years. The lower disruption rate (10 per cent) when compared to the UK studies may be explained by the younger age at placement and also by the fact that roughly half were adopted by relatives. She reported that many of the parents in the continuing placements reported difficulties and unmet needs.

Variables about the child and pre-placement history

Researchers and clinicians concur that, in addition to age at placement, variables relating to the child's behaviour and emotional well-being at the time of placement are most strongly associated with better or worse outcomes. These in turn are linked with biography, including experiences of early parenting and multiple caregivers. Having experienced early abuse or neglect has been found to be independently associated with less positive outcomes, whilst more positive outcomes are reported even for late-placed children who had formed a good-enough attachment with a parent or other main carer during the first few years of life.

Thoburn *et al.*[20] found that children of minority ethnic origin, whether placed with a family of the same or a different ethnic origin, were no more likely to experience break down than white children placed with white families. However, qualitative studies note that parents of a different ethnic and cultural background to the child have extra hurdles to overcome in the parenting process, and that some are unable to bring the child up to feel pride in his or her heritage, culture, and appearance, with consequent problems for self-esteem and identity.

Variables about the adoptive or foster families

Early studies reported an association between less positive outcomes and there being a 'home-grown' child younger or close in age to the placed child, but these have not been replicated more recently. The age of the adopters, whether they are single or in a partnership, experienced parents or childless, have not been consistently found to be significantly associated with placement break down. Two in-depth prospective studies of long-term foster care[21] and adoption[22] conclude that those new parents do best who can empathize with both the child and the family of origin, who enjoy a challenge, who have the skills to help the child with disabilities or emotional problems, and who, for older-placed children, can give out love even if the child gives little back and can find pleasure in tiny 'successes'.

Brodzinsky *et al.*[3] identify the importance of adopters treading a fine line between understanding and accepting the difference between parenting by adoption and parenting by birth, but not overemphasizing the difference.

Variables about placement practice and therapy

Few of the studies involving large enough numbers for statistical analysis look for statistically significant associations between outcome and placement practice or therapeutic interventions.

The child who remains for longer than a few weeks in temporary foster care is especially vulnerable to placement break down or moves made for bureaucratic reasons. Once settled in a planned long-term placement, when variables such as age at placement and behavioural difficulties are controlled for, break down rates for older-placed children are similar for children placed for adoption or in permanent foster families. Placements with relatives, and temporary foster placements confirmed as permanent (through adoption, guardianship, or administrative decision), have been found in most studies to have higher success rates that 'stranger' placements. Qualitative studies indicate that most children gain a sense of security from the legal status of adoption, but some can feel trapped and resentful, especially if they lose contact with birth relatives they want to see.[19,20,21,23]

Chapters by US and UK researchers in a book on birth family contact[24] indicate that post-placement contact, in itself, does not adversely impact on the attachment process, and that it can help new parents and children to be more comfortable in talking about adoption issues. For children placed when older, remaining in face-to-face contact with a birth parent, relative, or sibling, and being placed with a sibling, have been associated in several studies with more successful outcomes. However, this contact needs to be carefully managed and can sometimes be harmful to the child and to the stability of the placement.

The role of the psychiatrist

Child or adult psychiatrists will become involved in child placement work because they are asked to provide therapy for a child, young person, or adult who has been placed for adoption or in foster care, or for a teenager or adult who has lost a child to adoption. Whether working with task-centred or permanent carers, the special challenges of this different form of family life have to be acknowledged and incorporated into the therapeutic processes. It is for this reason that the need for specialist child mental health services for children in care or placed for adoption is now recognized, which adapt the full range of effective therapeutic approaches to the special needs of the adoptive or foster family. Barth and colleagues[25] have noted that adoptive parents tend to prefer attachment-based therapies to the parent-training approaches that have been demonstrated to be effective with non-adoptive families.

They hypothesize that in part this is because adopters believe that their special issues are better understood by the clinicians using these therapies, but they point to some potentially harmful effects of some of these methods, which can be experienced by the child as intrusive and coercive. Some clinicians who use attachment theories in their work also challenge the validity of some of these methods.[26,27]

Psychiatrists are often consulted about the advisability of adoption or a return to the birth parents or relatives. The message from research is that adoption and foster care will be better for most children than being left with parents who can not be helped to provide them with safe and loving care. However, they are not without risks, which have to be carefully weighed for each child, and the first step must always be to try to improve the quality of parenting in the birth family. At first sight, the break down rates for the placement of older children (now the majority of those needing placement) may appear discouraging. However, given their many difficulties, it should be welcomed that as many as 50 per cent of 11-year-old children, and more of those below that age, do find permanent substitute families. If permanent out-of-home placement does become necessary, the inherent risks demand the provision of the highest quality services provided for as long as needed.

Further information

For those who wish to pursue these issues in more depth look at references:

- 1, 2, 21, 22, 23 if you are providing therapy for a teenager or adult suffering the harmful effects of losing a child to adoption or foster care
- 7, 8, 10, 11, 12 on therapeutic and task-centred foster care
- 6, 9, 14, 15, 16, 17, 18, 19 to help with the decision-making process on substitute family placement options
- 2, 20, 21, 22, 23, 24 on birth family contact
- 3, 4, 14, 17, 21, 22, 25, 26, 27 on therapeutic approaches and methods when working with troubled children and their adoptive or foster families.

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9.3.6 Effects of parental psychiatric and physical illness on child development

Paul Ramchandani, Alan Stein, and Lynne Murray

Introduction

A broad range of physical and psychiatric illnesses commonly affect adults of parenting age. For example, approximately 13 per cent of women are affected by depression in the postnatal period, and the prevalence of depression in parents of all ages remains high. Many parents will also experience severe physical illness; breast cancer affects approximately 1 in 12 women in the United Kingdom, about a third of whom have children of school age. Worldwide HIV has an enormous impact on adults of parenting age. In some parts of sub-Saharan Africa up to 40 per cent of women attending antenatal

clinics are HIV positive. Many of these parental disorders are associated with an increased risk of adverse emotional and social development in their children, and in some cases cognitive development and physical health are also compromized. It must be emphasized that a significant proportion of children at high risk do not develop problems and demonstrate resilience,[1] and, many parents manage to rear their children well despite their own illness. Nonetheless these risks represent a significant additional impact and burden of adult disease (both physical and psychiatric) that is often overlooked.

This chapter reviews the current state of evidence regarding selected examples of psychiatric and physical conditions, from which general themes can be extracted to guide clinical practice. Some of the key mechanisms whereby childhood disturbance does or does not develop in conjunction with parental illness are considered, and strategies for management and intervention reviewed.

Parental psychiatric illness

There is now reasonable evidence to suggest that most types of psychiatric disorder affecting parents are associated with an increased risk of difficulties for their children. There are some differences in risk by type of disorder; however, there are also some commonalities, suggesting that some of the mechanisms may be shared. Children's disorders may resemble those of their parents[2,3] but there is also evidence of a much broader range of problems, including adverse effects on children's social, emotional, cognitive and physical development. In the following section we will focus on parental depression, schizophrenia, eating disorders, alcoholism and substance abuse, and anxiety, but similar issues apply for other disorders not considered here.

Depression

Depression in either parent is associated with an increased risk of child psychopathology and other developmental difficulties, with the risks continuing into adulthood.[3] The longest running longitudinal study[4] found that, as well as a three-fold increase in major depressive disorder, the adult offspring of depressed parents had increased rates of anxiety disorders and substance dependence, as well as greater social impairment and physical health impairment. There has been a large body of research focusing on depression affecting mothers in the postnatal period, with studies demonstrating that infants and children have an increased risk of emotional and behavioural problems.[5] Some studies have suggested that children's cognitive development may also be affected, although the results from studies are not consistent.[6] Similarly, there is a suggestion that boys may be more affected than girls in early childhood. As the children enter adolescence an increased risk of mood and anxiety disorders emerges.[7] More recently research in developing countries has shown an association between postnatal depression and an increased risk of physical health problems in infants such as poor growth and diarrhoel illness.[8]

Much less work has been done on depressed fathers, although consistent evidence is now beginning to emerge of an independent effect of paternal depression on children's development.[9] The overall impact may be less than that of maternal depression, and there are also conjoint effects to consider, as depression in one parent can often co-occur with depression or another psychiatric disorder in the other parent. Similarly there may be protective effects if one parent remains well.[10]

While genetic factors clearly play an important role in the transmission of risk from parents to children, environmental factors, and the interactions that occur between genetic and environmental factors, also have substantial influence.[11,12] In the case of depression, the core symptoms of low mood, loss of interest and low energy can have a significant impact on parenting capacity and parent-child relations. These include a parent's capacity to be responsive, consistent, and warm when interacting with their children, particularly in the first few years of life. For example, depressed mothers may be less vocal, less positive, and less spontaneous than controls, more negative, unsupportive, and intrusive, and have more difficulty in communicating and listening to their young children.[13, 14]

Depression in either parent is strongly associated with marital discord.[15] This may play a key role in mediating the effects of parental depression and may be a more proximal predictor of child outcomes than depression.[13] The way in which conflicts are resolved may be very important and depressed parents are likely to use less effortful strategies, such as withdrawal. Children are generally more at risk as they are exposed to an increased number of risk factors, and children whose parents are depressed are particularly at risk if they are also socio-economically disadvantaged.

The direction of effects is not all from parent to child, and temperamental and behavioural factors in the child may also contribute to increasing family discord, parental psychiatric disturbance, and parenting impairments, and ultimately to disturbances in parent-child attachment. Infant irritability and poor motor control, measured before the onset of any maternal depression at 10 days postpartum, increase the risk that a mother will become depressed.[16] The influence of parental depression on child development thus represents a complex bidirectional interaction between individual vulnerability (which may be genetic), influences of depression on parenting characteristics, parent–child relationships, the wider context of the parental relationship, and other aspects of social disadvantage.

Schizophrenia

Parents with a diagnosis of schizophrenia have a greatly increased risk of having children who later develop schizophrenia themselves. Risks to child development are identified from birth, with an increased likelihood of obstetric complications, not fully accounted for by maternal behaviour during pregnancy, or by genetic risk.[17] During childhood, prior to the onset of any psychiatric symptoms, attentional problems similar to those found in adult schizophrenic patients have been identified and these problems not only persist into adulthood, but attentional problems have been identified as key neurobiological indicators of risk for subsequent schizophrenia or other psychopathology in adolescence and young adulthood.[18]

Social difficulties with peers and teachers are found in many longitudinal studies of children with schizophrenic parents,[19] although not necessarily to a greater extent than in children with parents suffering from affective disorder and a higher IQ can be protective. Social relationship problems and associated thought disorder may become more marked in adolescence and seem to be predicted by attentional problems.[18] As young adults, children of schizophrenic parents are at high risk for schizotypal behaviour, although this broader range of difficulties does not necessarily distinguish them from parents with affective illness.

A pattern of disturbed communication has been described in families with a schizophrenic parent,[20] but the importance of these interactions in explaining long-term outcomes has been questioned.

Overall, cognitive and attention difficulties appear to be largely associated with specific brain abnormalities linked with schizophrenia, but other kinds of childhood problems are probably influenced

more by the general family disruption associated with a parent who requires hospital admission and who may have difficulties with employment and other social relationships beyond the family.

Eating disorders

Eating disorders occur commonly among women of child-bearing age.[21] Studies have raised concern that mothers' attitudes and behaviours regarding food and body shape, may influence their children's feeding, and ultimately the children's own attitudes to body shape and eating.[22] Children are particularly vulnerable at two stages of development—infancy and adolescence. During infancy feeding and mealtimes take up a significant part of the day and provide important times for close communication between parents and children. A Scandinavian study has indicated that failure to thrive may be a risk in the first year amongst women with a history of anorexia nervosa.[23] One controlled observational study of 1-year-old children of mothers with eating disorders found that the mothers were intrusive with their infants during both mealtimes and play, and they expressed more negative emotion and conflict during mealtimes than controls, and allowed their children less autonomy.[24] Furthermore, infant weight was independently and inversely related to mealtime conflict.[25] Follow up studies of children of mothers who have experienced eating disorders in the postnatal period indicate that in middle childhood they are more likely than control children to value themselves by body shape and weight, and to use dietary restriction.[22, 26]

During adolescence children become more aware of societal pressures and develop increasing interest in body shape and attractiveness while preoccupied with their own concerns about food, body shape, and weight. Children may model themselves on their parents, and parents may influence their adolescent children directly by expressing attitudes towards their children's weight, shape, and eating habits. However, it should be emphasized that, in common with most parent psychopathology, the children of parents with eating disorders are not invariably adversely affected. Some parents manage well and their children develop without apparent problems.

Alcoholism and substance abuse

A substantial body of evidence has been amassed on the effects of parental alcoholism and substance abuse.[27] They are wide ranging, identifiable throughout development, and work has highlighted the importance of the social effects on the child in addition to physical and psychological outcomes.[28] Both genetic and environmental factors seem to be involved.

The impact of maternal alcoholism on the developing child can be found from the prenatal period.[29] Present in 0.01 to 0.03 per cent of normal births, foetal alcohol syndrome appears in 5.9 per cent of births of alcoholic women. There is also considerable evidence that infants exposed prenatally to heroin and cocaine are at increased risk of a number of developmental difficulties which may persist throughout childhood.[30]

Studies of outcome consistently describe impaired cognitive and social development in children of alcoholics and heroin users.[27,30] An increased risk of attention-deficit hyperactivity disorder, attention problems, and impulsivity in children of alcoholics is the most consistent finding. Children of drug abusers may also be more aggressive and have fewer friends and are at risk for criminality, depression, anxiety, and somatic problems. Children of alcoholics have an increased risk of becoming alcoholics themselves and, similarly, children of drug abusers also have an increased risk of drug abuse in adolescence, although many are resilient and do not develop similar problems themselves.

Social factors such as poverty and social isolation are known to influence child development adversely and it has been difficult to differentiate between the effects of the disorder and the associated adversity. Substance abuse in the parent may lead to impaired parenting and an impoverished social environment, leaving children vulnerable to neglect or abuse and contributing to impaired social and cognitive functioning, psychopathology, substance abuse, and delinquency but the relative impact of each factor has yet to be resolved. Studies have identified deficits in parenting behaviour and, in particular, neglect and harsh discipline.[31] Divorce and marital conflict are also more likely and there is evidence of assortative mating, all of which are likely to compound the risk for the children.

Stressful life events, and in particular those related to family conflict, have proved to be important in accounting for the link between paternal alcoholism and alcohol use in their offspring.[32] Overall, clinicians have emphasized a family perspective when conceptualizing the multiple levels of stress and vulnerability associated with parental alcoholism and opiate abuse and the need to enhance social supports for the family.[30]

Anxiety disorders

There is less information about children whose parents have anxiety disorders but there appears to be a considerable degree of specificity in the familial transmission of anxiety disorder.[33] One study showed a two-fold increased risk of anxiety disorders among offspring of parent probands compared with offspring of substance abusers or controls.[34] However, they are also at risk of other kinds of problems such as depression.[35]

In a search for the mechanism of transmission, it has been suggested that children at risk for developing anxiety disorders have a temperamental vulnerability characterized by behavioural inhibition and autonomic reactivity, identifiable in infancy by an increased startle response.[36,37] The question of the relative influence of genetic or environmental factors does suggest a lesser genetic component for anxiety disorders,[38] and a developing line of research has led to a number of aspects of parenting behaviour of parents with anxiety being considered important, including over-protection and the limiting of children's opportunities to develop new skills[39]. There appear to be some specific differences by type of anxiety disorder with, for example, mothers with social phobia demonstrating characteristic patterns of modelling of anxiety by the parent, and failure to provide encouragement/opportunities for child autonomy.[40]

Recent lines of research have also explored possible risks in utero to the developing foetus. Children exposed to maternal anxiety in utero are at an increased subsequent risk of behavioral problems. This increased risk appears to persist through childhood, leading to suggestions that the mechanism may be in part mediated through an antenatal effect on the HPA axis of the developing foetus.[41] More research is needed to clarify the mechanisms but it is clear that children whose parents have anxiety disorders are at risk of developing psychiatric disturbance themselves.

Parental physical illness

Many families experience chronic parental illness and paradoxically, as treatment techniques improve illnesses may extend over longer time periods, which may have greater impact on family members

including children. There have been limited reports about the impact of physical parental illness on children, however the importance of this area is beginning to be recognized, particularly in relation to parental cancer, and also HIV.[42] Similar associated difficulties can arise with many other parental illness, particularly chronic ones such as diabetes. However we will here confine our comments to parental cancer and HIV, and the general issues that these conditions illustrate for the developing child.

Cancer

Parental cancer is likely to be associated with depression and marital difficulties, both risk factors for the child. The balance of evidence indicates that their children are at increased risk of developing psychological disturbance.[43] The impact of parental cancer on family communication and child outcomes may vary according to the child's developmental level, their gender, the presence of disability in the child, and the parent's level of psychological distress and marital discord.[44]

A recent review[45] found that adolescents who had a parent with cancer had higher levels of emotional disturbance, than a normal population sample, but younger children were not consistently found to exhibit higher rates of problems, although some studies suggest this. Children's own responses to their predicament are likely to affect their eventual adjustment. Problem-focused or active coping affects the stressors (e.g. seeking information, positive reinterpretation of stressful events) and is expected to be more adaptive while emotion-focused coping (e.g. venting emotions, denial, apathy) draws attention away from the stressors but may place children at risk for anxiety and depression.[46] Health professionals may need to assist parents in recognizing and coping with their children's distress when it is present. Specifically, communication about the parental illness and how the children feel appears to be crucial to children's coping.[47] The levels of anxiety and distress amongst the children are related to whether they are told about the illness and the quality of the communication with the parents, with informed children having lower levels of anxiety than those who are uninformed.[44]

HIV status and AIDS

Women of child-bearing age account for an increasing number of sufferers of HIV/AIDS in the developing world and there is now increasing evidence that their children are at increased developmental risk, even if the children are not HIV positive themselves.[43] Young children may have fewer problems[48] but those of school age are at risk for externalizing and internalizing problems, lower social skills, and academic achievement difficulties.[49] Maternal depression is relatively common amongst women diagnosed with HIV during pregnancy,[50] and the impact of this on their caregiving capabilities may be one of the key mechanisms by which children are affected. Given the enormity of the HIV pandemic and possible parallels with other common infectious diseases such as Malaria and TB, there is serious need for further research in this specific field.

Summary of mechanisms

Some of the key mechanisms by which increased risk for child disturbance is transmitted from ill parents to their children have been described above. In cases of psychiatric disorder the link may in part reflect genetic transmission, but clearly a considerable amount of the variance is accounted for by environmental mechanisms and a number of such mechanisms have been proposed.[5,11] Most of these can apply equally to parental physical or psychiatric illness. First, parental illness may interfere with parental functioning and parent-child interactions, for example where a parent becomes withdrawn or preoccupied and relatively unavailable to the child. Second, a number of family and environmental factors such as marital/family discord and severe housing or associated economic deprivation are associated with mental illness and these constitute risks in their own right, as well as sometimes being a direct consequence of the parental disorder (for example, parental depression leading to increased marital conflict, although the reverse direction of causality can also occur). Third, in rarer instances parental symptoms may impinge directly on the child, for example where the parent incorporates a child into the core symptomatology such as a delusion or an obsession. Finally, it should not be assumed that influences are unidirectional. Child characteristics such as early temperamental difficulties or behavioural problems may influence the outcome of parental illness, particularly in the case of depression.[3] Child characteristics such as coping style, intellectual ability, or sociability may be particularly important in explaining resilience.

Implications—responding to a parent in clinic and preventive interventions

As the influences of parental illness on children's development become better understood there is increasing recognition of the potential importance of addressing parent-child links. This is both in making better enquiry about child welfare when a parent is seriously ill, but also enquiring about parental health when a child presents with a disorder such as depression (e.g. NICE guidance on depression in children and young people[51]) Two particular areas will be considered here; when a parent presents with a serious illness (psychiatric or psychological) and potential intervention strategies with high-risk groups who may or may not be presenting to health services.

When parents with serious physical or psychological illness are seen as outpatients, especially if they are subsequently admitted to hospital, it should be routine to enquire about children—their ages, developmental and scholastic progress, child care arrangements, and family support. In the limited time available, it makes most sense to enquire about the areas most likely to be affected by the parental disorder.[52] For example, where a parent with depression has young children, enquiry could focus on the level of care that the parent feels able to provide to the children, the feelings that the parent has for their children, and the presence of any marital or family discord, as well as the available support. These questions obviously require sensitive handling, as parents can easily feel blamed for any effect that they perceive their illness may have had on their children. In many cases provision of appropriate support and treatment to the parent will have a sufficiently beneficial effect for the whole family that no further intervention is required, (for example, findings from a recent large randomized controlled trial identify improvements in children's outcomes when their mother's depression is treated.[53]) However it is crucial to ask and make at least this preliminary assessment. Close collaboration with the primary care team, including the family's general practitioner, is of considerable importance. The general practitioner's involvement may be critical, both in terms of potential treatment and support for the family, and also as a

source of knowledge of the wider family system, and the resources available to the family.

In those cases where a child is more severely affected (either directly by a parent's illness or by other related factors), consideration should be given to including relevant children's services, either Child and Adolescent Mental Health Services (CAMHS) or children's care services. Close collaborative relationships between child and adult mental health services clearly ease potential joint working, and should be the norm. Communication with, and the close involvement of, the family's general practitioner, can also contribute to a plan to benefit the whole family system.

In those cases where a parent has a chronic illness and the clinician gets to know them over a period of time, it can be helpful to ask about children's understanding and knowledge of the parental illness and the extent to which it has been discussed within the family. These issues will need to be carefully and sensitively handled. Families have a range of ways in which they communicate and it is important not to compound the parent's problems by making them feel their illness is harming their children. As far as inpatients are concerned, it is important that facilities should be available for children visiting and, unless specifically contraindicated, regular contact should be encouraged, and appropriate play and other materials should be available. Discussions should be held with the patient and/or relatives about child-care arrangements, and the patient and family need to be helped to think about providing the child with an appropriate explanation about parental health and absence.

Communication either about the parental illness or the associated discord may help to alleviate some of the risks for childhood problems.[44] Studies of marital disruption and divorce have found that children cope better with marital conflict when they are given some explanation or told that the conflict has been resolved.[54] Children often feel left out and, without knowledge of the parental illness, they may be particularly likely to attribute any family conflict or disruption to themselves in their effort to understand the changes taking place. Intervention, including working with family communication, can have significant positive effects if well handled. Some guidelines are available.[55,56]

Less work has been conducted in families where the parent is physically ill, although reports of treatment once the family members are experiencing problems are providing some ways forward.[45] Parents may consciously avoid disclosure because of the questions they anticipate from their children, particularly about death. Communication in this context is not only a matter of disclosure of the illness but a starting point for ongoing discussion and questions, without which children may be at increased risk.[57] The most important role of support services may be to rehearse with parents the kinds of questions that might occur and how they could respond, a strategy which may also facilitate discussion of the ill parent's anxieties.

Prevention

Beyond the scope of the individual parent and family in clinic there have been a number of studies which have examined the possibility of preventative intervention in high-risk groups. This is most often conducted in the context of parental depression. One series of intervention studies with children of parents with major depression has shown marked improvement in family functioning and child outcome.[58] These interventions have taken a variety of formats, but all include a component of psycho-education directed to the whole family. However, in another trial, the involvement of parents in a group programme for adolescent offspring of parents with depression did not show any additional benefit.

Some preventive intervention can clearly be accomplished earlier. In infancy it is possible to promote the development of secure parent–child attachment, which should be protective even if the parental illness is chronic. Maternal sensitivity can be enhanced using videotaped mother-child interactions, which can increase the rate of secure attachment in at-risk families.[59] In the case of maternal eating disorders there is now some encouraging evidence for the effectiveness of video feedback treatment to enhance maternal responsivity to the infant, and to decrease mother-infant conflict.[60] A recent review of treatments for mothers and infants in the context of maternal depression found that treatment of maternal depression alone did not appear to mitigate the impact of the depression on the child. However, a number of different mother-infant psychotherapies did appear to confer benefit on mother child interaction and child outcome. However, much work remains to be done.[61]

Conclusions

In conclusion, children of parents with physical or psychiatric illness are at risk of a wide range of developmental and psychiatric difficulties, although not all will develop problems. Future work should be directed to developing and evaluating ways of providing support so that parents can best manage their illnesses and to prevent or mitigate any negative effects on their children.

Further information

The SCIE parental mental health network (www.scie.org.uk/mhnetwork/resources.asp)

The Children of Parents with a Mental Illness (COPMI) (www.aicafmha.net.au/copmi/index.html)

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9.3.7 The effects of bereavement in childhood

Dora Black and David Trickey

Introduction

Bereavement is not an illness in itself, although it may cause illness or predispose to one. The reaction to the loss of a loved one may lead to temporary or long-term psychological distress and/or loss of function, and may occasion consultation with the general practitioner and referral to mental health professionals.

During the first two years of life, through instinctive behaviours which are modified by experience, infants and their main carers develop an attachment. This bond between a child and his caretaker(s) ensures the child's survival, enables his or her optimum physical, intellectual, and emotional development, and in due course ensures the survival of the species. The nature of the attachment between infant and carer(s) influences the way in which children come to view their social world; the pattern of attachment developed in the first two years of life often remains stable and is associated with the way in which children relate to other people later in their life. Attachment behaviour has been observed across different species and has obvious benefits for survival. However, part and parcel of attachment for the child is distress at separation. Infants who develop a secure attachment can gradually tolerate longer periods of separation from their carer and any distress is rapidly assuaged when they are re-united with their carer. When considered within the context of attachment theory, it is inevitable that permanent separation (e.g. through bereavement) will cause distress for the bereaved. Parkes reviews the body of attachment research and offers a comprehensive description of attachment with particular reference to its role in understanding the impact of loss.[1]

The DSM-IV-TR has a classification for 'Bereavement' (V62.82) differentiating it from 'Major depressive disorder' (296.2) which, unless the symptoms are severe, is generally not diagnosed until 2 months after the loss. ICD-10 has no separate classification for bereavement and suggests the use of 'Adjustment disorders' (F43.2) for temporary reactions to life-events, and 'Death of a family member' (Z63.4) for normal bereavement reactions not exceeding 6 months in duration.

In industrialized countries between 1.5 and 4 per cent of children are orphaned of at least one parent in childhood. Premature deaths in the parenting years may be due to illness, accident, war, civil conflict, natural and man-made disasters and the incidence of these are all higher in developing countries. It is estimated by UNICEF that, in some developing countries, 21 per cent of children are orphaned of at least one parent; with HIV AIDS responsible for up to three-quarters of the deaths.[2]

Reactions to the death of a parent

Research studies

It is generally accepted that loss of a parent in childhood is associated with harmful psychological consequences, however it is difficult to tease out the independent effects of adverse circumstances before the death, the loss itself and the subsequent disruption to the child's life, including the possibility of compromized parenting post-bereavement[3,4]. Most published research about bereaved children describes small-scale uncontrolled studies carried out on children and adolescents referred to mental health facilities. Dowdney comprehensively reviews the research examining the psychological impact of being bereaved of a parent in childhood. She concludes that despite methodological weaknesses, certain findings consistently emerge: 'Children do experience grief, sadness, and despair following parental death. Mild depression is frequent, and can persist for at least a year after parental death'. Bereaved children commonly exhibit a range of psychological symptoms that may not constitute a specific disorder, but the severity of which is likely to warrant referral to a specialist service for one in five bereaved children.[5]

Long-term effects of bereavement

There continues to be debate about a possible link between being bereaved of a parent as a child, and mental health as an adult. The debate is complicated by methodological weaknesses in studies, inconsistent results and difficulty in isolating the impact of experiences which may precede or follow the loss. Any long term consequences of parental bereavement can be mitigated by the subsequent provision of adequate parenting[6,7]. Furthermore, studies in behavioural genetics are increasing the understanding of how genetic endowment interacts with environmental hazards to lead to the presence or absence of mental health problems.[8]

Cultural and religious issues

Reactions to loss are biologically based and are therefore likely to transcend cultural differences, although culture may modify their expression.[3] Religious beliefs about what happens after death can be confusing to young children at the stage of concrete thinking and need to be presented taking account of their developmental stage. A helpful text[9] gives guidance on religious and cultural differences in the conceptualization of death.

Developmental issues

Young children react to the absence of a parent by developing an anxiety or depressive reaction, often expressed somatically (regression

in acquired control, anorexia, insomnia), but young children cannot distinguish temporary from permanent loss[3,10]. Research consistently demonstrates that children ordinarily do not develop a full understanding of the concepts of death before the age of 7 years, although younger children of 4 years and above can understand it with appropriate help.[11]

Pre-pubertal schoolchildren can be helped more easily to comprehend the reality of death, especially if they are given an opportunity to see for themselves the cessation of function. In cultures where viewing the body is the norm, there may be fewer misconceptions about death among children, but this should not be undertaken where the body is mutilated.[12] Although difficult to substantiate scientifically, clinical literature suggests that attending the funeral helps the grieving process.[5]

For adolescents the death of a parent may come at a time when they are freeing themselves from dependence and may have been in conflict with the parent who subsequently dies, leaving the young person with feelings of guilt and anger. Suicidal feelings are more likely to be acted upon if part of a depressive reaction. Adolescents are more able to sustain sad affects and express grief directly, but they may also react with behavioural and academic difficulties.

The reader is referred to Dyregrov for a more comprehensive description of common reactions to bereavement in childhood.[13]

Children and adolescents with learning difficulties may be at higher risk for developing psychological problems following bereavement, because of their cognitive difficulty in understanding the components of the concept of death and because of their greater dependency.[14] Everatt and Gale provide a helpful review of the available research and draw implications for bereaved children with learning disabilities.[15]

Traumatic bereavement

As with adults, children who witness horrific events involving the death or severe injury of people close to them, or upon whom they are dependent, are at risk of developing post-traumatic stress disorder (see Chapter 9.3.2). Traumatic symptomatology can impede the resolution of grief through mourning as for mourning to proceed, the child has to summon up an image of the dead person. However, if when she/he tries to imagine the deceased, a frightening picture appears or she/he experiences again the helplessness or terror he felt at the time of the death, she/he will tend to avoid recalling the person and thus will not be able to grieve for her/him. Similarly, children whose parents die through suicide or homicide are more likely to have difficulties. In such cases, not only is the nature of the death traumatic and more difficult to make sense of, but there is often an unhelpful media interest and, social support systems that would ordinarily be available may find the circumstances of the death unbearable. If there is a body at all it may be disfigured or its release to the relatives may be delayed the investigation or the authorities and mementoes or suicide notes may be retained by the authorities. Children who have been traumatized by experiencing the sudden, violent, or horrific death of someone close to them are unlikely to benefit from bereavement counselling or therapy until the post-traumatic stress symptoms have been treated[16,17].

Other losses

Much of our knowledge of the impact of bereavement on children and young people is drawn from research on children bereaved of a parent. Other losses have been less well studied; however reactions may be similar depending upon the relationship between the child and the deceased. The death of a grandparent, particularly if he or she lived with the child or carried out caretaking functions, can be devastating to child and parents. Sibling death carries a high morbidity for the survivors, but this can be mitigated by preparation for the death when possible and by participation in community rituals.[18] Adolescents losing a sibling often deny the finality and universality of death, even when these concepts are well established prior to the death.[19] The losses of friends, of pets, or of homes, whilst eliciting sadness, are less likely to provoke pathological grief reactions provided that the child is supported by parents and other adults who are not themselves withdrawn in grief. However, adolescents are affected by the suicide of a friend. A controlled study found that there was a higher incidence of depression than in a matched population sample, although the incidence of attempted suicide was no higher.[20]

Evaluation of treatments

Many of the adverse sequelae of childhood bereavement can be modified or prevented by an intervention before the death or shortly afterwards. In a controlled study, a brief family intervention 2 months after the death of a parent significantly reduced children's morbidity at 1 year post-bereavement. The differences between the treatment and the control group were no longer significant at 2 year follow up, but some of the more affected children had been lost to follow-up, making comparison difficult. But even if by 2 years the effect of the intervention is no longer significant, there is an argument for intervening to relieve symptoms and reduce suffering in the short and medium term.[21] Schut & Stroebe's most recent review concludes that, although in the general adult population a bereavement intervention is more effective for those with more complicated grief reactions, 'children are likely to be a special case, perhaps benefiting from primary intervention.' (A primary intervention is one which is open to all bereaved people rather than targetted at those who are at risk of difficulties such as following traumatic death, or those experiencing complicated reactions.).[22]

Management

Children whose symptoms reach the threshold for a diagnosable psychiatric disorder require a careful clinical assessment to determine the most appropriate treatment, and other sections detail the appropriate interventions for disorders such as depression (Chapter 9.2.7), anxiety (Chapter 9.2.6) or Post-traumatic Stress Disorder (PTSD: Chapter 9.3.2). Some studies have found that parents report fewer symptoms in their bereaved children than the children do themselves; this means that it is important in research and clinical practice to interview the children individually if possible.[5]

Children bereaved of a carer urgently need to be looked after and will transfer their attachment to a new caretaker who is available for them and responsive to them. Supporting a widowed parent in his or her grief, and enabling the process of mourning to occur by providing practical help (child care, financial advice, etc.), may be as important as counselling in helping the children. It may be also appropriate to offer support, supervision and guidance to other adults involved in the child's life such as teachers and religious leaders.

The therapeutic elements of appropriate interventions include the promotion of communication within the family about the dead

parent, the promotion of mourning through reminiscing, the appropriate expression of feelings, and making sense of the death. An overview of techniques used directly with children and young people is provided by Stokes.[23] Techniques for use with children, many of which can be done in groups, include:

- Using art and story-telling
- Writing letters to the deceased
- Creating memory boxes to store reminders of the deceased
- Rituals (such as lighting candles, releasing balloons)
- Making something that represents different aspects of the person (e.g. salt statues of different colours)
- Playing games which encourage children to open up
- Role playing

As with all direct interventions with children, the choice of which techniques to use depends upon the child factors such as age and intelligence, the therapist's or counsellor's training, skill, and experience, the nature of the therapeutic relationship and the organizational context. These interventions can be provided by carefully selected, well-trained and well-supervized volunteers. As part of an intervention, young children may require help to understand what has happened to the deceased by offering a careful and sensitive explanation of what death means in straight-forward biological terms ensuring that the child understands what they are being told. They may also need help to recognize, understand and cope with sad affects both in themselves and in the surviving family members.

Given the indications that problems may develop much later, a useful intervention strategy should include follow-up appointments after any time-limited intervention. Children who have been prepared for the death of a family member have been shown to fare better, in terms of anxiety levels, than those who have not.[24]

Conclusion

Bereavement in childhood, particularly the loss of a parent, represents a significant adversity, although the majority of bereaved children do not develop anything other than transient symptoms. Nevertheless, there is evidence that a brief preventive intervention can reduce subsequent morbidity. Children, who lose a parent through suicide, homicide, accident, or disaster, especially if they have witnessed the death, are at high risk of developing post-traumatic stress disorder and other psychiatric disorders and their treatment needs should be assessed by mental health professionals.

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Various useful resources for professionals, parents, carers and bereaved young people and children are provided by the UK child bereavement charities: The Child Bereavement charity (www.childbereavement.org.uk) and Winston's Wish (www.winstonswish.org.uk).

Help is at Hand: A resource for people bereaved by suicide and other sudden, traumatic death. This booklet is published by the National Health Service in the UK, and provides particularly good advice for parents of suddenly bereaved children. www.dh.gov.uk/assetroot/04/13/90/07/04139007.pdf

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9.4

The child as witness

Anne E. Thompson and John B. Pearce

Introduction

In the last 20 years, many societies have paid greater attention to children's rights and the importance of protecting children from abuse. As perpetrators of abuse have been tried in court, so more children have been called as witnesses. From being described as 'the most dangerous of all witnesses', children have become recognized to be able to provide valuable and credible testimony in the correct circumstances. Many jurisdictions are now making allowances for children so that their testimony can be delivered in court as fully and accurately as possible. It is no longer tenable to dismiss the capacity of a child to be a witness in court simply because of their age. **Children may be less reliable, as reliable, or more reliable than adult witnesses, depending on a variety of developmental and environmental factors**.

Developmental considerations for children as witnesses

Memory is immature below the age of 12

In the first 3 years of life recall is via preverbal memory (sometimes called eidetic memory), which allows early events to be recalled visually but not in words. Preverbal memory is very accurate but is largely lost around the age of 3 years as language develops. This form of memory remains longer in children who have delayed language development. The loss of this early form of memory explains why **adults do not usually have conscious memories from the first 3 years of life**. It is only around the age of 3 years that experiences begin to be memorized in 'explicit memory', which is accessed by verbal recall.[1] Memories laid down in explicit memory are organized according to hierarchical cognitive structures formed by the child's past experiences of the world. In young children, this organization is rudimentary. This means that new memories are stored with little selection or adaptation to reflect preconceptions, and **in younger children the retrieval of stored memories from poorly organized mental representations is difficult**. External prompts and cues help children recall more fully but in court witnesses are not generally allowed to be 'led' by questioning. Younger children are more likely than older children to forget information over time. **By the age of 12 years, children are generally considered to have the same capacities to lay down memories and recall information as adults**.

Surprisingly, children's **immature memories can sometimes actually improve the quality of information provided in witness statements**. For example, events may be memorized without being influenced by the prejudices that affect adult perceptions, and seemingly trivial details may be memorized by a child whose primitive cognitive schemas allow incoming sensory information to be memorized unselectively.[2] However, cognitive **immaturity more often acts as a barrier to children giving their testimonies fully and accurately**, especially when they are asked to talk about what they remember, rather than being allowed to communicate by behaviour or play.

Children's expectations of adults in conversation influence testimony

By the age of 6 years, many children are using the syntax and grammar of adults in their spoken language. However, their vocabulary is still limited and they are **easily confused by sophisticated or complex speech**. A particularly important aspect of language development for child witnesses is the role played by children and adults in conversational partnerships. **Children are used to being co-operative partners when talking with adults**. They attempt to please by providing answers to questions, even if they do not know the answer or have not understood the question.[3] Young children rarely answer 'I don't know' to a question they are uncertain about and prefer to give a false 'yes' or 'no'. This willingness to provide an answer is probably encouraged by the frequent experience of being 'tested' by adults who already know the answers to the question (for example, 'Look at this picture! Can you see the duck?' or 'How many sweets am I holding?'). As children expect adults to know the answers to their questions, and if a question is repeated, children may change the answer they gave in the assumption that the questioner feels it to be wrong. **The readiness of young children to please adults in conversation no doubt adds to their suggestibility.**

Young children are particularly suggestible

The suggestibility of children as witnesses has been a major concern in legal arenas. Modern research does not endorse the stereotype of the child witness as being highly suggestible. However, there is clear evidence that both children and adults are prone to suggestion at times, and that **pre-school children are particularly vulnerable to this**. The necessity to avoid suggesting information or answers to a child has led to the development of guidelines for interviewing child witnesses in several jurisdictions.

Immature moral development is not always a problem

According to Kohlberg's seminal description of children's moral development, children below 10 years of age operate with 'pre-conventional morality' and evaluate events according to whether the child themselves will gain reward or avoid punishment. Only after this age do children develop 'conventional morality' and begin to be motivated by the approval of other people and society. Therefore, **only older child witnesses have a full understanding of their moral obligations in court**. However, young children, who may hold concrete worldviews such as 'bad people must be punished' as moral imperatives, may be strongly motivated to tell the truth in court.

Children do not lie more than adults, but their lies are more easily detected

Contrary to popular belief, children above the age of 3 years have no more difficulty than adults in distinguishing fact from fantasy. Neither do children tell more lies than adults (although children's lies are more often unconvincing and therefore more easily discovered). **Children and adults are motivated to lie for similar reasons**.[4] The two motives for lying, which may particularly influence child witnesses asked to testify in cases of child abuse, are fear of personal recrimination and a wish to protect those to whom a child feels loyal.

Immature sense of time and short attention span influence testimony

Before 8 years of age, children do not generally have a clear sense of time. Young children therefore often have a muddled recollection of the timing of events and they may have difficulty saying how many times an event occurred. Similarly, **children below this age have short attention spans**, and readily become bored or overwhelmed by prolonged questioning. Both of these developmental limitations can cause difficulties in the preparation of witness statements when a child appears in court.

Traumatic memories may be recalled with more or less clarity

Many of the events child witnesses are called upon to remember were unpleasant and frightening at the time. These events will have been experienced in a state of high emotional arousal. Clinical experience suggests that **emotional arousal can either enhance or diminish recalled information**. For example, extremely traumatic events such as watching a parent being killed can be remembered by child witnesses in a series of highly accurate and detailed visual images that persist in memory over time. By contrast, some children process potentially overwhelming experiences using a variety of psychological defense mechanisms, which limit the amount and accuracy of material available in explicit memory. The psychological trauma associated with the witnessed event and the emotional state of the child during subsequent recall are both likely to have an influence on a child's capacity to give evidence in court.

Table 9.4.1 Guidelines for interviewing child witnesses

- Allowing a child to talk freely without being questioned maximizes the chances of accurate recall
- Questioning children may elicit additional information but lessen accuracy
- Accuracy is greatest when children give their statements as soon as an event occurred
- Children's accounts are most likely to be accurate when they tell the story for the first time
- Children are less likely to give a full account if they feel under pressure from the interviewer
- Younger children may attempt to please an interviewer by providing information even if it is untrue

Environmental considerations for children as witnesses

Skilled interviewing is essential

Because of their developmental immaturity, child witnesses face many disadvantages in the legal system, but these can be substantially offset by a skilled interviewer. Not only must an interviewer facilitate a child to say as much as he or she can remember, but the interview must be conducted so that its process and content will be considered to be acceptable evidence by the court.[5] Psychological research guides the practice of interviewing child witnesses. Some principles are outlined in Table 9.4.1.

Helpful adjustments to legal standards and courtroom process

Many jurisdictions have now made allowances for the developmental and emotional needs of children appearing in court as witnesses. Some legal systems have **relaxed their rules of evidence** where children are concerned so that hearsay evidence (in other words, reports from other people about what a child said or did) may be admissible and lawyers may be allowed to ask child witnesses leading questions. The detrimental effect of extreme stress on children's abilities to give accurate and credible evidence in court is well recognized. A variety of actions have therefore been taken by courts to make a child witness's appearance in court less stressful. The **courtroom may be rearranged to be less formal** and professionals may not wear gowns or wigs. The public may be excluded from courtrooms. Means of **preventing the child from having to face the accused** such as the use of screens or a live video-link to the child in a separate room may be used. Although many professionals agree that children generally find giving evidence by video-link less stressful than giving their entire evidence in open court, there is concern that the child's testimony may have less impact on a jury when viewed on video.[6]

Child witnesses need extra support in the courtroom

There is no doubt that **children find appearing in court a stressful event**. Typical concerns of child witnesses are shown in Table 9.4.2. Feelings of anxiety, confusion, humiliation, embarrassment, and the fear of retaliation or of not being believed

Table 9.4.2 Children giving evidence are often concerned about[7]

- People shouting at them in court
- Not understanding the questions
- Not being believed
- Giving 'wrong' answers
- Speaking in front of strangers
- Crying while giving evidence
- Needing to go to the toilet

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are common. The stress of appearing in court may be a further trauma for a child who was first traumatized by witnessing or experiencing the alleged crime in question. Some children report that although giving evidence was stressful, they also derived some satisfaction from playing their part in bringing a perpetrator to justice.

Anxiety created by the unfamiliarity of the surroundings and a lack of knowledge about what is happening in the courtroom can be addressed by **preparing child witnesses for their appearance in court**. Preparation often involves visiting the courthouse, receiving age-appropriate written information about the court process, and having the opportunity to ask questions. Ideally, the professional who prepares the child should be available to attend court with the child on the day of the trial. Some helpful information for child witnesses is shown in Table 9.4.3.

Receiving therapy prior to being a witness

Many child witnesses have been traumatized by witnessing or experiencing the alleged crime to which they will testify. Some of these children will develop emotional or behavioural problems as a result of the trauma and will be referred to child and adolescent mental health services. Treatment of trauma-related symptoms usually involves recounting the past experiences in a therapeutic setting. At this point **a conflict of interests arises between the needs of the child as a patient and as a witness**. The psychotherapeutic treatment of traumatized children centres on eliciting the child's subjective truth by the therapist using a variety of means to encourage communication. Within the legal system, the child's recollection of events must be examined in a neutral setting to determine the objective truth, while the rights of both the accused and the witness are protected. Therapy is seen as potentially detrimental to child witnesses because of the potential for their recollections to be altered by repetition or suggestion. The very fact that a child needs psychiatric help may be used to discredit the child as a witness in the eyes of the jury.

If a child witness clearly requires psychological or psychiatric treatment and cannot wait for many months until the trial is over for the treatment to begin, mental health professionals should discuss the child's needs with a representative of the legal service. **The therapy may be allowed to proceed with some restrictions about what can be discussed**. The therapist should be prepared to be called to court themselves in order to give evidence about the nature of their work. **The court will want to be satisfied that the child has not been coached by the therapist or told about information given by other witnesses**.

Table 9.4.3 A child appearing as a witness in court should know that[7]

- They have not done anything wrong
- They should always tell the truth
- They can take time to answer a question
- They should speak to the judge as clearly as possible
- Its OK to answer a question by saying 'I don't know' or 'I don't remember'
- Its OK to answer a question by saying 'I don't understand'
- They must not guess or make up an answer

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Children's testimony is worth hearing

The status of child witnesses has improved considerably in the last 20 years. Modern psychological research shows that although most children under 3 years of age lack the cognitive capacities to be competent witnesses, many older children are able to produce useful evidential information provided they are questioned competently. To make full use of what children can remember, they need to be allowed to talk in a comfortable setting, guided by professionals who are sensitive to developmental issues and aware of legal constraints. Child witnesses have often been traumatized by their experience. It is important that further distress caused by appearing in court is kept to a minimum and that children who need therapeutic help to deal with their trauma are not denied access to this in the pre-trial period.

Further information

www.childrenslegalcentre.com
www.victimsupport.org.uk
http://www.homeoffice.gov.uk/justice/what-happens-at-court/being-a-witness
http://www.homeoffice.gov.uk/documents/achieving-best-evidence/guidance-witnesses.pdf

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9.5

Treatment methods for children and adolescents

Contents

9.5.1 Counselling and psychotherapy for children

John B. Pearce

Introduction

There is a remarkable lack of high quality research to support an evidence base for counselling and psychotherapy for children. And the words 'psychotherapy' and 'counselling' are so non-specific that they should always be clarified in more detail. Nevertheless, these approaches are used frequently in child mental health. While most psychotherapeutic approaches are based on work with adults it is important to note that there are marked differences between children and adults. In spite of these obvious differences, psychotherapy for children is usually based on techniques used for adults. However, psychotherapy that may work perfectly well for adults has to be modified to accord with the developmental level of each child.

Definitions

We each have a mental image of 'a child'. Often this is a stereotypical child aged about 5 to 10 years old. But the word 'childhood' covers the whole period from birth to adulthood, and of course every adult is also somebody's child. In this chapter **the term 'child' will be used to refer to anyone who is not an adult**, but who has matured sufficiently to develop a clear concept of themselves as individuals and of the nature of the real world around them. The ability to distinguish fact from fantasy is an important prerequisite for psychotherapy. This develops as a gradual process with an important stage at around 2.5 years of age when children normally start to refer to themselves as 'I' for the first time. Another stage occurs around 7 to 8 years of age when children develop a clear understanding of time and of the real world. If the therapist ignores these developmental issues it is likely that treatment will be harmful rather than helpful.

Psychotherapy is a very general term that implies treatment of mental dysfunction by psychological rather than physical methods. The aim is to improve function by changing cognition and emotions through the therapeutic relationship, by means of language, play, art, or drama. Dynamic child psychotherapy can be defined as a highly specialized technique where the primary aim is to explore a child's conscious and unconscious thoughts, feelings, and conflicts in such a way that inner resources become strengthened and enabled. It is child-led so that the child is able to follow and explore his or her own agenda, thus helping the child to make sense of the world and to find his or her own solutions to problems and dilemmas. Therapy is mediated by language, which can either be verbal or non-verbal and may use play or creative activities such as drawing, painting, and modelling. Counselling children is very similar, but the therapist usually takes a more passive role than in psychotherapy and would not be so concerned with the interpretation of

unconscious processes. Cognitive behaviour therapy on the other hand is a highly structured approach focused on challenging false cognitions in order to change behaviour and emotions. It is an approach that can be rather easily adapted to children and made sufficiently enjoyable to engage their interest and cooperation.

Differences from adult psychotherapy

A number of **interesting paradoxes and dilemmas** occur when treating children with psychotherapy (Table 9.5.1.1). For example, who should give consent for treatment. Should it be the child, a parent, both parents, or all three? Clearly, this depends on the age and understanding of the child, and each case should be approached in a way that puts the child's needs first. It is generally best to obtain consent from the child and both parents. Any other arrangement is likely to lead to problems at some stage. Psychotherapy and counselling are traditionally non-directive and patient-led, but children, unlike most adults, need to be given some direction otherwise they become easily lost and confused. They cannot be expected to find their own solutions without guidance and support. Most psychotherapeutic approaches for adults are based on coming to terms with and finding explanations for problems that are rooted in the past. However, children are still busy making their past, and their main focus of concern and interest is the present and the immediate future. A further dilemma in child psychotherapy concerns the management of the transference relationship between therapist and patient, which is a reflection of the parent–child relationship. A high degree of trust has to be established to use transference effectively. At the same time, it could be argued that it is not really appropriate for young children to develop high levels of trust and dependence on a therapist whom they only meet briefly in very artificial circumstances. Thus any interpretation of the transference relationship in child psychotherapy must be done carefully and with a good understanding of the subtle complexities of a child's dependency on the parent.

Adult psychotherapy is usually based on a single theoretical model that explains mental mechanisms. Children, however, benefit from the freedom to experiment with a number of different models of their inner world and to learn how to use these ideas in a flexible and constructive way. The use of a single-theory therapy in child psychotherapy is best avoided.

Counselling and psychotherapy

There are undoubtedly differences between child psychotherapy and counselling, but they are difficult to define precisely. This may be because there is a continuum of therapeutic interventions, from advice and guidance at one end of a therapeutic spectrum through more specific counselling and psychotherapeutic techniques to intensive child psychoanalysis at the other end. Counselling children requires a high level of skill, but less theory and technique than in psychotherapy, and it is focused primarily on normal reactions to abnormal events.

Psychotherapy is directed more at psychopathology than normal reactions to stress. It is therefore essential to know about the normal range of children's responses to life events. For example, a 5-year-old child whose mother has just died will grieve differently from a 10-year-old child, because at 5 years of age most children have not yet developed a clear concept of death. Grief in a 5-year-old is most strongly influenced by the way the adults around the child react to the death, whereas a grieving 10-year-old child, although responsive to guidance from the adults around, will also have his or her own unique way of coping with grief. As a general rule, the younger the child the more important it is to consider the attitude and mental state of the parents.

Table 9.5.1.1 Differences between children and adults in relation to therapy

Consent for treatment usually given by parent/carer rather than child
Child may nor understand or want therapy
Children need more help with problem solving than adults
Therapy should be relatively more directive and instructive
Problems tend to be rooted in present or recent past
Therapy should be mostly enjoyable for child to benefit
Transference is complicated because children live with and need parents/carers
Different approaches needed at various stages of development
Children are developing and changing quickly so therapy needs to do this too

Other psychotherapies

Dynamic psychotherapy based on the theories of Sigmund Freud and his daughter Anna Freud,[1] Melanie Klein,[2] and others has been the mainstay of individual child therapy. More recently, Virginia Axline[3] adapted the ideas that Carl Rogers[4] applied to counselling (trust, genuineness and understanding) and developed 'play therapy' as a specific technique for children. Subsequently, brief psychotherapy and interpersonal therapy have grown out of the need to update psychodynamic methods. Various forms of cognitive therapy are now increasingly used for children, although they were originally developed for the treatment of adults by Beck.[5] These therapies focus on a problem-solving approach to resolve current issues, rather than on resolving unconscious conflicts based in the past.

Natural emotional healing

Counselling and psychotherapy have been used with increasing frequency to help children cope with traumatic events such as death, divorce, abuse, illness, and so on. It is arguable whether this trend is at all helpful. Fortunately, the human psyche is remarkably resilient and there are powerful healing processes that take time, which in most cases achieve a satisfactory result. There are similarities between the way the body and mind respond to trauma and a strong correspondence between the natural healing processes that accompany both physical and emotional trauma. The initial healing process starts with a brief period where no pain or distress is felt whatever the cause of the trauma, and this is often accompanied by disbelief that such a thing could have happened. This first phase of shock and 'denial' is then replaced by the full impact of what has happened and is accompanied by high levels of physical or emotional pain. During the second phase the pain may be so severe that it interferes with everyday life, but this stage is usually over within 2 weeks. In the third stage, the healing process continues for a period of up to 6 weeks when the emotional or physical wound is normally healed sufficiently for the traumatized person to be able to return to everyday life, albeit with continuing pain and discomfort at times. The final phase of the healing process then continues over the next 6 to 12 months, leaving a scar that will always remain.

Routine counselling following traumatic events carries the risk of interfering with this normal healing process. Psychotherapy could also be misused to check that all is well, rather like opening up a wound unnecessarily, which will only serve to delay the healing process and might even introduce a secondary 'infection'. The parallel between physical and emotional healing provides some guidelines as to when and how counselling and psychotherapy should be used, as well as the dangers that can occur when they are misused.

The use of play in therapy

The fact that **children are still developing language and communication skills** means that play is often useful as a method of communicating in a therapeutic way with children. Play is essential for normal development. It helps children to develop a repertoire of responses and encourages behavioural flexibility. The main developmental stages of play need to be appreciated if it is to be used in an effective way in either counselling or psychotherapy (see Table 9.5.1.2).

Children will play with almost anything, and so the choice of play materials for use in psychotherapy requires careful thought. It is best to select toys that encourage imagination and creativity. It is important to remember that the type of play equipment provided will actually constrain the way the child plays. For example, there is a limit to what cars or toy animals can do. Similarly, a set of family dolls may have their usefulness restricted if there are not enough of them to represent all the key figures in the child's life. A thoughtful and appropriate choice of toys and play materials including drawing, modelling, and painting equipment can make the difference between the success and failure to engage a child in therapy.

Children's play can be a pointer to what is going on in the child's mind (either conscious or unconscious), but it can also mislead. A child's play will only give a very general indication of what the child thinks and feels and will not provide precise information. Perhaps the best way of viewing the use of play in therapy is that it is an aid to communication—and no more than that.

The treatment setting

There are a number of steps that can be taken to make the treatment setting more beneficial for the child and help to create a relaxed atmosphere. How the therapy and the therapist are introduced to the child is of some importance. The attitude of the therapist is undoubtedly more important than the way the therapy room is organized and the more anxious and tentative the introduction, the more uneasy the child will be. There are no hard and fast rules about the duration or frequency of the treatment session. It is best to arrange it to suit the child. Some children find more than 20 min with an adult extremely difficult to cope with whatever their age. Many children find the formal structure of therapy quite stressful and it may help to put the child at ease by talking about unimportant issues before the session itself begins. Anxiety can also be reduced by organizing the sessions to be as predictable as possible in place and time. Making it clear to children that the therapeutic time is specially for them and that they are the focus of all their therapist's attention can also motivate children to be more co-operative and to be less wary.

Table 9.5.1.2 The development of children's play

Age	Type of play
12–18 months	Symbolic play (e.g. block of wood is a car)
18 months–3 years	Imaginary play (e.g. blocks become a family)
3–7 years	Imaginary friends in 30 per cent of children
5–7 years	Rule-governed make-believe games
7 years onwards	Imagination founded much more strongly on the real world

The treatment process

Each child's experiences of distress must always be considered in the context of the child's family circumstances, the child's stage of development, and his or her temperamental characteristics. It follows that the therapeutic approach needs to be individually tailored and adjusted for each child. Nevertheless, it is possible to arrive at some general guidelines that will assist in treatment. The concern, interest, and supportive attitude of the therapist is central to the treatment process, since it is the interaction between the child's need and the adult's response that establishes the transference relationship.

The start of the first session is particularly important because it sets the tone for the future treatment. The child should know the role that the therapist has and what the aims of the treatment are. Something needs to be said about the limits to behaviour in the session and the nature and degree of confidentiality. It is important to remember that complete confidentiality cannot be assured. For example, the therapist may be presented with information that concerns the health or safety of the child as in the case of abuse, where information has to be disclosed for the benefit of the child.

The timing and number of sessions needs to be agreed at this stage. Therefore it is often a good idea to suggest a limited number of sessions in the first instance, together with an agreement to review whether or not further sessions are required. This type of introduction helps children to feel their needs are being taken seriously, especially if they are actively involved in the process. Sessions should start and finish on time and the links between the present and any previous sessions should be clarified.

Most children will remember their treatment experience for many years to come—if not for the rest of their lives. The child who feels uncomfortable, embarrassed, and misunderstood is likely to retain a memory that is painful and unhelpful. On the other hand, if the therapeutic experience was positive, where the therapist was seen as supportive, encouraging, and understanding, the memory is likely to be one that the child returns to again and again for emotional strength and support.

Different approaches to psychotherapy

There is a wide variety of jargon associated with various types of psychotherapy. Each approach has its own 'language' and associated special techniques. However, there is no evidence that any one method is better than another, and it would appear that the personal preference of the professional involved is more important in determining which approach is used rather than the characteristics

of the child. Whichever approach is used it is likely that there will be the same common themes in the focus of treatment. Common themes include dealing with feelings of anxiety and insecurity, difficulties in relationships, low self-esteem, and a feeling of failure. These emotions are often generated by difficulties with aggression, jealousy, sexuality, and death.

A focus on the past versus the present

The child comes to psychotherapy with a range of problems rooted in the past. A decision has to be made whether to focus the therapy on trying to understand and come to terms with the past, or to consider how a child might best cope with what is actually happening in the here and now. **The danger** in concentrating primarily on the past is that it may interfere with the child's ability to cope with the present and plan for the future. While it is important to learn from what has happened in the past, children tend to learn more from what is happening in the present in their daily lives. Understanding how their emotional stress was generated in the first place may not lead to a resolution or to a greater ability to deal with current problems. It is generally helpful to start therapy with an acknowledgement of what has happened in the past and a consideration of how that might affect what is happening in the present. In a few cases it may be helpful to focus more on the past, but only if the child has clearly become preoccupied with a particular issue from the past and is unable to move on. Normal development moves on so rapidly during childhood that any fixation with the past can have serious consequences, thus every effort needs to be made to promote and sustain developmental progress.

Theory vs. common humanity

While it is undoubtedly helpful to have a theoretical framework within which treatment can take place, the observation that different psychotherapeutic approaches for the same type of problem can be equally effective suggests that the precise theoretical framework underpinning treatment may not be that important. The basic human qualities of kindness, trust, caring and understanding are perhaps the most important qualities in psychotherapy. It is not an unusual experience for therapists to find that their early cases turn out to be the most successful, which is probably due to the enthusiasm and therapeutic optimism of the new therapist. It is clearly important to hold on to these therapeutic qualities as one becomes more experienced.

Supportive counselling vs. in-depth psychoanalysis

It is easy to assume that the more intensive the psychotherapy and the more it explores the deep unconscious world, the more effective it must be. Clearly there is no reason why this should be the case. For example, one would not expect a surgeon to cut deeper for greater effect or a physician to prescribe more medication than is necessary. This would only increase the adverse effects of the treatment. It is not difficult to see that regular psychoanalysis two or three times per week could be quite disruptive to family life merely as a result of the time commitment alone. There are also other potential problems for children who are treated with intensive psychoanalysis over a period of years, as this may delay or shape a child's development in an unhelpful way. On the other hand, it might be equally inappropriate to commence supportive counselling for a child who is deeply disturbed and whose need for loving care and protection is not being met. These primary and basic needs must always be given priority.

Cognitive therapy vs. psychoanalytically based psychotherapies

There has been an increased interest in cognitive therapy and cognitive behaviour therapy, where the emphasis is much more on the here and now and the behavioural consequences of abnormal thought patterns. The techniques used in cognitive therapy for anxiety and depressive disorder are described in Chapters 6.3.2.1 and 6.3.2.3 respectively. The only modification that is required for their use in children is to adapt them to the developmental stage and the level of cognitive ability that the child has reached. Cognitive therapy has a theoretical advantage for use in children in that its focus is more on the present and the future, in contrast to most psychoanalytically based psychotherapy. Its approach is strongly based on learning new ways of coping. Cognitive therapy is pragmatic and active rather than passive and reflective, making it generally more appropriate for the needs of younger children. Unfortunately, there is as yet limited evidence to support the theoretical underpinning of the various cognitive models of childhood disorders.

Limit setting vs. free expression

It is a common dilemma to know how much freedom children should be allowed to express themselves. Some children appear to enjoy pushing the limits to see how far they can go. Other children appear too inhibited and need encouragement to express themselves. Part of the art of child therapy is to strike a comfortable balance between control and freedom. Children gain nothing from disruptive and destructive behaviour, even if they normally tend to be quiet and inhibited. Indeed, they rapidly develop overwhelming feelings of anxiety and insecurity if they do not feel sufficiently contained. It is essential that the therapist retains a very clear notion of what behaviour is acceptable and what is not. It is therefore the therapist's responsibility to set the scene and establish the boundaries of acceptable behaviour within the therapeutic context. Should the child go beyond the limit then a warning should be given, and if the child persists it is quite acceptable to end the session early or at least until the disruptive behaviour has stopped. The therapist's reaction to bad behaviour should make it quite clear that it is unacceptable. However, the emotional response should be neutral or sad, in much the same way that one might behave in a shoe shop when a desirable new shoe does not fit as expected.

Closeness vs. distance

It is natural for an adult to be physically much closer to younger children and then to become more distant as they grow older. For example, it is quite natural for an adult to hold the hand of a 3- or 4-year old and to physically guide the child. On the other hand, any physical contact with a teenager can easily be misconstrued and is likely to be most unwelcome. In addition to the developmental perspective, every child has its own preferred degree of closeness or distance from other people. The task of the therapist is to judge what is right for each occasion. It is absolutely essential that the therapist must always avoid intruding into the child's space in any way that could be construed as abusive. This may prove difficult where children are unsure of their boundaries and seek out physical contact (in cases of sexual abuse, children may seek out

sexual contact). However, to maintain an artificial physical or emotional distance can be perceived as disinterest or even rejection by some children. To achieve a comfortable level of emotional warmth and physical closeness in therapy is obviously a very important matter, but a relaxed approach in the therapeutic relationship is generally best.

Individual vs. group therapy

There are no agreed guidelines to determine which child would benefit from an individual or from a group approach to psychotherapy. Some children, however, find the emotional intensity of undivided adult attention too much to cope with and learn better from others in a group situation. As the selection of cases for group psychotherapy tends to be determined by the therapist's skills, there is no clear evidence that one approach is better than any other. Nevertheless, there is a growing literature on group therapy for children and an increasing interest in this method of treatment if only because the cost per case is likely to be less.

Practical issues in child psychotherapy

Involving parents and the school

The younger the child, the more helpful it is to involve the parents in treatment. How this is managed will depend to some extent on the resources available. It is generally best for separate therapists to work with the child and with the parents. However, there will usually be occasions when it is helpful for the child's therapist to have some direct contact with the parents as a way of monitoring progress and keeping a link between the therapy sessions and the child's real world. As children grow older they tend to become increasingly inhibited by the involvement of their parents, so it is helpful to check with the young person how they would like the contact with their parents to be organized. All children should be considered in the context of the family and the school because so many risk factors are associated with these environments. Parental support for the treatment is a critical factor in a successful outcome. Equally, it is possible for parents to undermine treatment by their negative comments or overintrusiveness after each session.

The extent to which a child's school should be involved needs to be carefully considered on an individual basis. Although it is best to have the full co-operation of the school, this may not be possible.

Confidentiality and record-keeping

Confidentiality in relation to the individual therapy sessions is clearly important, but it should not dominate. The most vital issue is that the children feel safe in what they say and do during the session. They need to know that there is a reasonable level of confidentiality. However, there are obvious exceptions to the general rule of confidentiality: for example, in cases of child abuse or criminal activity, or in cases where the child may be at risk from harming itself. A suitable compromise is to indicate to the child that their session would be treated as confidential, but there may be occasions when it is best to inform their parents or somebody else about important information that was given during the session. This would only occur after discussion between the therapist and the child—the overarching rule is that it must be in the best interest of the child.

There is obviously a need for accurate record-keeping for each session, partly to keep track of the therapeutic process and also for medico-legal purposes. It is unnecessary to include the more detailed recording of the therapeutic process in the official notes if the record is purely for training purposes. These training notes then remain the property of the therapist and must be kept at the same level of security as the official notes.

Failure to attend

If there is a genuine and legitimate reason why a child is unable to attend for a therapy session then little or no importance needs be attached to this. On the other hand, if therapy sessions are cancelled repeatedly, cancelled with inadequate excuses, or simply not attended it is essential for the therapist to question whether therapy is actually achieving the original stated goal. Factors such as family disadvantage, parental stress, and the severity of the child's problems all increase the likelihood of failing to complete therapy successfully. It is all too easy to either blame the carers or to rationalize failure to attend as being some problem in the child, rather than an issue that could be due to the therapist. Of course, one good reason for failure to attend is that the child no longer needs to. It is usually unhelpful to send out repeated appointments that are not attended. A reasonable compromise is for one reappointment to be made, then if this is failed for a letter to be sent to the carers or to an older teenager asking them to contact you if further sessions are thought necessary. Occasionally, if treatment is at a very critical stage or if there is high level of concern about the child, it would then be appropriate for more effort to be put into arranging a further appointment.

Individual and cultural issues

A child's sociocultural background needs to be taken into account when planning therapy. Temperament, age, gender, and intellectual ability are also important factors to be considered. Ethnicity, on the other hand, is probably not that significant an issue. Some children enjoy, and benefit from, the freedom to express themselves openly and with little constraint; others need direction and structure if they are to resolve psychological problems. There is no point in trying to fit the child to the therapy rather than the other way round. One factor that supersedes all cultural and individual factors is that children generally wish to enjoy themselves and to have a good time. They are pleasure-seeking beings who, unlike most adults, see no benefit from the experience of pain and distress of therapy.

Ending therapy

Bringing therapy to a satisfactory conclusion is more likely to happen if the treatment was well set up in the first place and if achievable goals were agreed. Because it is so easy for therapy to lose its focus, it is helpful to consider how and when the treatment will be concluded at the same time as setting it up. Even if treatment goals have not been achieved it should still be possible to end the therapy on a positive note, identifying areas where self-knowledge has been increased and anything that has been positive in the therapeutic relationship.

Training and supervision

The objectives of training in psychotherapy are primarily to do with increasing knowledge and understanding of the issues that arise during psychotherapeutic treatment, many of which have been referred to above. It is essential for this to be underpinned by a sound knowledge of child development, but less important for

therapy to be founded in any particular psychodynamic theory. The key training method in psychotherapy is the conduct of therapy under supervision. Choosing a supervisor and the role of that supervisor are critically important issues for the trainee therapist, since the relationship between supervisor and trainee mirrors that of the therapist and client even if there is a clear understanding that the purpose of the supervision is not intended to be therapeutic. Most therapists find that supervision continues to be helpful even after they are 'trained'.

Measures of effectiveness and outcome

There is a lack of high quality research to support an evidence base for counselling and psychotherapy for children. One of the main difficulties in conducting research is in the selection of an adequate control group. Reviews of psychotherapeutic treatments indicate a general improvement in children that persists for many months after the intervention. However, it seems likely that the theoretical basis for the psychotherapeutic approach is less important than the caring and supportive relationship that develops between the child and the therapist.

Further information

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Useful web sites

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www.psychnet-uk.com
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9.5.2 Psychodynamic child psychotherapy

Peter Fonagy and Mary Target

Introduction

Psychodynamic psychotherapy for children is based on a range of assumptions concerning mental functioning that have gradually evolved over the past 100 years out of the theories of Sigmund Freud. As these assumptions have been widely reviewed, we need to provide only a very brief introduction here.

Psychodynamic child clinicians assume that:

(a) The child's presenting difficulties may usefully be seen in terms of thoughts, feelings, wishes, beliefs, and conflicts. This entails the assumption that mental disorders can meaningfully be understood as specific organizations of a child's conscious or unconscious mental states.

(b) To understand conscious experiences, we need to consider non-conscious narrative-like experiences, analogous to conscious fantasies, which powerfully affect behaviour, affect regulation, and the capacity to handle the social environment. Modern neuroscience, with fMRI studies that show cortical response reflecting processing to meaning in the absence of awareness and non-conscious motivation,[1] has put the existence of an unconscious beyond debate.

(c) Intense relationship experiences are represented in the mind as structures of interpersonal interaction, and are aggregated across time before coming to form a schematic mental structure, which is often represented metaphorically as a neural network. Within many models, self-other relationship representations are also considered the organizers of emotion, as feeling states are seen as coming to characterize particular patterns of self-other and interpersonal relating (e.g. sadness and disappointment at the anticipated loss of a person).[2]

(d) Inevitably, wishes, affects and ideas will at times be in conflict with one another. The psychodynamic therapeutic approach sees such conflicts as key causes of distress and the lack of a sense of safety. Adverse environments either increase the intensity of conflict or fail to equip the child with the capacity to resolve such incompatibilities through mental work.[3] They may also set the child on a developmental trajectory in which the normal development of key psychological capacities is undermined, thereby reducing the child's competence to resolve mental conflict.[4] For this reason, while reviewers of psychodynamic psychotherapy often contrast conflict and development-focussed approaches, the reality of developmental trajectories means that the conflict and deficit often come together.[5]

(e) The child's mental mechanisms for dealing with intrapsychic conflict include defence mechanisms that distort mental representations in order to reduce conflict and unpleasure.[6] Such self-serving distortions of mental states relative to an external or internal reality are frequently demonstrated experimentally and have become accepted.[7–10] Classification of defences has frequently been attempted,[11,12] often as a method for categorizing individuals or mental disorders,[13,14] but few of these approaches have stood the test of time or achieved general acceptance.

(f) Behaviour may be understood in terms of 'complex meanings', that is, mental states that are not explicit in action or within the awareness of the person concerned. Thus, symptoms of disorders are classically considered as condensations of conflicting wishes together with the failed defence against conscious awareness of those wishes. Therapy is an effort to seek *personal* meaning,[15] and to elaborate and clarify implicit meaning structures—a process that may turn out to be

the essence of psychodynamic psychotherapy—rather than to give the patient insight in terms of any particular meaning structure.

(g) A relationship with a supportive and respectful empathic adult will benefit the young person, not least by enhancing their own understanding and emotional responsiveness. The nature of the relationship with psychodynamic therapists varies across therapies—from the highly transferential and fantasy oriented[17] to the quite practical and supportive,[18] although most therapies contain elements of both.[19] Establishing an attachment relationship with a clinician (i.e. with an interested, understanding, and respectful adult) may be a new experience for some young people[20] and is believed to trigger a basic set of human capacities for relatedness that appears therapeutic, apparently almost regardless of content.[21–27] The child's relationship with the therapist often appears to become the vehicle for disowned aspects of the child's thoughts and feelings, creating a process termed transference, which enables the psychoanalytic clinician to understand the child's representation of relationships and his or her feelings about them.[28]

Background

The roots of child psychoanalysis lie in Freud's observation of young children, most notably of the young Anna Freud's wishful dream for strawberries,[29] his grandson's separation game,[30] and his case study of Little Hans, a 5-year old with a phobic disorder who was treated by his physician father under Freud's supervision.[31]

Play therapy, incorporating both an insight-oriented interpretive approach and the developmental assistance perspective, was introduced by Hermine Hug-Helmuth.[32] Thereafter, two women, in strong opposition but frequently making reference to each other, established the field: Anna Freud[33] and Melanie Klein.[34]

Klein's approach was to regard children's play as essentially the same as free association with adults; that is, motivated by unconscious fantasy and activated by the relationship with the therapist (transference). The child's anxiety required verbalization (interpretation) if it was to be addressed. The focal point of therapy was the verbalization of anxieties concerning destructive and sadistic impulses, whilst the child's external relationships (with parents, teachers, etc.) were seen as peripheral and irrelevant.

A key construct was the notion of projective identification.[35] This term referred originally to the infantile tendency to project unwanted aspects of the self on to another person. The clinician, by understanding the child's perception of her as a person, could gain valuable insights about conflictual aspects of the child's experience of himself. Bion[36] described how the 'container's' capacity to understand and accept the projections could be critical both to successful therapy and to normal development. More recently, Kleinian child analysts have been less likely to offer early interpretations of deeply unconscious material; defences beyond projective identification are more commonly considered.[17,37]

Strongly influenced by Melanie Klein, Donald Winnicott firmly endorsed her emphasis on the impact of the first years of life on childhood psychopathology.[3] However, he also introduced new techniques (e.g. drawing) and various theoretical innovations, including the identification of a transitional space between self and other where the subjective object and the truly objective object could simultaneously be recognized.[38] The notion of transitional space, an intermediate area between the intrapsychic and the interpersonal, was critical to the development of an interpersonal[39,40] and intersubjectivist[41] approach within psychoanalysis.

Along this continuum, Anna Freud was perhaps most concerned with the child's developmental struggle with a social as well as an internal environment. Her background as a teacher may have led her to be as concerned with children's actual external circumstances as with their unconscious worlds.[42] Her focus was restricted to complications and conflicts arising from the child's libidinal impulses and, unlike Melanie Klein, she rarely focussed on innate aggression. The interpretation of defence was central to her technique.[43] Her approach paid careful attention to limitations on the child's cognitive capacities (ego functioning) and had as its explicit aim the restoration of the child to a normal developmental path.[44] Her concern with normal development led her to evolve a model of pathology as a disturbance of normal developmental processes, and she developed a systematic analysis of such anomalies using the concept of developmental lines.[45] Her propositions are in many respects consistent with modern developmental psychopathology.[46] Developmental help is aimed at facilitating the forward movement of the psychological processes that underpin social cognition and interpersonal function, and which include mentalization, impulse control and emotion regulation, symbolization and the use of metaphor, and the capacity for play.[47,48] Notwithstanding the curious historical fact that Anna Freudian, Kleinian, and Winnicottian approaches all originated in London, the Anna Freudian approach came to dominate child therapy in the United States,[49] whereas in the United Kingdom and in Latin America Melanie Klein's approach proved more popular.[50] Two comprehensive and detailed histories of the field have been provided.[51,52]

Techniques

Techniques of child therapy differ considerably depending on the degree of pathology manifested by the child. Two sets of technique may be distinguished: those with single diagnosis, usually involving anxiety, are offered what most would recognize as 'classical' forms of psychodynamic, insight-oriented therapy. Those with multiple diagnoses, severe behavioural problems and/or emergent personality disorders[53] require a different psychodynamic treatment approach. These will be discussed separately.

Principal features of 'classical' technique

Child psychotherapy involves the elaboration of distorted and, to a lesser or greater extent, non-conscious mental representations. The therapist, using the child's verbalizations, non-verbal play, and other behaviours, aims to provide a rational understanding of the child's non-conscious thoughts, feelings, and expectations. This understanding may encompass and integrate earlier modes of the child's thinking into a more mature, age-appropriate framework.[54] With young children, the treatment involves the use of toys, play, and any device that helps to engage the children in a process of self-exploration. The therapist works to elaborate the children's understanding of their emotional responses, their unconscious concerns about their body, and the way their symptoms might link together anxieties about relationships, including non-conscious aggressive or sexual thoughts and other conflictual feelings in relation to the parents, siblings, and peers.

The techniques used by the child therapist go beyond interpretive interventions and were usefully enumerated by Paulina Kernberg.[55] She delineated: (i) *supportive interventions*, which are aimed at addressing the child's anxiety and increasing the child's sense of competence and mastery through the provision of information, reassurance, empathy, and suggestions (ii) *facilitative statements*, which seek chiefly to maintain the therapeutic relationship with the child by reviewing, summarizing or paraphrasing the child's communications and (iii) *clarifications*, which review and summarize the child's communication, and which usually involve relabelling communication or behaviour. Clarifications also serve to focus the child's attention on certain patterns in his behaviour indicative of unconscious determination.

Interpretation may centre on: (i) the content of the child's communications (ii) the contents that the child systematically omits from verbalization (iii) the child's non-verbal behaviour (iv) the nature of the child's play, including the roles that he or she tends to assign to himself and to the therapist (v) the child's current emotional state, particularly sadness, anxiety, or guilt, and; (vi) dreams that the child recounts in his sessions. While the therapist may be able to link the child's therapeutic material to past experiences with attachment figures, such reconstructive interpretations are rare in child therapy. It is only gradually that the therapist hopes to be able to generate an emotionally meaningful understanding of the impact of past experiences on current anxiety and conflict.

Paulina Kernberg[55] distinguished between three types of child therapeutic interpretation. First and most common are interpretations of defences, which aim to show the child how it protects itself from thoughts, feelings, and actions that it considers unacceptable. For example, the therapist may draw the child's attention to repeated examples of self-denigration, and hint at his anxiety about being thought boastful; this serves a dual function in both bringing to the child's awareness what he is protecting himself from and also in prompting him to find alternative strategies to cope with warded off ideas. Second are interpretations that address the child's unconscious wishes, which are themselves thought to underpin behaviour. Frequently, these interpretations are made following interpretations of defences. Finally, child therapists might address the child's past experiences. The therapy may reveal traumatic experiences, and some therapists consider it helpful to bring these memories into consciousness. It should be noted that current psychodynamic theory in no way assumes that addressing such trauma directly is essential to cure. Far more important in terms of therapeutic progress is addressing the distorted relationship representations that are sequelae to early trauma.[56]

Whatever the interpretation, the child therapist aims to address the child's anxiety and how other emotions relate to it. Thus, destructive wishes would most likely be taken up in connection with the child's anxiety about his or her angry feelings. Child therapeutic technique also demands that the child's attempt actively to struggle with these wishes be clearly acknowledged. Interpretations are ideally tied to a highly specific context, such as the child's experience of anxiety associated with his anger that an ungenerous but otherwise valued therapist will not give him a special treat for his birthday.

An important part of child therapeutic work involves the child's parents. Some of this work is psychoeducational; in particular, parents often need guidance on appropriate, uncritical, warm, and playful methods of child rearing. Discussion of the child's symptoms may enable parents to gain greater awareness of the child's difficulties and how their own representation of the child may be distorted.[57]

Psychodynamic technique with complex childhood disturbances

The child psychotherapeutic approach has been extended to apply not only to so-called neurotic disorders, but also to the understanding and treatment of borderline, narcissistic, delinquent, and conduct disordered youngsters, as well as schizoid and even psychotic children.[53] The classical psychoanalytic approach as outlined above has clear limitations with these children: anxiety may not be accessible; there may be little evidence of conflict/of the child's struggle with wishes; defences may be hard to identify, and the child may be developmentally inaccessible to insight. Taking these issues into consideration, we have suggested that a dramatic modification of child psychotherapeutic technique may be in order[19] based on what Anna Freud called 'developmental help'. We have described this intervention in detail,[58] and colleagues in the Netherlands have elaborated and researched this form of therapy.[59]

Essentially, the therapist begins by performing mental functions of which the child is incapable, or by showing the child ways of performing these functions until he or she can take over and do it himself. These interventions are used with pathologies traditionally defined as ego defects, deficiencies in relationships, or developmental disturbances—pathologies understood here as mental process disturbances. These techniques have sometimes been labelled remedial education or ego-support, but, broadly, the therapist's aim is to free the mental processes from inhibition and to aid in the development of these processes. The therapist achieves this by: (i) providing a safe place and relationship within which the child can dare to change or wish to be different (ii) making up for some deficits in the parenting that the child has received by providing him with the missing elements (iii) stimulating delayed or stunted developmental processes by drawing the child's attention to what is missing, and encouraging his interest and desire to function better and (iv) using interpretations not to uncover the source of his difficulties but to help the child understand the extent and impact of his problems, his contribution to his developmental difficulties, and to confront the role played by his environment. The main foci of these modified forms of child therapeutic intervention are six-fold:

(a) *The enhancing of reflective processes*; that is, the understanding of how mental states (beliefs, desires, wishes, and emotions) determine human behaviour. This is achieved by encouraging the observation and labelling of both physical and psychological experiences in the immediate situation. It is assumed that, regardless of cause, the final common pathway of most personality disorders is a dramatic impairment of reflective function.[60]

(b) *The enhancing of impulse control* by identifying and helping the child to exercise ways in which impulses may be channelled into socially acceptable forms of behaviour. The therapist may initially have to control this herself by 'setting limits'. She tells the child what he may and may not do during the sessions, explaining that she will not let him hurt her or himself, or damage things in the room.

(c) *Affect regulation* is an important aim of developmental work. It can be assisted by the verbalization and labelling of affect, and by explaining to the child possible reasons for his feelings; for example, that his aggressive attacks are reactions to his sense of being threatened and endangered.

(d) The elaboration of strategies involving symbolization and metaphor for *enhancing cognitive self-regulation*. The therapist demonstrates her own capacities for reflection and the moderation of experience through mental representation rather than physical action or coercion.

(e) Focussing the child's awareness on *the mental states of others*. This is achieved initially by focussing interventions around the child's perception of the therapist's mental states, which can be a precursor to reflective processes in relation to the self.

(f) Developing the child's *capacity for play*, initially with physical objects, then with another person and, ultimately, with ideas. Play is not simply the creation of a pretend world but has the aim of creating a safe opportunity for alternative meanings of the child's experiences to emerge. This can show the child how his habitual ways of thinking and feeling represent but one of multiple ways of construing reality. Perhaps more importantly in this context, he can experience adults relating to him, as perhaps they have not often related to him before.

While the child therapist working with such severely disturbed children is still 'working in the transference', in the sense that the child's feelings about the therapist remain central, this is no longer thought to entail the displacement of feelings and ideas from one person to another, e.g. from the parent to the therapist. Rather, the clarification of the child's feelings about the therapist may be the most effective route towards assisting the child to acquire a reflective capacity. In this way, the therapist conveys that the child's affect can be understood and managed by another person.

Indications, contraindications, and the selection of procedures

There is general agreement on the indications for child psychotherapy.[61] These have traditionally included: (i) high IQ and verbal ability (ii) supportive environment (iii) conflict-related pathology (iv) adequate internal and external object relations and (v) the presence of anxiety. Contraindications include: (i) pervasive developmental disorder (ii) psychosis (iii) major deficiencies in psychological capacities and (iv) family constellations incompatible with adherence to treatment, for example, chaotic home environments or psychologically severely disturbed parents.

As described above, however, by substantially modifying traditional technique, child therapists have successfully worked with populations beyond this restricted group,[62] treating children with a variety of psychological deficiencies.

One way of conceptualizing the difference between the needs of the two groups is by using the classical distinction between mental representations and mental processes in cognitive science.[63] Classical techniques primarily impact on the organization and shape of the child's mental representations of self-other relationships.[64] By contrast, developmental help for the more severely disordered group aims at developing the function of mental processes, which may have been defensively distorted in early development,[65] by strengthening and supporting the patient's adaptive defences and helping them to label and verbalize their thoughts and feelings.

The distinction between classical technique and developmental help is a heuristic one. In reality, all child therapeutic treatments involve both, but it is nevertheless suggested that developmental help is essential for the effective treatment of severe disturbances, whilst it remains an 'optional extra' for children with neurotic disturbances.

Managing treatment

Starting treatment

At the beginning of psychodynamic child therapy, the therapist's aim is to communicate that: (i) sessions have the purpose of expressing thoughts and feelings through play and words and (ii) the therapist is trying to help the child make sense of his experience so that he can master his inner turmoil in a more effective manner. Children are generally able to develop a therapeutic alliance in the context of an empathic, respectful, non-exploitative relationship with an adult. A similar collaboration is established with the parents, and early meetings also allow the child therapist to acquire relevant information and assess family interactional patterns that may be relevant to the child's treatment. In early sessions, the therapist attempts to interest the child in meaning and to link mental states to activity by using child's play to address the child's understanding of momentary anxieties, and to explain that his struggles, thoughts, and feelings may be explored through his verbalizations and behaviour.[66] Children who are able to use therapy tend to respond to these interventions with an enhancement of the therapeutic alliance, showing greater freedom in their play and verbalizations. It is important to note that the same process of relationship building characterizes other individual therapies such as cognitive behaviour therapy (CBT).[23]

The middle phase

The middle phase of child therapy is expected to focus on the systematic use of transference interpretations and the initiation of the 'working through' process. The development of the transference is facilitated by a therapeutic structure that emphasizes regularity, consistency, and the specialness of the hour of therapy.[67] The child's play is 'interpreted', but normally these interpretations are kept within the context of the play situation. Clinical experience suggests that interpreting the play in reference to the child's 'real' feeling only serves to disrupt the child's communication.[68]

The major themes in children's lives are often expressed in the therapeutic relationship. Internal working models of attachment relationships, for example, will come to colour the child's relationship with the therapist,[69] be that a pattern of reluctance in forming trusting relationships (avoidant/dismissing) or excessive anxieties about separation and sometimes angry preoccupation with the therapist's thoughts and feelings (resistant/preoccupied). This, of course, opens up the possibility of correcting distortions and mastering conflicts and associated anxieties. The child gains information (insight) in a relational context that elaborates his understanding of his experience, provides him with a way of coping with thoughts and feelings that make him feel uncomfortable and interrupts maladaptive interactions.[70] Progress is often slow, and regression and progression tend to alternate as children struggle to face these anxieties without undue repression. Yet, gradually, given a capacity for the symbolic transformation of experience, they are able to bring their experience of internal and external

worlds into play and can therefore internalize the therapeutic attitude to solving their problems. Of course, development is helpful too in this regard.[71]

The ending of treatment

Indications for the termination of long-term child psychotherapy are both external and internal to the therapy. External indications are not restricted to symptomatic improvement, but include considerations of changes in family interaction and peer relationships. Relational changes indicate a capacity to use caretakers, teachers, and others as sources of protection, guidance, comfort, and models of identification. In general, the therapist seeks evidence that the child has returned to the path of normal development, can cope effectively with stress and conflict, and can respond with greater freedom to adaptive demands coming both from within and without.

There are also indications from the child that the therapy may be ended. Some were enumerated by Paulina Kernberg[72] as follows: (i) the therapist finds more opportunities for interpretation than for confrontation (ii) the child manifests more reflectiveness and the capacity to make interpretations (iii) there is greater freedom, expressiveness, and pleasure in play (iv) there is insight and humour and (v) the child assumes responsibility for his own actions.

The end of psychodynamic therapy often generates anxiety and can bring a temporary reactivation of symptoms and the re-emergence of dysfunctional patterns of interaction within the family. As the therapy creates an attachment relationship, its termination may be an essential part of resolving attachment-related concerns (e.g. fear of abandonment, rejection). Mourning the anticipated loss of the therapy and the therapist may well be an essential part of a successful ending to treatment. Other issues may involve children's disappointments—with themselves, with the unfulfilled promise of the therapy, and with adults who do not measure up to their expectations. Research and clinical experience suggests, however, that as this turmoil subsides, progress continues to be made after the end of treatment.[57,73,74]

Efficacy

The most comprehensive survey of outcome studies specifically concerned with psychodynamic treatment was undertaken by Kennedy[75] as part of a project sponsored by the British Association of Child Psychotherapists—although there are other comprehensive reviews of treatment of children[76–80] and of psychosocial interventions limited to 'evidence-based treatments'.[81,82] The general reviews highlight that research of psychodynamic treatment in the field of child therapy has lagged behind the evaluation of other approaches.

There are relatively few randomized controlled trials of psychodynamic psychotherapy.[83–88] All but one of these trials contrasted individual child psychotherapy with another (evidence-based) treatment. In addition, several studies employed quasi-randomized methods of assignment, such as postcode[89] or therapist vacancy.[90,91] Six studies reported on findings with matched comparison groups.[92–96] A further two studies reported non-matched control groups.[97,98] Two further studies used an untreated but poorly matched control sample.[99,100] In addition, there are a number of open trials of child psychotherapy employing no comparison groups.[101–107] Three studies used an experimental, single-case methodology.[108–110]

Considering studies of the common disorders of childhood separately, Muratori and colleagues[91] contrasted the efficacy of 11 weeks of psychodynamic therapy to treatment as usual in 58 children with depression and anxiety. At 2-year follow-up, 34 per cent of the treated group were in the clinical range on symptomatic measures, compared to 65 per cent of the controls. Treatment effects increased during the 2-year follow-up period (the so-called 'sleeper effect'), including a move into the non-clinical range for the average child with internalizing problems (in the psychodynamically treated group only).[90] It is encouraging that psychodynamic psychotherapy patients sought mental health services at a significantly lower rate than those in the treatment as usual comparison condition over the 2-year follow-up period.

In a multi-centred European trial,[88] moderate childhood depression was shown to be accessible to a brief individual psychodynamic psychotherapy. At 7-month follow-up, none of the moderately to severely depressed young people met criteria, which is comparable to children treated with a combination of fluoxetine and CBT.[111] The presence of anxiety/dysthymia signalled particular suitability for individual treatment, whilst comorbidity with oppositional defiant disorder (ODD) or conduct disorder (CD) contraindicated it. These children, however, appeared to do better with family based approaches that were also psychodynamic in orientation. A classic study, and for many years one of the only studies that considered the issue of intensity of psychological therapy, focussed on specific learning difficulties as a target of therapy.[93,112] Boys in middle childhood with serious reading problems benefited significantly from psychodynamic psychotherapy. There was a dose-response relationship over the 2-year treatment period. Children who received more intensive help (more sessions per week) benefited most from the therapy in terms of self-esteem, the capacity to form relationships, and the capacity to work, including frustration tolerance. Particularly interesting is the multi-centred randomized trial of the treatment of sexually abused girls treated in individual psychotherapy and psychoeducational group therapy.[87] Trowell *et al.*[87] randomized 71 sexually abused girls to either 30 sessions of individual psychoanalytic psychotherapy or 18 sessions of group psychotherapy with psychoeducational components. These young people presented with a range of psychiatric problems, most commonly post-traumatic stress disorder (PTSD) and depression. Psychodynamic treatment was somewhat superior to psychoeducation, but the difference was not as marked as might be expected. Superiority was particularly evident in relation to PTSD and generalized anxiety disorder (GAD). Depression, however, was relatively less likely to improve, as was separation anxiety. A subsequent report underscored the importance of the mother's support for the therapy as a predictor of improvement in the children and the benefit that the mothers gained in terms of their own mental health from the child's treatment.[113]

A chart review of the outcome of 763 cases in child psychotherapy has been carried out at the Anna Freud Centre in London.[103] While this retrospective methodology has severe limitations, the study reached a number of fairly robust conclusions, that need to be explored further in controlled, prospective investigations. The main findings were:

(a) Attrition was low compared to reports of other treatment approaches.

(b) Children with pervasive developmental disorders (e.g. autism) or intellectual disability did not do well, even with prolonged, intensive treatment. Children with serious disruptive disorders also had relatively poor outcomes.

(c) Younger children improved significantly during psychodynamic treatment, and gained additional benefit from 4–5 weekly sessions.

(d) Anxiety disorders, particularly specific rather than pervasive symptoms, were associated with a good prognosis, even if the primary diagnosis was of a different type, e.g. disruptive disorder.

(e) Children with emotional disorders and/or severe or pervasive symptomatology responded very well to intensive treatment (4–5 sessions per week), but did not show satisfactory rates of improvement in non-intensive psychotherapy.

(f) Predictors of improvement varied considerably between subgroups of the full sample, and, by subdividing the sample according to diagnostic group and developmental level, it was possible to predict a majority of the variance in outcome within the subgroups.

Limitations of the psychotherapeutic approach

The empirical status of all psychodynamic approaches remains controversial. The body of rigorous research supporting psychodynamic therapies for adults for most disorders remains limited, particularly relative to research supporting pharmaceutical treatments and even other psychosocial approaches such as CBT.[80] There are both practical and theoretical difficulties to mounting trials of dynamic therapies, and these go some way to explaining the lack of evidence (e.g. identifying suitable control groups for long-term intensive treatments, difficulties in operationalizing the treatment methods, the expense of mounting trials sufficiently powered to yield information on what treatments are appropriate for which disorder, the failure to tightly manualize psychodynamic treatments, etc.). Those who argue for continued investment in this approach (correctly in our view) point to the limitations of the evidence base supporting CBT[114] or pharmacological approaches.[115] Ultimately, however, such a negative case cannot persuade policy makers and funders, and, without intense research on the effectiveness of the method deeply rooted in and shaped by psychological models of pathology, the long-term survival of this orientation is not assured.[116] This is not to say that the techniques that have evolved as part of this approach will not survive (they are effective, and clinicians, being pragmatic people, will continue to discover and use them), but they will be increasingly absorbed into alternative models, and the unique approach pioneered by Freud and outlined in this chapter might not continue. Child therapists thus face formidable challenges to their clinical and theoretical convictions, to their professional status, and, as evidence-based medicine and managed care relentlessly expand their control over reimbursement and deny payment for child therapy, to their livelihood. If child psychotherapy is to have a future, its unique effectiveness for specific childhood disorders must be demonstrated in randomized controlled trials. We believe that children with severe disorders of personality and multiple psychiatric diagnoses are indeed well suited to and dramatically benefit from a psychotherapeutic approach. Such faith, however, now requires support from empirical investigations.

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9.5.3 Cognitive behaviour therapies for children and families

Philip Graham

Introduction

Cognitive behaviour therapy (CBT) is derived from both behavioural and cognitive theories. Using concepts such as operant conditioning and reinforcement, behavioural theories treat behaviour as explicable without recourse to description of mental activity. In contrast, mental activity is central to all concepts derived from cognitive psychology. Both sets of theories have been of value in explaining psychological disorders and, in the design of interventions they have proved an effective combination.

Central to that part of cognitive theory that is relevant to CBT is the concept of 'schemas', first described in detail by Jean Piaget.[1] A schema is a mental 'structure for screening, coding, and evaluating impinging stimuli'.[2] The origin of mental schemas lies in the pre-verbal phase when material is encoded in non-verbal images that, as the child's language develops, gradually become verbally labelled. They form part of a dynamic system interacting with an individual child's physiology, emotional functioning, and behaviour with their operation depending on the social context in which the child is living. There are similarities but also differences between schemas and related concepts in psychoanalysis, such as Freudian 'complexes' and Kleinian 'positions'.

Schemas can be seen as organized around anything in the child's world, especially objects, beliefs, or emotions. They develop from past experience. The processing of new information in relation to such schemas can usefully be seen as involving the evaluation of discrepancies between information that is received and information that is expected. If there is a discrepancy, (the information not corresponding with that expected), then during the coding process information may be distorted so that it no longer creates discomfort, or, more adaptively, it may be incorporated into a modified schema.

Cognitive development

The theory of cognitive development that Piaget constructed on the basis of an immense amount of experimental work was characterized by stages of development. He described characteristic features of the sensori-motor (0–2 years), pre-operational (2–7 years), concrete operational (7–12 years), and formal operational (12 years onwards) stages. Before the end of a stage is reached the child is incapable of showing more advanced thinking. In particular, the child's thinking before the concrete operational stage is characterized by egocentricity and an inability to take the perspective of another person. Abstract reasoning is not possible for the child until the formal operational stage is reached.

Even though Piaget's views of the limitations of the cognitive abilities of young children have been strongly criticized especially on the grounds that he was judging egocentricity on the basis of findings obtained in highly artificial situations, Piaget remained a dominant influence in cognitive psychology and education throughout the twentieth century. It is now widely accepted that, although obviously young children are less competent than those in middle childhood and these are less competent than adolescents, cognitive competence advances much more rapidly than Piaget described and the social context in which a child's competence is investigated has a much more profound influence on performance than he allowed. Children do a great deal better in naturalistic circumstances than when they take part in experiments. Further, coaching can improve performance to a level not previously obtainable. For example, it has been shown that, with preliminary training, 3-year-old children understand that drawings of thought bubbles can represent what people think. They can distinguish between thoughts and actions, recognize that thoughts are subjective and that two people can have different thoughts about the same events.[3]

Investigation of the development of the 'theory of mind' held by children has revealed that between 3 and 4 years they begin to realize that other children can be deceived by appearances and hold false beliefs they themselves do not hold. This shows that, given the right circumstances, children of this young age are able to 'de-centre' and are not necessarily limited by egocentricity. By the age of 8 years children have such stable concepts of their own self-esteem that they are capable of reliably completing self-esteem questionnaires about their own feelings and performance in comparison to other children.[4] Some schemas in young children are however relatively unstable, gradually increasing in stability as they get older. For example, it has been shown that attributional style (the tendency to attribute adverse events either to the self or to external circumstances) does not become stable until early adolescence,

though it may be identified earlier if the events are particularly salient to the child in question.[5]

It has been hypothesized[6] that maladaptive schemas developed during childhood are responsible for the formation and maintenance of adult psychopathology. Building on this model, a therapeutic approach (schema-focused therapy) based on the identification of particular maladaptive schemas has been proposed for adults. Subsequently Stallard and Rayner[7] have developed a schema questionnaire that builds on adult work to identify such maladaptive schema in 11 to 16-year-old school children.

Technique and management in the paediatric age group

Although there are certain common principles, CBT does not involve, as will be seen, a single approach that can be applied across all disorders; it is better seen as a family of approaches with certain core elements in common. In adults the type of disorder and the individual circumstances of the patient will determine the choice of therapeutic methods. In children and adolescents the cognitive level of the patient will also need to be taken into account. Though the age of the child will give some indication of the cognitive level of the child, there is wide variation in competence amongst children of the same age. Further, the therapist may use the skills of an educationist to bring the child's competence up to a level at which the child can more actively participate in therapy. Kendall[8] suggests indeed that one of the therapeutic roles that the therapist should adopt is that of *educator*, who needs communication skills to assist children to learn to think for themselves.

Behaviour therapy or CBT?

In principle, the decision as to whether to include a cognitive component in therapy depends on whether the clinical formulation incorporates cognitive distortions or biases. In practice, because of their cognitive limitations CBT is rarely used in children under the age of 7 years. Treatment in children younger than 7 years is predominantly behavioural, with the cognitive component limited to coping self-talk. Conditioning approaches to the treatment of feeding and sleeping problems as well as enuresis and encopresis usually have a very small or no significant cognitive component.

In some conditions such as anxiety disorders, especially specific phobias, where desensitization and reinforcement approaches are widely used in adults, the use of a mainly behavioural approach does not reduce effectiveness. A cognitive component may nevertheless be incorporated because the CBT principles of collaboration, openness, and guided discovery, usually less marked when purely behavioural approaches are applied, are advantageous to the patient.

Aids to cognitive tasks

Where experience with adults suggests that cognitive tasks add significantly to the effectiveness of treatment, as in depressive disorders and problems of social relationships, even young people in early adolescence will usually be able to co-operate as well as adults. The cognitive treatment of younger children with these conditions may be helped by the use of age-adapted techniques.[9] For example, card-sorting *games* have been devised to help children distinguish between thoughts, feelings, and situations. *Puppets* can be used to facilitate discussion as part of the assessment process, to model alternative ways the child might cope with difficult situations and to engage the child in rehearsal and practice of new skills. *Story telling* can provide an insight into the child's inner world; they provide a way of externalizing and accessing the child's cognitions, allow an opportunity to challenge the child's assumptions, introduce the child to more positive ways of coping, and can be used to model success and help the child gain more functional assumptions and beliefs.

Working with parents

Parents play many roles in the delivery of CBT to children and adolescents. To begin with, even up to mid-adolescence, it is nearly always parents who identify the behaviour and emotional problems that lead to advice being sought. They are the people most likely to press for psychological help. It is they who have to persuade often reluctant children and adolescents to attend and participate in a service that their offspring may fear, not without reason, will result in stigmatization.

They are then likely to play a major part in the assessment process. From mid- to late adolescence, the patient or client will be the main source of information, but before that it is the parents and teachers who will often provide most relevant information. If treatment is proposed it is they who need to give consent, though their child will also need to assent if the therapy is to have any chance of succeeding.

Once treatment planning has begun, the part that parents play will depend very much on the age of the child or adolescent, the diagnosis, family circumstances (especially the quality of the relationships between parents and child), and the degree to which the assessment has revealed that the parents as well as being the main carers are also involved in the origin and maintenance of the problem. Most explanatory theories of anxiety disorders in children, for example, point to the ways in which parents can provide inappropriately anxious models for imitation by their children. In a small scale study it has been shown that changing parental attributions can, in itself, result in improvements in problem behaviour scores on a questionnaire.[10] Parents may also be seen as clients in their own rights in parallel sessions, as co-therapists or as facilitators of therapy for their children. Therapists dealing with adolescent offspring are often in a difficult position *vis-à-vis* parents in that they will wish to encourage autonomy and independent decision-making in the child or adolescent, while needing the parents to monitor homework, encourage further attendance, and provide information on progress.

The involvement of parents also brings ethical dilemmas. There are three main areas of ethical concern.[11] The therapist often has to balance the different viewpoints of parents and children, a particular problem in the management of oppositional and conduct disorders where children often fail to acknowledge the existence of problems that are causing distress to their parents. There is frequently need to address family issues such as marital conflict that are clearly relevant yet not the reasons why the child has been brought for treatment. Finally, there is the need to achieve genuine collaboration with parents, making explicit their role as co-therapists. This is made easier if children are also actively involved as fellow

collaborators, taking responsibility for progress and being encouraged to make suggestions for alternative approaches. A collaborative stance may however not be possible if it becomes clear that there are child protection issues with one or both parents involved in maltreatment of their children. Wolpert and her colleagues provide a useful checklist for clinicians to help assess how far they are attempting to balance different viewpoints in issues involving different family members and promoting collaboration.

Failure to engage and failure to respond

In adolescents, lack of motivation for change is often a major impediment to engagement in therapy. Not only is there often a failure to recognize the importance of a problem, to accept the need for change or to appear to understand why change is necessary, but there may also be an absence of the level of self-belief, self-confidence, or self-efficacy that is necessary before hopeful steps can be taken in the right direction. In these circumstances techniques of motivational interviewing will help the therapist to achieve engagement.[12]

The reasons for non-response to CBT in adults have been discussed by Kingdon *et al.*[13] Common problems include unsuitability for treatment possibly arising from misdiagnosis, resistance to treatment, an inadequate number of sessions, difficulties in the therapeutic relationship and the presence of concurrent social and/or physical pathology. Non-response in children and adolescents arises from similar issues, with, additionally, complicating problems arising from negative parental attitudes and behaviour.

Anxiety disorders

Cognitive distortions and deficits

A characteristic constellation of cognitive deficits and distortions underlies the presence of anxiety disorders in children and adolescents. A central feature is the exaggerated perception of threat arising from an inability to assess accurately the seriousness of danger. Thus a deficit in perceptual competence results in cognitive distortion. The characteristic nature of the threat involved will depend to a considerable degree both on the stage of cognitive development of the child and on the social demands that are encountered during that particular phase of life. Pre-school children are most likely to be threatened by separation from parents; children aged 5 to 12 years by feared situations at school and adolescents by social situations as well as wider concerns such as environmental pollution. Certain fears and phobias such as fear of spiders and snakes appear more biologically based and are present through childhood to adolescence.

These cognitive deficits and distortions both result in and are maintained and increased by abnormal levels of physiological arousal and by behavioural avoidance of the feared situations. Autonomic arousal produces symptoms such as dry mouth, palpitations, and abdominal pain and these may be misinterpreted as implying serious threatening illness. Panic attacks may be catastrophized and taken to mean that death is imminent. Avoidance of feared situations such as separation from parents in younger children, refusal to go to school in older children or to social events such as parties in adolescence prevent cognitive testing of the reality of the supposed threat and reinforce the cognitive distortion.

The fact that anxiety disorder is partly genetically determined means that children suffering from this condition have an increased risk of having anxious parents. Such parents are likely to model anxious behaviour, especially in the way they show over-protection to their children. Anxious children are therefore likely to be exposed to social learning situations at home that will increase the risk of avoidance of feared situations. Gene-environment interactions ensure that many parents who cannot bear to be separated from their children or who are anxious every time they leave the house will transmit their fears to their children both directly and indirectly. In adolescence, anxious young people may selectively choose shy, inhibited friends who reinforce their sense of unrealistic threat.

Techniques of assessment and intervention

The assessment of children with anxiety disorders by a cognitive behaviour therapist focuses on the identification of cognitive deficits and distortions and the manner in which they are currently being reinforced, especially by avoidant behaviour. Nevertheless it is important that before enquiry is made along these lines a full history is taken of the development of anxious symptoms, the presence of other symptomatology, the situations that increase and reduce anxiety, the presence of anxiety in parents, sibs, and friends, and the measures that have already been taken, especially by parents, to improve the condition. Skilled assessment involves listening to the anxious preoccupations of both children and parents sympathetically and without any hint of criticism.

There are a number of systematic cognitive approaches to the reduction of anxiety in children of which the most widely used is the four-step coping or FEAR plan, in which F = **F**eeling frightened (awareness of anxiety symptoms such as somatic aches and pains), E = **E**xpecting bad things to happen (awareness of negative self-talk), A = **A**ttitudes and actions that can help (problem-solving strategies), and R = **R**esults and rewards (rewarding for success, dealing with failure).[14] The 'Cool Kids' programme is generally similar but puts more emphasis on parent involvement.[15] When parents show significant levels of anxiety themselves, effectiveness of treatment is enhanced if parental anxiety management is included as part of treatment.[16] A self-help book for parents broadly based on the same principles provides a practical approach to the management of anxiety, using the so-called COPE programme.[17]

Treatment begins with one or two psycho-educational sessions in which the child and parent(s), together or separately, are given information about the way anxiety develops and is maintained, the manner in which the body shows anxiety (somatic symptoms), and the effects of avoidant behaviour and exposure to feared situations. It is important that these sessions are interactional with the child being encouraged to talk spontaneously about, for example, how he or she experiences somatic symptoms. The next few sessions involve children engaging in an exercise to identify their own negative thoughts, to test them against reality and to develop positive thinking in situations that have previously triggered anxiety. This will usually need to be done in imagination before it is tried out using 'graded exposure' in real situations. There are advantages in teaching relaxation techniques before the child embarks on exposure to feared situations. The use of imagery, such as the 'stepladder' approach to a hierarchy of feared situations may also be helpful. When the child makes progress, as is usually the case, rewards such as outings or other treats may be built in to the procedure.

Therapists vary in the degree to which they involve parents in management. The therapy can be delivered in a family context, parents can be seen separately from children, parents may not be seen at all, or the therapy may only be delivered to parents. Some centres use a group approach, with one or two therapists providing a group experience for parents and anxious children who go through the stages of treatment together and benefit from learning of each others' experiences. Some programmes have now been developed for use via the Internet with minimal personal contact with the child and family. Some therapists combine CBT with the use of medication, generally not anxiolytic agents because of the risk of dependency, but tricyclics or selective serotonin reactive inhibitors.

Evaluation of effectiveness and efficacy

A systematic review of the effectiveness of CBT for anxiety disorders in childhood and adolescence identified 10 randomized controlled trials that met inclusion criteria.[18] The outcome measure used was the remission of anxiety disorder. The remission rate was higher in the CBT groups (56.5 per cent) than in the control groups (34.8 per cent). The pooled odds ratio was 3.3 (CI = 1.9–5.6). The authors of this review conclude that CBT definitely provides benefit to children and adolescents with anxiety disorder, but that there is a lack of information concerning the value of CBT in younger children and that there are virtually no satisfactory studies comparing effectiveness with alternative treatments.

There is contradictory evidence concerning the importance of involving parents in therapy. Some[19,20] find little or no benefit, while others[21,22] find a trend towards benefit. A pilot study has found benefit from a programme that did not involve children directly but only involved parents seen in a group, who applied what they had learned in the group in managing the situations in which their children showed anxiety at home. Information on the use of therapy delivered via the Internet is limited, but those that exist suggest that Internet treatment is highly acceptable to families, creates minimal dropout and is effective when added to clinic treatment.[23] Dropout from more conventional treatment is likely to be high in single-parent families, ethnic minority families, and where anxiety levels are not conspicuously high.[24] There is evidence that the presence of co-morbid disorders does not reduce the efficacy of CBT.[25] The addition of antidepressants may increase the efficacy of CBT, especially in the treatment of school refusal.[26] Limited findings from long-term studies suggest that treatment benefits from the delivery of CBT to anxious children are maintained over at least 6 years.[27]

There is also evidence from controlled studies for the effectiveness of interventions, especially the FRIENDS programme[28] in the prevention of anxiety and depression in early adolescence. Stallard *et al.*[29] have shown how this programme can be delivered successfully by school nurses.

These evaluative studies have provided most encouraging findings for the effectiveness of CBT in this condition. However the findings also make clear that CBT, while producing worthwhile and persistent benefits in most children and adolescents with anxiety disorders, is not effective in a significant number of cases and in a significant number of others it is only partially effective. It is also less effective in socially disadvantaged groups. Finally, most evaluative studies have been carried out in highly specialist centres and there is a lack of evidence for their value in everyday practice.[18]

Depressive disorders

Cognitive distortions and deficits

The classical signs of depressive disorders, such as chronic misery and unhappiness, lack of interest in food, and motor retardation, may be seen as early as the first year of life. Infants and young children who show such symptomatology may well suffer depressive experiences similar to those of older people though in the pre-verbal phase there is no reliable method available to confirm this possibility. Awareness of feeling states develops towards the end of the second year of life.[30] By 2 to 3 years children realize that there can be a variety of personal reasons for an emotional reaction. By 4 years there is some consensus about the kind of situations that will provoke the common emotional reactions, including fear, sadness, and anger.[31] By 5 or 6 years a child is capable of understanding the concept of stability of mood, 'always being unhappy or just now and again', and by 7 or 8 years concepts of shame and guilt are understood at least in simplified form. Enduring and relatively stable negative attributions about the self become possible at around this age and the concept of death as a permanent state is established. By 13 to 14 years, emotional experiences of adult intensity occur and mature cognitions about different mood states will have been attained. Although the above account relates stage of development to chronological age, there is wide variation in the ages at which cognitive competence is gained. Further, the settings in which children are questioned or encouraged to express themselves freely and spontaneously, for example in play situations will greatly influence their capacity to show their abilities.

The cognitive model underlying CBT approaches to children and adolescents does not differ from that with adults. It is assumed that thoughts are the primary experience of depression and that depressed mood is secondary. Dysfunctional assumptions, including low feelings of self-worth, self-blame for events in the past, and hopelessness about the future are present either as stable features of a depressive personality or as a reaction to adverse experiences, real or imagined. Depressed children and adolescents systematically distort their experience to match their beliefs about themselves. At some point, these negative thoughts are automatically experienced without reflection. Increasingly situations are avoided because of a fear of negative outcomes. Therapy involves identifying and reality testing these negative thoughts. In addition the patient is encouraged to enter into activities that will be rewarding and disconfirm pessimistic assumptions.

Techniques of assessment and intervention

Initial assessment will involve taking a full history of the development of symptoms and the factors that reduce or exacerbate them, the child's functioning in different settings, and an account of family relationships. If the child is taken on for CBT, a typical approach[32] begins with the establishment of symptom status by the use of questionnaires such as the Children's Depression Inventory[33] in young patients and the Mood and Feelings Questionnaire[34] in adolescents. The goals of therapy are then discussed in a collaborative manner with emphasis on what the child or young person wishes to achieve. The proposed therapeutic approach is then explained together with the importance of homework outside the

therapy sessions. An indication of the number of sessions likely to be required, usually 12–16, is given. In early sessions an account of the child's current daily activities is obtained. Adolescents are helped to keep a diary of their activities and moods. In a form of 'affective education' a check is made on the vocabulary the child uses to describe feelings and links are then established between the child's mood and the activities he or she is undertaking.

During the next sessions, in collaboration with the child, homework is planned that aims to increase activity to the level previously undertaken. Emphasis is placed on the resumption of everyday activities rather than offering treats or special occasions. At this point a problem-solving approach may be indicated. This begins with problem definition, followed by brain-storming a number of different solutions. The outcomes for different solutions are discussed and a plan developed to achieve what seems to be a satisfactory outcome. Homework involves attempts to implement the plan while keeping a record of progress and how this has influenced mood.

At least from early adolescence it will usually be possible to introduce self-monitoring procedures, in which the child identifies and notes his level of mood in relation to the thoughts he is experiencing. The child is encouraged to imagine different situations and to record how each situation makes him feel. The child is encouraged to continue this process at home, recording what happens so that his experience can be discussed in the next session. This process is accompanied by self-evaluation training, a form of cognitive restructuring in which children learn to evaluate themselves in a more positive manner. They are encouraged to consider the evidence for having a poor opinion of themselves and then to examine carefully more positive alternative explanations. This process may be expected to reduce negative automatic thoughts.

Evaluation of effectiveness and efficacy

A comprehensive evidence-based review of controlled evaluation of cognitive behavioural psychotherapy for children and adolescents with depressive disorders[35] identified 12 studies that fulfilled methodological criteria. Most reported positive outcome for CBT post-treatment and at short-term follow-up. However, studies with longer follow-up periods from 9 months to 2 years found that a sizable percentage of subjects continued to report significant depressive symptoms or a recurrence of their depressive illness.

More recently the results of a major multi-centre trial, the Treatment of Adolescent Depression study (TADS) have been reported. In this study 479 adolescents, aged 12 to 17 years with depressive disorders were allocated randomly to a combination of fluoxetine and CBT, fluoxetine alone, CBT alone and an inert pill placebo. After 12 weeks of treatment the effects of combination therapy were clearly superior to either form of monotherapy and greatly superior to pill placebo. Fluoxetine alone was superior to CBT alone and to placebo, but CBT alone was not superior to placebo. On the other hand, fluoxetine alone was accompanied by higher rates of suicidal events and this did not occur in the combined group. It seemed therefore that CBT protected against the suicidality linked to fluoxetine use. The investigators concluded that the combination treatment produced the best outcomes.[36]

The published evidence mainly relates to children and adolescents with mild or moderate depressive disorders. There is some indication both from the TADS described and from other evidence that more severe depressive disorders do not respond as well or perhaps not at all to CBT alone.[37]

Attempts to use CBT to prevent depression in adolescents have met with varied success. One universal school-based approach found no difference at 2 to 4-year follow-up in children who received a teacher-administered cognitive behavioural intervention compared with a control group.[38] In contrast, positive effects were found for a CBT intervention targeting 13–18-year-old children of parents with depressive disorders.[39] Application of the Resourceful Adolescent Programme has also been shown to produce promising results in preventing depression in younger adolescents.[40]

Conduct disorder

Cognitive distortions and deficits

Both young children with oppositional disorders and adolescents with more severe conduct disorders show characteristic cognitive distortion in their thinking. They recall inaccurately high rates of hostile cues in social situations and when neutral remarks and movements are made by their peers, they see these as hostile.[41] In competitive situations with peers they exaggerate the aggressive behaviour of others and underestimate their own aggressiveness. These distorted attributions lead them into aggressive behaviour which then triggers angry behaviour from peers so that the originally neutral environment does indeed become more hostile.

Aggressive children and adolescents also have difficulties in problem-solving, both in experimental and naturalistic situations. They prefer rapid action-orientated solutions to those that require reflective thinking before any action is taken. Underlying this tendency to prefer rapid, aggressive solutions is the fact that their social goals relate more to the need for dominance and revenge than for affiliation.[42]

Parents of aggressive children also show cognitive distortions that are of relevance to the way they discipline their children.[43] This is of relevance both to the understanding and the management of childhood conduct disorders. For example, it has been shown that mothers of children with conduct disorder tend to attribute their children's difficult behaviour to deliberate wilfulness that is not within their children's capacity to control. They perceive themselves as helpless in the face of their children's behaviour. These cognitive distortions prevent them from acting effectively as parents, for example by drawing firm boundaries between acceptable and unacceptable behaviour.

Techniques of assessment and intervention

Until the early 1980s there were really no effective, evidence-based psychological interventions for children and adolescents with conduct disorder. Since that time a number of moderately effective psychological measures have been developed. All of these, including the cognitive behavioural techniques described below are only likely to be successful if they are combined with psychosocial measures directed towards the family as well as with appropriate education. All approaches require preliminary assessment of the child and family to identify the severity of the disorder and the possible presence of co-morbid disorders as well as to determine suitability for the approach envisaged.

Cognitive approaches to conduct disorder have been summarized by Lochman *et al.*[44] *Problem-solving skills training (PSST)*

has been developed for children aged 7 to 13 years. The programme is delivered over 25 sessions.[45] The group leaders teach problem-solving skills such as generating multiple solutions to a problem and reflecting on the different consequences of the alternatives. The skills are applied to interpersonal situations with teachers, parents, peers, and siblings. Parent participation is a major component of the training, with parents observing the sessions and acting as co-therapists in supervising the use of the new skills in the home.

The Anger Coping and Coping Power Programme is a school-based prevention programme delivered in group sessions to 13–14-year-old children.[46] The group sessions focus on enhancing emotional awareness, anger management training, attribution retraining and perspective-taking, social problem-solving and social skills training, behavioural and personal goal-setting, and handling peer pressure.

Multi-system therapy was designed as a multi-level intervention for 12 to 15-year-olds with multiple, severe antisocial problems.[47] Highly trained and closely supervised psychologists manage individualized programmes in the home setting. A variety of treatment approaches, including parent training, family therapy, school consultation, and individual therapy are employed in association with social measures such as helping lone mothers to find employment are used. The aim is to achieve change in one area before targeting another.

Functional Family Therapy combines family systems and cognitive behavioural approaches. The programme begins with an engagement and motivation phase in which the therapist addresses maladaptive beliefs in the family system thus aiming to increase expectations for change, reduce negativity and blaming, build respect for individual differences and develop a strong alliance between family members and the therapist. Practical behavioural interventions are designed to produce change and this is followed by a generalization phase in which the family is encouraged to interact effectively with the various systems in the community with which it is in contact.

Parent Management Training Programmes usually involve parents of young children with oppositional or conduct disorders. They derive from work originally carried out by Patterson and his colleagues at the Oregon Social Learning Center. His findings established the importance of coercive parental behaviour in the development of childhood aggression. Many treatment programmes have been based on their work,[48,49] which focus on ways of reducing parental coerciveness, often in group settings. Parents are taught to pinpoint problem behaviours, to apply positive reinforcement when their children's behaviour is more appropriate and to learn problem-solving and negotiating techniques. It has been suggested that the incorporation of a cognitive component into parent training, using a 'thoughts, feelings, behaviour cycle' can improve effectiveness of this approach.[50]

Evaluation of efficacy and effectiveness

All of the above programmes have been evaluated in controlled clinical trials and have been shown to be moderately effective.[51–53] However, virtually all the controlled studies have been carried out in highly resourced specialist centres. There is a conspicuous lack of studies of effectiveness carried out in routine clinical care.

Attention deficit hyperactivity disorder (ADHD)

Cognitive distortions and deficits

Children with ADHD show a range of cognitive deficits of attention and concentration with a strong predisposition to impulsivity, accompanied by explosive temperament and poor regulation of affect and impulses. Until recently these problems have been explained on the basis of deficits in one of two cognitive pathways. It has been proposed that there is a deficit in executive function, based on deficient inhibitory control arising from frontodorsal striatal brain networks.[54] Such failure of control results in deficits in self-monitoring, planning, attentional control, and executive skills. Stimulant medication remedies these deficits by increasing the activity of inhibitory pathways. Alternatively the condition has been attributed to disturbances in motivational processes, manifest as aversion to delay in gratification. More recently Sonuga-Barke,[55] has proposed that both these mechanisms are supported by the evidence and that there are two distinct but complementary neuro-developmental bases for ADHD which is thus, at least in the pre-school period, psychologically heterogeneous. As children get older and executive function matures, deficits in executive functions may become more prominent in affected children, especially in the areas of inhibition, set shifting, working memory, planning, and fluency.[56]

Techniques of assessment and intervention

Information about children with suspected ADHD needs to be obtained from both parents and school teachers as the child is likely to behave differently in the two settings. Both interviews and rating scales should be used. Observation of the child can confirm the presence of ADHD, but cannot rule it out as some children who are clearly showing symptomatology both at home and in school may appear normal in the clinic. Although it is helpful to reach a diagnosis, increasingly treatment approaches are focusing on the presence of specific impairments rather than on the presence of symptoms.[57]

Three types of psychological interventions have been found of value: ensuring the child's environment is structured and, when the child is engaged in a task that there is an absence of extraneous, distracting stimuli; counselling to parents and teachers, and behavioural and/or cognitive behavioural approaches directed to the child.

In the classroom the child will benefit if seated close to the teacher, task demands are kept short and there are interspersed periods of physical exercise. Teachers should be helped to reduce negative interactions by focusing on positive reinforcement for appropriate behaviour however brief this might be. Short periods of timeout before potentially problematic situations get out of hand may reduce the number of painful, angry confrontations. Similar principles can be applied in the home situation with parents being helped to understand and act on the principles of the identification of antecedents that result in problematic behaviour which will then have consequences that either increase or reduce the likelihood of recurrence. Positive reinforcement can be provided in the form of star charts, tokens, or other rewards. Training of parents of children with ADHD follows similar lines to training of parents with children with conduct disorder (see above) and, of course, many children show co-morbid ADHD and conduct disorder.

Cognitive behavioural approaches generally aim to achieve increased self-control. Most approaches involve encouraging appropriate self-instruction. The child is taught separate steps of self-instruction ('Stop: What is the problem?—Are there possible plans?—What is the best plan?—Do the plan—Did the plan work?) This approach can be applied when the child is faced with cognitive tasks which would usually be tackled impulsively or to social situations that often result in confrontations, such as arguments with parents or friends.[58]

Evaluation of effectiveness and efficacy

The delivery of behavioural treatment to children with ADHD presents particular problems. As a group they are slow to respond to conditioning procedures. Their distractibility and short attention span leads to problems in co-operation. Parents are likely to show similar behavioural and cognitive characteristics to their children, so collaboration of parents in treatment regimes may be problematic. The children's lack of reflectivity is a barrier to the use of cognitive approaches.

There is no good evidence that cognitive approaches alone are significantly effective in children with severe ADHD.[59] The most thorough evaluation of behavioural approaches to date is the Multi-modal Treatment of Attention-Deficit Hyperactivity Disorder (MTA) study carried out in the 1990s. The 579 children in the study were randomly allocated to one of three conditions: medication with stimulants, intensive behavioural treatment, and a combination of the two. The behavioural approach involved a parent training component, a two-part school intervention and an intensive summer treatment programme. It can therefore hardly be regarded as typical of psychological interventions applied in everyday clinical practice. There were slight advantages to combined treatment over medication alone. Behavioural treatments alone were much less effective for ADHD, though more useful for co-morbid anxiety disorders.[60] A 9-month follow-up revealed that the effectiveness of behavioural management approaches had been maintained over this period.[61] Interpretations of the findings of this study have been divergent. Re-analysis suggests that it may well be that medication alone or in combination with behavioural treatment is strongly indicated in severely affected children, while behavioural treatment and parent training are equally effective where impairment is mild or moderate.[58]

Parent training alone is effective in pre-school children with mild or moderately severe ADHD when delivered in a specialist setting, but is not when provided as part of routine primary care by non-specialist nurses.[62]

Obsessive–compulsive disorders

Cognitive distortions and deficits

The core cognitive distortion in children and adolescents with obsessive–compulsive disorder (OCD) is thought to lie, as it does with adult patients, in the appraisal of responsibility.[63] This is defined as 'the belief that one has power which is pivotal to bring about or prevent subjectively crucial negative outcomes'. Now we all do have responsibility for our actions; what makes patients with OCD different is that they take upon themselves quite unreasonable levels of responsibility. A 13-year-old might, for example, think 'I am responsible for making sure my mother does not die'. This sense of responsibility leads to attempts both to suppress and to neutralize the unwelcome thoughts of responsibility.

'Neutralizing' is defined as voluntary activity intended to have the effect of reducing the perceived responsibility. 'If I tap on my glass three times before I drink from it, my mother will not die'. But neutralizing activities increase discomfiting cognitions and this leads to further neutralizing activity. Attempts to suppress the intrusive thoughts also increase the likelihood of their recurrence. An additional complicating feature of the cognitive distortion is that, in the mind of the child with OCD, thoughts become imbued with unrealistic or magical powers. It is enough just to have a thought for it to be translated into action, so-called 'thought-action fusion'. 'If I allow myself to think about my mother dying this will mean that she will die'.

In general the cognitive distortions made by children and adolescents with OCD are similar to those seen in adults. However in a study comparing the various components of OCD cognitions in children, adolescents and adults it was found that children experienced fewer intrusive thoughts and these were less distressing and less uncontrollable than those experienced by adolescents and adults.[64] On the other hand, cognitive processes of thought-action fusion, perceived severity of harm, self-doubt and cognitive control were similar across the three age groups.

Techniques of assessment and intervention

The aim of cognitive therapy is to help the patient reach the view that obsessional thoughts, however distressing, are irrelevant to any activities that may be undertaken in the future. This is achieved by increasing the patient's sense of personal efficacy, predictability, controllability, and self-attributed likelihood of a positive outcome. The techniques used involve the conduct of tasks involving exposure to feared stimuli as well as response prevention, stopping the activities that reinforce the unwelcome thoughts.

The most widely applied treatment approach to OCD in children and adolescents is that developed by John March and his colleagues.[65] The treatment protocol involves 12 sessions of which the first two are spent on psycho-education and cognitive training and the next 10 sessions on exposure and response prevention with the first and last two sessions, as well as an intermediate session involving parents. The effectiveness of exposure depends on the fact that anxiety diminishes after repeated contact with a feared stimulus. Thus the anxiety of a child worried about germs will be reduced by prolonged contact with a surface the child thinks has germs on it. Encouraging parents not to provide reassurance to children who compulsively and repetitively demand it, removes reinforcement, and results in extinction of the behaviour. Some children become extremely distressed when their parents, on instruction, fail to provide such reassurance; more success is achieved by putting the child in control of reducing parents' inappropriate behaviour. Modelling and shaping behaviours are also helpful in giving the children or adolescents the skills to expose themselves to feared stimuli. Liberal use of rewards when the child behaves appropriately is also helpful in reinforcing desired behaviour.

Evaluation of effectiveness and efficacy

The most informative findings on efficacy come from the Pediatric OCD Treatment study (POTS) Team.[66] 112 patients aged from 12 to 17 years, suffering from OCD were divided randomly into four

groups: CBT alone, sertraline alone, combined sertraline, and CBT treatment and a pill placebo. Both sertraline alone and CBT alone were superior in outcome to pill placebo at 12 weeks after the beginning of treatment. But combined treatment was superior to both treatments administered separately with a clinical remission rate of 54 per cent, compared to 4 per cent for placebo.

Most studies report the results of individual treatment with children and adolescents, with limited input from parents. Initial findings suggest that for middle-school aged children, aged 8–14 years, CBT delivered with a stronger focus on parental involvement than is usually the case with adolescents is effective in reducing symptomatology[67] at least in the short-term. Success has also been reported for similar treatment provided in a group format. Group CBT is as effective as sertraline, and shows better results than sertraline at 9 months follow-up.[68] The presence of tics does not reduce the effectiveness of CBT in the treatment of OCD.[69] There are few studies investigating the longer-term effect of CBT on OCD. However it has been shown that improvement after both individual and group therapy is maintained for at least 18 months without attenuation.[70]

Application of CBT for miscellaneous purposes

There are a number of other conditions and adverse psychosocial situations occurring in childhood and adolescence in which the use of CBT is an important component of management. For a further discussion of these conditions and their management, see other sections of this book.

Chronic fatigue syndrome (CFS)

In this condition, characterized by severe fatigue and overwhelming exhaustion, with excessive sleepiness and a variety of other unexplained physical complaints, cognitive distortions involving an enhanced tendency to believe in the presence of disease in the absence of medical evidence (illness attribution), and deficits in the use of problem-solving techniques related to illness and disability have been identified.[71] The illness is not uncommon, occurring in around 2 per 1000, 11 to 15-year-olds. Rehabilitative methods, including the use of CBT have been found to be successful in adults and are also employed in the paediatric age group. A controlled clinical trial has found 10–17-year-olds with CFS to show greater improvement with CBT than a waiting list control group.[72]

Substance abuse

Cognitive distortions in young people presenting with substance abuse commonly relate to denial they have a serious, ultimately life-threatening problem, unwillingness to believe that effective help is available, and lack of belief in their own self-efficacy to change their behaviour. There is increasing evidence from controlled clinical trials that cognitive behaviour therapies can achieve positive results.[73] Motivational interviewing preceding the use of CBT is important with many children and adolescents and this is likely to be particularly the case with those suffering from substance abuse.

Eating disorders

Central features of both anorexia nervosa and bulimia nervosa include distorted cognitions about shape and weight. Cognitive behavioural approaches used with adults with these conditions require modification when used with adolescents, with greater emphasis on involvement of parents.[74] While CBT is the most effective treatment for bulimia in older adolescents and adults,[75] family counselling is now established as the most effective intervention for anorexia nervosa in younger patients.[76]

Post-traumatic stress disorder

This condition is characterized by disorders of thinking including repetitive, intrusive thoughts, phobic avoidance of the situation in which the individual was exposed to trauma, 'survivor guilt', and problems in concentration. CBT is the most effective, evidence-based technique in the management in both children and adolescents.[77,78]

Non-organic pain

Abdominal pain and headache for which no physical cause can be found are commonly seen in primary health care. Although when these conditions occur it is often difficult to establish a psychological mechanism for the pain, it is reasonably well established that management based on CBT is the most effective approach. CBT is also effective in reducing pain from organic disease as well as in reducing distress when painful paediatric procedures are carried out. For a review of the use of CBT in the management of pain in childhood, see McGrath and Goodman.[79]

Adverse psychosocial situations

Children and adolescents in adverse psychosocial situations are frequently troubled by distorted perceptions of their predicament. In particular, they may feel themselves responsible for the separation and divorce of their parents or that they have deserved the maltreatment, either physical or sexual, inflicted on them by adults who have abused them. CBT has a significant part to play in helping children adjust to parental separation and divorce.[80] It has also been shown to have demonstrable value when applied to victims of sexual abuse.[81]

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9.5.4 Caregiver-mediated interventions for children and families

Philip A. Fisher and Elizabeth A. Stormshak

This chapter summarizes interventions that have been developed to address child and adolescent behaviour problems and externalizing disorders within the therapeutic milieu of the family. Although it has long been recognized that caregiver-mediated treatments can be employed to address children's problems, research with families in the past two decades has resulted in numerous systematic, theory-driven approaches that have been subjected to rigorous scientific evaluation and have been found to be effective at improving outcomes. Although no intervention is certain to work for every child and it is not possible to engage every family in the intervention process, caregiver-mediated interventions are among the most promising approaches currently available to practitioners.

In recent years, progress in the field of caregiver-mediated interventions has included an expansion of the evidence base supporting specific intervention practices for use with the general population, with high-risk segments of the population (e.g. children in foster care and children in Head Start settings), and with underserved populations (e.g. girls and racial/ethnic minorities). In addition, an increasing emphasis has been placed on the dissemination of proven interventions on large-scale bases within community settings in North America, Europe, and Australia. Evidence is currently being gathered to evaluate the impact of many of these large-scale dissemination efforts. The chapter that follows contains background information on the theoretical underpinnings of caregiver-mediated interventions to address child behaviour problems. Specific interventions that have been developed for children in specific age groups—prenatal through early childhood, the school-age period, and adolescence—are then described. Finally, we discuss adaptations that have been made to address issues of gender and cultural diversity within this field.

Before providing a background on caregiver-mediated interventions, a disclaimer is necessary. The term 'caregiver-mediated' is employed throughout this chapter, rather than the term 'family-mediated', to convey the sense that these interventions need not occur specifically within the context of the child's biological family. Recognition of the diversity of family types in which children are raised requires a shift from a nuclear family conceptualization to include multigenerational families, lesbian/gay/bisexual families, and other nontraditional family configurations. In addition, many children are reared in contexts that include no direct biological relatives. For instance, increasing numbers of children are reared in foster care. To a certain extent, this is indicative of the need to address and prevent child maltreatment and to provide services that allow children to remain in their biological families. In addition, it represents a positive development to the extent that many children who have previously been cared for in institutional settings are now being placed in community families, which have the potential to provide more adequate rearing environments than institutions. However, it is often the case that caregivers in these foster/adoptive families will require additional support services to improve outcomes for children. Thus, we have adopted the term caregiver-mediated interventions to reflect the spectrum of existing rearing environments.

Background on caregiver-mediated interventions

There is no single predominant cause of the development of behaviour problems in an individual child. Rather, as is noted in Patterson *et al.*[1] research has implicated a broad array of factors that contribute to behaviours at virtually every level of analysis. From a *societal perspective*, factors such as poverty, discrimination, and unemployment have been implicated as contributing to higher rates of disruptive behaviour in children. In addition, children in various underprivileged contexts (e.g. children in foster care or with incarcerated parents) show higher rates of disruptive behaviour. *Neighbourhood factors* such as crowded living conditions and violence also appear to be associated with higher rates of behaviour problems. At the *individual level*, psychosocial and neurobiological factors have been associated with higher rates of behaviour problems. For example, poor social skills, and low cognitive functioning appear to be linked to the development of behaviour problems. Recent evidence also indicates that genetic factors play a role in whether or not a child develops externalizing behaviour.[2]

With this multitude of factors implicated in the development of disruptive behaviour, one might question why caregiver-mediated interventions have become so predominant. The answer is straightforward. In as much as children exist within the context of their families, caregivers exert the single largest influence on children's behavioural and developmental outcomes. Numerous longitudinal studies have shown that caregiver factors predict over and above individual-, neighbourhood-, and society-level variables in determining trajectories towards disruptive behaviour.[1] Moreover, randomized trials of caregiver-mediated interventions have provided information that manipulating caregiver behaviours (e.g. teaching them to use effective parenting strategies) is extremely powerful for improving child outcomes.[3] Thus, although child behaviour problems are multidetermined, the most direct method for improving child outcomes appears to be via the caregiver–child relationship.

The interventions that are described below are categorized by developmental epoch. It is also important to recognize that interventions exist across the spectrum of risk, including proven interventions to reduce problem behaviours in children with disruptive behaviour disorders and preventive efforts to deflect children from developing disruptive behaviour disorders. An overall synopsis of the interventions to be described is that they have shown great promise for addressing problems at a number of levels. Although a number of issues still confront the field, the progress that has been made in the last two decades has been remarkable in demonstrating that it is possible to address child behaviour problems through caregiver-mediated interventions.

Prenatal and early childhood caregiver-mediated interventions

Caregiver-mediated interventions for the prenatal period through infancy and early childhood are generally oriented towards

prevention. That is, rather than addressing concurrent problems with the child, they are based on the supposition that targeting known precursors of child problems is an effective way to prevent those problems. Among the most influential work in this area has been the home visitation programme developed and evaluated by Olds and colleagues to prevent antisocial behaviour in children.[4] This project involved a randomized control trial that included longitudinal data collection over more than 25 years.[5] A total of 400 pregnant women were enrolled in the original study according to one of the three following criteria: under 19 years of age, unmarried, or of low socioeconomic status. Those assigned to the intervention condition received an average of 9 home visits during pregnancy and an average of 23 home visits between birth and the child's second birthday. The home visits were conducted by nurses. The focus of visits was upon prenatal and neonatal maternal health behaviours, child care skills, and maternal life issues (e.g. education and employment).

Olds and colleagues reported 15-year outcomes for the children of the mothers in this study. Those in the intervention group reported fewer arrests than those in a comparison group.[6] In addition, among those in higher risk categories (as indicated by their mother being both low SES and unmarried at the time of the child's birth), youth in the intervention group reported lower rates of running away, arrests, criminal convictions and parole violations, and smoking and alcohol consumption than did youth in the comparison group. The authors concluded that this approach may be an effective means of preventing early-onset conduct disorder, which has been considered the more treatment-resistant and complex form of the disorder.[1] However, it is also noteworthy that, in subsequent randomized trial studies to examine how variations in the programme structure affect outcomes, the intervention was found to be less effective when paraprofessionals were used in place of nurses as home visitors.[7] More recently, and consistent with other evidence-based programmes, the emphasis of Olds and colleagues has shifted to a wide-scale dissemination of the intervention in community settings.[8] Within this context, emphasis has been placed on maintaining the programme's effectiveness by developing practices to ensure that the intervention is delivered with high fidelity to the original model.

It is important to recognize that not all caregiver-mediated preventive interventions for children in this age group have shown positive effects. For example, the Healthy Start programme was designed to prevent child abuse and neglect, using an initial screening process (usually in hospital settings) to identify families at risk for child maltreatment and employing a home-visitor model of service delivery for the intervention. Although modest positive effects were observed in individual sites implementing this programme, the overall results from a large-scale randomized trial of this intervention did not support the efficacy of the intervention.[9] A related problem in the evaluation of the Healthy Start programme was low rates of family participation. Of those recruited, many received very few home visitation sessions. With such low dosage rates, it can be difficult to determine whether the lack of positive intervention effects was due to a failure of the approach to impact targeted behaviours or was due to families receiving too little of the intervention for it to have been effective. Thus, as is true across the span of child development, it is important to consider what the critical elements of effective interventions are and to make sure that these are included in any intervention efforts.

Caregiver-mediated interventions in the preschool years

The preschool years are marked by a number of important developmental changes. Dramatic increases in the use of language and physical mobility in addition to an increase in autonomous behaviour provide challenges for many parents. Preschool children need substantial support from parents and caretakers in socialization in their family and school environments. As a result, as with interventions in infancy, the majority of caregiver-mediated interventions at this age are focused on parenting, and the parent–child relationship.

Within the parenting literature, there are many different types of parenting skills that have been found to be important in promoting healthy child development. Typically, interventions include a dual focus on increasing positive parenting and decreasing negative parenting. Positive parenting usually refers to supportive, warm, involved parenting that includes praise, positive support, approval, and responsiveness to children and their needs.[10] Negative parenting usually refers to parenting deficits, including poor limit setting, inconsistency, verbal and physical aggression, and harsh discipline.[11]

Caregiver-based interventions at this age typically focus on building a strong positive parent–child relationship in addition to teaching specific behaviour management techniques to promote healthy child adjustment and prevent later problems. Although some programmes target at-risk children, such as Head Start children or those with early behaviour problems, there is some evidence that more severe child behaviour problems are related to more limited effects of parent training programmes.[12]

One of the more successful caregiver-based programmes for preschoolers, *The Incredible Years*, was developed by Webster-Stratton and colleagues.[13] This videotaped programme includes a number of salient parenting skills for preschoolers, starting with building a positive parent–child relationship, praise, and rewards and moving to limit setting, problem solving, and discipline. Randomized trial evaluations have suggested that this programme is effective when administered in group and individual settings.[14] Results have suggested that the programme improves parent–child interactions immediately post-test and 1 year later. The programme was subsequently expanded from basic parenting skills to include more advanced parenting, such as anger management, communication, and self-control skills, which is also effective at improving the parent–child relationship and reducing child behaviour problems at short-term follow-up. The advanced programme is able to deal effectively with parent–child problems and the mediators that might influence the parent's ability to effectively manage the child, including depression and the marital relationship. Overall, this parent training programme has been effective with a number of different age groups and populations, including preschool- and school-aged children as well as clinic-referred and community Head Start families.[15]

Other caregiver-mediated interventions involving preschoolers have also shown success at enhancing the parent–child relationship and decreasing child behaviour problems. For example, *Parent–Child Interaction Training (PCIT)* was originally developed for at-risk children enrolled in Head Start.[16] Training involved teaching parents effective play skills and positive interaction. The programme has been effective at reducing teacher ratings of behaviour

problems 1 year after the intervention was completed. This approach has subsequently been used for other populations. For example, Chaffin *et al*. reported on the results of using *PCIT* to prevent child abuse among families with a history of child welfare system involvement.[17] The intervention significantly reduced future reports of maltreatment. Interestingly, an enhanced version of the intervention designed to provide individualized services to meet families' needs was no more effective than the original *PCIT* intervention.

Home visitation programmes in preschool have also shown promise as effective interventions. Head Start, for example, provides home-visiting services to all families in the programme. The goals of these visits are to work with families on meeting the Head Start performance standards in four areas: education, health–nutrition–mental health, social services, and parent involvement. Research has indicated that children who received home services have shown improvements in the parent–child relationship and early academic achievement compared to children and families who did not receive the home visits or parenting model.[18] Home-visiting programmes at this age also provide families with social support, self-efficacy, and a positive therapeutic relationship with the visitor. This relationship serves to enable parents to process and understand parenting and family histories that impede the development of successful parenting skills with their own child.

Caregiver-mediated interventions in the school-aged years

As children move into the school years, they face a new set of challenges at home and in the school environment, including academic achievement, negotiating peer relationships, and the demands of teachers and the school context. At this age, children who have begun developing problems in the context of their families may generalize these problems to the school environment, which is associated with increased risk of later difficulties in addition to problems across multiple domains of functioning.[19] As a result, the transition to school serves as an important target of preventive intervention programmes.[20] Furthermore, school problems and peer difficulties may exacerbate problems in the home as parents struggle with issues such as homework completion and their child's social skills and changing peer network. Thus, effective interventions aimed at school-aged children must support families and parents through an emphasis on parent training and improving or maintaining academic achievement and positive peer relationships at school.

Although the emphasis on increasing positive parenting skills and decreasing negative parenting is typically maintained in interventions at this age and across development, additional components targeting academic achievement, learning and early literacy, and parent–school involvement are important aspects of comprehensive, caregiver-mediated interventions for school-aged children. There are multiple examples of comprehensive, caregiver-mediated prevention programmes that have been associated with positive outcomes for school-aged youth and families, including the *Families and Schools Together (FAST) Track* programme,[21] the *Linking the Interests of Families and Teachers (LIFT)* programme,[22] and the *Schools and Families Educating (SAFE) Children* programme.[23]

The *FAST Track* programme began in 1991 and targeted children in early elementary school at risk of developing later conduct disorder and delinquent behaviour problems.[21] Children and families received a multifocused intervention package targeting development across multiple domains, including peers, the school environment, academic achievement, and the family context. The family intervention integrated successful approaches to parent training regarding the development of school-aged children, including parent–school involvement and early reading.[21,24,25] Parents met in groups weekly during the first-grade year and bi-weekly in second grade. One hour of parent–child learning activity that emphasized positive parent–child interactions in a controlled environment and early literacy was also provided. Home visits and individualized programming were implemented to meet the special needs of families, such as stress management, marital problems, and maternal depression. The intervention continued through adolescence, with new components adapted to the changing development of children and families. Results of this programme indicated that it was successful at the end of first grade and third grade in improving outcomes across a number of different domains, including parenting and child peer relations, emotional understanding, and reading skills. Specifically, parents showed less physical discipline, more consistent and appropriate discipline, and more warmth and positive involvement in their child's school.[24,26,27]

The *LIFT* programme was designed as a preventive intervention for at-risk children to decrease the development of conduct problems and delinquency.[28,29] *LIFT* was designed for first- and fifth-graders living in at-risk neighbourhoods. Intervention components consisted of a school-based intervention focused on social skills and problem solving, a parent training group, a playground behavioural programme, and communication between parents and teachers. Creative intervention techniques were employed to increase parent participation in schooling; for example, a phone answering machine was installed in each classroom, and newsletters were sent home to parents about school *LIFT* activities. Parents could call in to the answering machine or leave messages for teachers at any time. This programme was successful at decreasing aggression on the playground and increasing positive behaviours with peers during the year following the intervention. Additionally, the programme was successful at decreasing negative parenting during observed mother–child interactions.[28]

The *SAFE Children* programme was administered within different schools in high-risk neighbourhoods in the Chicago area with a diverse group of families.[23] Youth were randomly assigned within classrooms to receive the intervention, which included parenting and academic tutoring components. The parenting component focused on disseminating information about child development, parenting, skill practice, and home assignments to increase parenting skills and on group problem solving around skills when parents needed additional assistance with implementation. The intervention targeted children during the transition to elementary school, based on developmental research and theory suggesting that transition points are key points for intervention.[30] The intervention significantly impacted academic performance and parental involvement in school, with additional outcomes for high-risk families that included higher parental monitoring and reductions in child behaviour problems.[23]

In summary, effective school-aged, caregiver-mediated interventions for families are multifocused, typically including foci on parenting skills and school success for children. The majority of

successful interventions at this age combine a family component with additional interventions to support school adjustment (e.g. social skills training, academic tutoring). Clearly, interventions that target parenting, families, and other domains of children's functioning are the most efficacious interventions to administer during the transition to school.

Caregiver-mediated interventions involving adolescents

By adolescence, many youth have achieved a degree of independence from their parents and are embedded in the culture of peers, school, and/or their community. The extent of adolescents' autonomy in today's world is such that there may be a temptation to consider addressing adolescent mental health problems in a different manner than those of younger children. However, among the interventions that have demonstrated the biggest impact for adolescents are those focused on youth in the context of their families.[31] The only difference between these programmes and those discussed for younger children is that there is an increased emphasis on parenting skills that are more relevant to adolescent youth, such as problem solving, helping your adolescent gain autonomy, and communication. Adolescents may be more active participants in the interventions that target this age group, attending family sessions and practicing the skills outside of the treatment at home or at school.

There have been a variety of programmes associated with positive outcomes for adolescent youth. At this age, effective interventions tend to be brief, family-centred approaches that teach parents the skills needed to parent effectively during adolescence. Caregiver-mediated interventions at this age may be delivered in the school context or in a community centre or clinic. Within the school context, several programmes have shown success at decreasing adolescent problem behaviour, including substance use over time.

The *Adolescent Transitions Program* (ATP) is a caregiver-mediated treatment for adolescent problem behaviour and substance use. Over the past 15 years, the *ATP* has been shown to be effective in a number of different randomized control trials with parents recruited from the community and schools.[32,33] The *ATP* curriculum focuses on building a positive parent–adolescent relationship, decreasing known risk factors at this age for problem behaviour (e.g. lack of parental monitoring), and increasing communication and listening skills. In the first series of research studies using the *ATP* , the intervention was delivered in a group format and was successful at reducing problem behaviour.[34] More recently, the *ATP* curriculum has been developed into a caregiver-mediated, tailored approach to treatment called the *Family Check-Up* (*FCU*).[31] The *FCU* is a preventative intervention based on a health maintenance model appropriate for at-risk and high-risk youth. The *FCU* incorporates the content from the *ATP* curriculum into a model that targets parent engagement in treatment. In the *FCU* model, families receive a comprehensive, ecological assessment, videotaped observation, and feedback using motivational interviewing to engage families in treatment. Across numerous randomized controlled intervention trials, the *FCU* has been shown to be effective at reducing teacher-reported risk behaviour, reducing arrest rates, increasing attendance and achievement at school, reducing substance use, and reducing antisocial behaviour.[35–37] Interestingly, these outcomes have been mediated by an increase in family management skills, including parental monitoring.[36]

There have been several other comprehensive, caregiver-mediated approaches to the treatment of adolescents that have also shown positive outcomes. Many of these models are school-based and preventive, focusing on at-risk and high-risk youth and families via group format from schools. Spoth *et al.*[38] examined outcomes associated with both the *Preparing for the Drug Free Years* programme[39] and the *Iowa Strengthening Families Program*.[40] Both of these parenting programmes have been hypothesized to reduce the initiation of substance use in the high school years and to reduce antisocial behaviour and were both strength based and focused on teaching parents the skills necessary to prevent adolescent problem behaviour. Schools were randomly assigned to deliver the interventions at the school level as universal programmes. Results suggested that these programmes were associated with decreases in the growth of substance use from 6th through 12th grade at the school level. Interestingly, both programmes were brief (up to seven sessions), and effects were found all the way up to 12th grade. These results are promising because they suggest that caregiver-mediated interventions in adolescence can be delivered as universal school-based curricula to decrease problem behaviour in the school population.

The intervention programmes discussed previously are appropriate for typically developing youth, at-risk youth, and high-risk youth. Unfortunately, adolescent youth who have had serious problems with behaviour since early childhood may need caregiver-mediated support that is more intensive and focused around their specific problems. One intervention programme that is particularly promising for high-risk youth is *Multidimensional Treatment Foster Care* (*MTFC*).[3] *MTFC* is an alternative to institutional treatment for youth with severe emotional and behavioural problems, including those involved with the juvenile justice system. Within the *MTFC* intervention model, youths are placed with foster parents who have received specialized training and who receive a high level of support and supervision from professional staff during the placement. The *MTFC* approach includes the use of highly structured behaviour management programmes as a component of the treatment plan implemented in the foster home. This structure provides the youth with the opportunity to practice the skills that the foster parents are attempting to develop while adjusting the youth's access to risky situations at a rate that matches the youth's progress in treatment.

While the youth is in foster care, his/her biological parents receive intensive behavioural parent training. As the youth shows signs of progress in the foster home, he/she is allowed home visits. The duration of these visits is gradually increased, until the child is ready to return home. An extensive aftercare programme ensures that gains are maintained following reunification.

The *MTFC* approach was evaluated via a randomized control trial and was found to reduce recidivism, especially in comparison to group care.[41–43] Moreover, Chamberlain and Reid reported that youth in the *MTFC* programme had lower incidences of substance use and less contact with delinquent peers than youth in institutional settings.[3] Subsequently, variations of the *MTFC* programme have been developed for a variety of populations, including preschoolers[44–46] and adolescent girls.[47]

Mediators of intervention efficacy

There are many commonalities among the intervention programmes discussed in this chapter. First, each programme is

grounded in developmental research and theory suggesting that families are central to the process of intervening in the mental health issues of children and adolescents. Transitions are critical times for intervention, including transitions to school and puberty.[48] Each programme is strength-based, focusing on increasing protective factors for children and reducing risks in the environment. Lastly, each programme is multifocused, targeting parenting skills and other important factors in a child's life that impact development (e.g. academic achievement).

The literature in this area has suggested some common themes in the mediators that impact the success of interventions for families. First, recruiting parents into treatment can be difficult.[49] Heinrichs *et al.* examined recruitment issues by examining participation in parent training groups in their sample of about 600 families using the *Triple P* parenting programme.[50] They were only able to recruit 31 per cent of low-income families into their parenting groups but were able to retain 77 per cent of families once they began treatment, which included the retention of high-risk families. Similarly, Brody *et al.* found that having more children in the household and youth risk-taking behaviour was related negatively to attendance and engagement in their parenting intervention.[51] Recruitment and attendance issues have been a constant struggle in parenting interventions and have led to the development of brief parenting interventions[52,53] and more tailored, individualized approaches to family treatment.[31] For many families, issues such as childcare, work schedules, and the time commitment required to attend parenting groups prohibit participation. In addition, a parent's interpersonal problems, such as depression and parental resources, can impact participation and outcomes associated with family-based interventions.[54,55]

Second, *culture* plays a key role in the efficacy of family interventions. Parenting skills are inextricably connected to cultural values. For example, racial differences are evident in the literature linking parenting practices to child behaviour problems.[56–58] In previous decades, there was an emphasis on cultural sensitivity within interventions targeting diverse groups. Currently, interventions must be not only culturally sensitive but also adapted to meet the needs of the community in which they are implemented.

Several interventions for families have attempted to address the issue of culture by providing ecologically valid and culturally sensitive interventions to ethnic minority groups in the United States. For example, the *Effective Black Parenting Program* integrated a cognitive behavioural parent training programme with information of relevance to inner-city African-American families, including discussions of traditional discipline (physical punishment) versus modern discipline (internalizing standards of behaviour).[59,60] Components such as helping the child deal with racism at school and positive communication about ethnicity were also included. This programme has been effective at changing parenting behaviour and child behaviour 1 year later.

Another example may be found in the work of Szapocznik and colleagues, who have developed a programme to engage Latino families in treatment that includes less traditional forms of engagement and more of an emphasis on the ecosystems of families and the cultural context. These researchers have hypothesized that resistance to change occurs during the initial stages of therapy and that traditional forms of engagement may not be successful within the Latino population. Instead, engagement includes joining and encouraging the family to participate in home visits and meetings with significant family members. This type of engagement strategy has been effective in retaining families in treatment with the final goal of reducing drug use and other problem behaviours in adolescence.[61–63] Although these programmes serve as examples of specific approaches to working with populations of different cultures, intervention research is just beginning to emphasize these differences and integrate approaches to culturally diverse families into more traditional caregiver training curriculums. More work in this area is clearly necessary, both inside and outside of the United States.

Summary and conclusions

In this chapter, we have attempted to provide a framework for understanding caregiver-mediated, developmentally based interventions for parents and families and to provide examples of interventions that fall within that framework. As is noted previously, the field of caregiver-mediated interventions and the variety of interventions available is a much broader topic than has been addressed here. We hope that the information presented here serves both to inform about the specifics of certain interventions and to organize the study of the larger field.

Further information

http://www.oslc.org/index.html
http://www.mtfc.com/index.html
http://cfc.uoregon.edu/
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9.5.5 Medication for children and adolescents: current issues

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Introduction

Problems of mental health and behaviour in children are multidisciplinary in nature and optimal treatment is often multimodal. This article focuses on aspects of psychopharmacology that has special relevance in children and adolescents, especially the recent controversies. In general, this article provides information about classes of medication and not detailed information about specific medicines. Treatment recommendations of the specific disorders have been dealt within the appropriate chapters.

The use of psychotropic medication in children is higher in the United States than in many other countries, and polypharmacy is common. About 1 per cent of overall medical consultations visits by children and adolescents in 2003–2004 in the US resulted in a second-generation antipsychotic (SGA) prescription. The majority of the visits involving antipsychotics were by Caucasian boys aged over nine years, visiting specialists, without private insurance, with a diagnosis of bipolar disorder, psychosis, depression, disruptive disorder, or anxiety.[1]

Pre-school (2 to 4 year olds) psychotropic medication use, between 1995 and 2001 increased across the US for stimulants, antipsychotics, and antidepressants, while the use of anxiolytics, sedatives, hypnotics and anticonvulsants remained stable across these years, suggesting non-psychiatric medical usage.[2] Ethnicity may influence differential prescription rates; for example, as compared to Caucasian youths, African-American youths are less likely to be prescribed psychotropic medications especially methylphenidate.[3]

Information assisting psychopharmacological decision-making

Apart from a thorough diagnostic assessment, a full medical history including present, recent, and past prescribed and over-the-counter medication, response to treatment, and attitude towards interventions play a major role in deciding treatment. History of substance misuse needs to be elicited to ascertain potential medication-misuse liability and because certain medication significantly interact with illicit drugs. A family history of mental illness, suicide, substance abuse, neurological or medical conditions, especially early onset coronary artery disease, hyperlipidemias and diabetes, and specifically the response of family members to psychotropic medication are all important.

Medication as a part of multimodal treatment package

Disorders that have an extended course, where emergence of new problems is common, require continuous, dynamic treatment planning and monitoring to ensure effectiveness of the current treatment. The treatment should stress multi-modal intervention and address co-morbid psychiatric disorders. Treatment plans

should be individualized according to the pattern of target symptoms and strengths identified in the evaluation. A thorough functional analysis of problems or symptoms is central to pharmacological decision-making. Treatment should target situations in which symptoms cause the most impairment. Custom-designed target symptom scales or daily behavioural report cards are useful in monitoring treatment progress.

One way to conceptualize paediatric psychopharmacotherapy is the 'Symptom-based Approach' for core symptoms as follows:

- *Symptoms that require and are likely to respond to medication alone*: inattention, impulsivity, hyperactivity, tics, obsessions, psychotic symptoms, labile mood etc;
- *Symptoms less likely to respond to medication alone, requiring both medication and psychosocial interventions*: aggression, rituals, self-injury etc;
- *Symptoms that are unlikely to respond to medication that need specific remediation or rehabilitation:* skill deficits in academic, social, or sports domain.[4]

Psychosocial interventions may be required to address either primary or secondary relationship problems associated with the core deficits or to deal with co-morbidity.

The 'art' of prescribing medication

Pharmacological interventions do not necessarily work exclusively because of their 'neurochemical' effects. Response to medication also includes the inherent 'placebo response' or 'expectancy effect' that prescribing can induce, as well as the effect of the 'therapeutic concordance' achieved through getting the agreement and acceptance of why the medication is being prescribed and also what is the expected response.

Parents as well as patients respond better when they feel understood, accept why treatment is necessary and are in agreement with the prescriber regarding the need for treatment. Simple strategies help this process: collaborating with parents, patients, school and care providers; giving a clear explanation of diagnoses, of why medication (with or without psychosocial interventions) is necessary; setting realistic expectations (for example, aiming for a 40–60 per cent reduction in anxiety symptoms in a child with multiple co-morbidity); keeping track of the larger systems of care at school and home; providing clear, appropriate information sheets, websites etc to obtain further information if needed; prioritizing and tracking target symptoms; asking them to 'opt in' to treatment after having weighed all the pros and cons, as opposed to them perceiving that they are being 'told' that they 'have to' start medication; using short telephone-based medication monitoring during the stabilization phase in order to pick up emerging side-effects and monitoring dosage accordingly; initiating medication using small doses and titrating it up over a period of 4 to 6 weeks, to identify the **minimum effective dose (MED)**, which is the minimum dose with which 'acceptable' improvement with minimal side-effects is achieved; involving school in monitoring symptoms regularly even if it means maintaining a school-home dairy; and willingness to change treatment if the expected outcome has not been achieved.

Domains that each therapy focusses on should be clearly documented and periodically evaluated. The designation of a case manager is essential for chronically disabled individuals to coordinate the wide range of services necessary for their care and to ensure periodic diagnostic reassessments.

Developmental issues, pharmacokinetic and pharmacodynamic factors affecting pharmacotherapy in children

Generally, drug response may vary with age, weight, sex, disease state, absorption, distribution, metabolism, and excretion. Thus, developmental factors that influence these are important to consider. Although the extent of drug absorption for most medication is similar in children and adults, the rate of absorption may be faster in children and peak levels are reached earlier.[5]

Absorption is also dependent on the form in which it is administered, i.e. liquid versus tablet, and levels peak faster for liquid preparations. Generally speaking, hepatic metabolism is highest during infancy and childhood, 1 to 6 years, approximately twice the adult rate in pre-puberty at six to 10 years, and equivalent to adults by the age of 15.[5] This is clinically important as younger children may require higher mg/kg doses of hepatically metabolized medications than older children or adults.[6]

Adolescence is a period of particularly high ketosteroid levels, which have significant impact on brain neurotransmitter systems. A transient decrease in metabolism for some medication has been reported in a few months before puberty, which is believed to be due to the competition for hepatic enzymes with sex hormones.[7]

Fat distribution varies in children raising during year one then gradually falling until puberty and increasing with obesity. Substantial fat stores slow elimination of highly lipo-soluble drugs from the body (e.g. fluoxetine and pimozide).

Protein binding and volume of distribution affect the pharmacokinetics of medications. These parameters differ in children and have practical clinical implications such as the fraction of the drug that is active and unbound.[8] This is especially a factor when medicating children with eating disorders such as anorexia.

Overall, it is recognized due to the various factors covered in this article, non-pharmacological strategies are more effective in preschoolers. Pharmacotherapy in school-going children has a reasonable risk/benefit ratio and older adolescents behave more like adults. As patients mature, treatment plans often must be adapted to change according to the changing individual, family, and environmental conditions.

Antidepressant efficacy

The poor antidepressant response in childhood depression may have its basis on differences in the hormonal milieu of the brain, and incomplete maturation of the neurotransmitter systems involved in the control of affect, inclusion of adolescents who will over time become bipolar, adolescents with depressive phenocopies, and possible differences in pharmacokinetics and pharmacodynamics. The more rapid hepatic metabolism of imipramine and amitriptyline results in noradrenergically active metabolites, shifting the ratio of the noradrenergic to serotonergic activity of these compounds in children to a ratio higher than that seen in adults. This activity shift is significant because the noradrenergic system does not fully develop both anatomically and functionally until early adulthood.[9,10]

Cardiotoxicity

The maturation of vagal modulation of heart rate increases during the first decade of life, peaks sometime during the second decade,

and declines gradually with age through the sixth decade of life. Sympathetic modulation follows a similar pattern, but rate of maturation of the two branches differ. Furthermore, there is considerable variation between individuals of similar age in autonomic maturation. The relative loss of vagal modulation associated with tricyclic antidepressants may be accentuated in some younger subjects because of these maturational factors, leading to cardiotoxicity.[11]

Stimulants

Stimulants have been used for decades and good research evidence exists for their short-term use in ADHD. More recently, various stimulant delivery systems have been developed (the osmotic controlled-release system (OROS) – Concerta XL®; the wax-matrix-based beaded system – Metadate CD®; the patch release system – Daytrana®; etc) resulting in long-acting preparations which makes it possible to avoid medication needing to be administered in school. This once daily dosing schedule possibly reduces stigmatization and embarrassment. The release systems and preparation of stimulants (immediate release / slow release ratios) allow the tailoring of the long-acting preparations to suit the need of individual children.[12]

Precautions with stimulants

Stimulants are contraindicated in schizophrenia, hyperthyroidism, cardiac arrhythmias, angina pectoris, glaucoma, or a history of hypersensitivity to drug. They can be used with caution in those with hypertension, depression, tics (or family history of Tourette's syndrome), pervasive developmental disorders or severe intellectual disability. Occasionally tics can be made worse with stimulants.

(a) Rebound effects

It consists of increased excitability, activity, talkativeness, irritability and insomnia beginning 4–15 h after a dose. It may be seen as the last dose of the day wears off or for up to several days after sudden withdrawal of high daily doses of stimulants. This may resemble a worsening of the original symptoms and is encountered frequently by clinicians. Management strategies include increased structure after school, addition of a smaller dose of medication in the late afternoon, use of long-acting formulations or the addition of clonidine or guanfacine to the regime.

(b) Seizures

Methylphenidate can be used in the presence of well-controlled epilepsy. If seizures worsen or emerge during treatment, methylphenidate should to be changed to dexamfetamine, which is supposed to increase seizure threshold.

(c) Growth retardation

The MTA study indicates that there is significant growth reduction with stimulant use.[13] It would be advisable not to start stimulants in children who are short and are biologically predisposed to short stature (e.g. short parental stature, growth hormone deficiency etc.).

(d) Cardiac problems

Stimulants may increase pulse rate and rarely can lead to increased blood pressure. Importantly, African-American male adolescents may be at a higher risk from mild chronic elevation in blood pressure on methylphenidate, needing more rigorous blood pressure monitoring for hypertension than their Caucasian counterparts.[14] After several reports of death of patients on Adderall®, there is currently a warning to clinicians to be aware of possible cardiac side effects, especially in the presence of known cardiovascular illness. The reported rates of sudden death on Adderall® do not exceed reported rates of sudden death on other stimulants or the base rate of sudden death per age group in general off medication.

(e) Abuse potential of stimulants

There is little evidence that substance misuse or dependence results from the prescription of stimulants for ADHD. Self initiated increase in dose by emotionally unstable adults with substance use disorders is possible and needs to be suspected in those who repeatedly claim to have lost medication or in parents who repeatedly insist that higher doses are necessary to control symptoms, when the child is functioning well in other settings. In such situations it may be better to use long-acting preparations such as Concerta XL®, as the delivery system makes it difficult to abuse. Other drugs that are useful in this setting are atomoxetine and bupropion.

(f) Psychotic symptoms

Post-marketting surveillance led to enhanced labelling warnings regarding psychosis, mania and hallucinations as adverse events (*http://www.fda.gov/ohrms/dockets/ac/06/minutes/2006-4210m_Minutes%20PAC%20March%2022%202006.pdf*). Many such drug-related psychiatric adverse events may be self-limited and resolve with drug cessation. Stimulants are better avoided in those who have a first degree relative with a psychotic disorder or in children who have psychotic or quasi-psychotic experiences.

Non-stimulants

Atomoxetine is a non-stimulant noradrenaline reuptake inhibitor, which has a high affinity and selectivity for the noradrenaline reuptake site over serotonin and dopamine transporters. In extensive metabolizers (EMs), inhibitors of CYP2D6 e.g. *paroxetine, fluoxetine and guanidine* increase atomoxetine steady-state plasma concentrations. Atomoxetine is long acting and can be used once a day and does not result in rebound symptom worsening and has little potential for abuse. It does not worsen tics, anxiety or low mood and hence may be useful in some children with ADHD and co-morbid disorders.

(a) Precautions with atomoxetine

Atomoxetine is contraindicated in hepatic insufficiency/impairment, glaucoma, uncontrolled seizures or a history of hypersensitivity to drug. They can be used with caution in those with hypertension or with any condition that may predispose to it, tachycardia, cardiovascular problems in patients with congenital or acquired long QT or a family history of QT prolongation or with cerebrovascular disease.[15]

(b) Growth retardation

Acute treatment studies show that atomoxetine-treated patients lose some weight. Michelson *et al.* (2004)[16] reported a change of 2–3 percentiles in mean height, which appear to be similar to effects observed in stimulant-treated patients. Patients treated for extended periods should be monitored regularly.

(c) Seizure liability

A review of risks and benefits of atomoxetine in 2996 led to warnings on the risk of seizures when taking atomoxetine. It is not to be

used in uncontrolled seizures and should be discontinued in those who develop seizures or who experience an increase in frequency of their seizures.

(d) Cardiac problems

Atomoxetine increases noradrenergic tone and produces increased heart rate and small increases in blood pressure, which subsides on discontinuing atomoxetine. QT interval prolongation can occur but ECG monitoring is not necessary unless one suspects cardiac problems.

(e) Suicidal risk

In September 2005, a 'black box' warning was added to the product labelling of atomoxetine as a result of an analyses that showed that suicidal ideation was more frequently observed in clinical trials among children and adolescents treated with atomoxetine (5/1357 [0.37 per cent]) compared to those treated with placebo (0/851[0 per cent]) (*http://www.fda.gov/cder/foi/label/2007/021411s004s012s013s015s021lbl.pdf*). There was one suicide attempt in the atomoxetine treated group. No completed suicides occurred during these trials. There was, however, no evidence of increased suicidal thoughts in adults taking atomoxetine. Prescribers should monitor for signs of depression, suicidal thoughts or suicidal behaviour and refer for appropriate treatment if necessary.

(f) Hepatic dysfunction

Reports indicate that atomoxetine can cause severe liver injury in rare cases. The spontaneous adverse event database search identified two cases that were probably associated with atomoxetine use as a cause or contributor to the event. One spontaneously reported case of liver injury (fulminant hepatitis) appeared probably related to atomoxetine therapy by positive re-challenge in a population exposure of about 2.2 million patients within the 2-year period (2002–2004) after approval which is likely to be an underestimate. Less severe liver dysfunction indicated by abnormal liver enzymes is more common and such reactions may occur several months after therapy is started and atomoxetine should be discontinued in patients with jaundice abnormal liver enzyme levels, which should be done upon the first symptom or sign of liver dysfunction (e.g. pruritus, dark urine, jaundice, right upper quadrant tenderness or unexplained 'flu-like' symptoms).

Antidepressants

The pattern of antidepressant use in children and adolescents has changed significantly over the last couple of decades. Tricyclic antidepressants were predominantly being used for ADHD, enuresis, depression, and anxiety disorders during the eighties and early nineties. Over the last 10–15 years, this has changed to predominantly prescribing the newer antidepressants, especially for anxiety disorders and depression, despite little real evidence for its efficacy. Current data suggests that other than fluoxetine, no other antidepressant has evidence to clearly support its use in depression in children and adolescents.

Tricyclic antidepressants

Tricyclic antidepressants include amitriptyline, desipramine, nortriptyline, imipramine, and clomipramine. Historically, they have been used as a second-line pharmacologic treatment for ADHD (though only as an off-licence drug), following stimulant medications. Their use has declined in recent years due to concerns of cardiac arrhythmias and case reports of sudden death in the paediatric population. Drawbacks include potential cardiotoxicity, especially in pre-pubertal children, the danger of accidental or intentional overdose, troublesome sedation, anticholinergic side-effects, lowering seizure threshold and possibly declining efficacy over time.

New generation antidepressants

Fluoxetine, sertraline, fluvoxamine, citalopram, escitalopram, and paroxetine are all specific serotonin reuptake inhibitors (SSRIs) while venlafaxine is a specific noradrenaline and serotonin reuptake inhibitor (SNRI) and reboxetine a specific noradrenaline reuptake inhibitor (SNI) and mirtazapine. Few trials exist in pre-pubertal children with these drugs; however, the data suggest that the SSRIs have reasonable efficacy in severe anxiety disorders such as OCD (fluoxetine, sertraline) but only fluoxetine is effective in depression. Theoretically, the SNIs may help managing symptoms of ADHD but little research evidence exists.

Precautions with antidepressants

(a) Antidepressant-induced behavioural activation

Frequently children prescribed antidepressants (especially those with developmental disorders or intellectual disability) develop increased motor activity, restlessness, excitability and impulsivity. This usually occurs early in treatment and is often misconstrued as being a manic or hypomanic switch and wrongly treated as if the child had a bipolar disorder. This can be managed by reducing the dose and may need the cover of a benzodiazepine for a few days. This side-effect can be reduced by initiating antidepressants in vulnerable children in small doses (about a fourth of the final dose needed) and gradually increasing the dose over a few weeks.

(b) Antidepressant-related suicidal ideation and behaviour

It was realized two decades ago that imipramine was not effective in pre-pubertal major depression.[17] The Committee for Safety of Medicines (CSM) in December 2003 reviewed all the relevant trials with new generation antidepressants on remission, response to treatment, depression symptom scores, serious adverse events, suicide-related behaviour, and discontinuation of treatment because of adverse events and concluded that the evidence was adequate to establish effectiveness only for fluoxetine and contra-indicated all SSRIs (except fluoxetine) for depressed children (*http://www.mhra.gov.uk/home/groups/plp/documents/drugsafetymessage/con019472.pdf*).[18] This was then followed by the FDA asking for black box warnings on all SSRIs warning about the possibility of suicide-related behaviour as a side-effect in depressed children (*http://www.fda.gov/Cder/drug/antidepressants/SSRIPHA200410.htm*). It is currently advised that children or adolescents being started on or dose being increased of antidepressants should be monitored closely for emergence or worsening of suicidal ideation or behaviour.

Antipsychotics

Recent years have witnessed increased antipsychotic treatment of children despite limited long-term safety data in children. Second generation antipsychotics (SGAs) are the most frequently prescribed ones; for example, risperidone, quetiapine, aripiprazole, olanzapine, ziprazidone, and amisulpiride.

Risperidone is the commonest atypical antipsychotic used in children and adolescents to manage psychoses and disruptive behaviour in autism. It is a potent dopamine D2 receptor blocker (hence reduces positive symptoms but produces hyperprolactinaemia) and 5HT-2A receptor blocker (enhances dopamine release in certain brain regions, thus reducing motor side effects and possibly improving cognitive and affective symptoms). The Research Units for Paediatric Psychopharmacology (RUPP) studies have shown that risperidone is effective in managing disruptive behaviours in autism spectrum disorders.[19] Apart from the side effects of sedation and weight gain, hyperprolactinaemia is common and a few develop extrapyramidal side effects such as tardive dyskinesia.

Aripiprazole is dopamine partial agonist or dopamine stabilizer, used in managing schizophrenia and bipolar disorder. The partial antagonism of D2 receptors reduces dopamine output when dopamine concentrations are high, thus improving positive symptoms and mediating antipsychotic actions. Blockade of 5HT-2A receptors may cause enhancement of dopamine release in certain brain regions, thus reducing motor side effects and possibly improving cognitive and affective symptoms. Actions at D3 receptors and partial agonism of 5HT-1A receptors could also theoretically contribute to aripiprazole's efficacy.[20] Even though symptoms may improve in the first week, it is recommended to wait for four to six weeks to determine efficacy due to the pharmacokinetics of the drug. The mean elimination half-life is 75 h of aripiprazole and 94 h for the major metabolite dihydro-aripiprazole and is primarily metabolized by CYP450 2D6 and CYP450 3A4. ketoconazole, fluvoxamine, and fluoxetine all increase plasma levels of aripiprazole, while Carbamazepine decreases plasma levels of aripiprazole. Early experience of aripiprazole suggests that it produces less weight gain, diabetes, or hyperlipidaemia, compared to the other SGAs. It is however less sedating and rather activating. Little published evidence exists currently on its use in managing non-psychotic disruptive behaviour in developmental disorders but clinical experience suggests that very small doses (2 to 5 mg per day) are sufficient.

Quetiapine is an effective SGA with moderate effect on weight, but usually needs to be taken at least twice daily because of relative weak receptor binding. *Ziprasidone* is being increasingly used and is the only SGA that is weight neutral, but has greater impact on cardiac rhythm and QTc interval. *Clozapine* is used in those with resistant psychoses or those with tardive dyskinesia, but can lead to neutropaenia, sialohorrea, and significant weight gain. *Olanzapine* is used less in children and adolescents because of the propensity to weight gain and metabolic syndrome. Evidence from adults suggests that Clozapine, Olanzapine, and low-potency conventional antipsychotics such as chlorpromazine are associated with increased risk of insulin resistance, hyperglycaemia, and type 2 diabetes mellitus.[21,22]

Precautions with antipsychotics

(a) Movement disorders

Tardive dyskinesia and extrapyramidal side-effects are more common with conventional antipsychotics. Ethnicity may be a risk factor for dyskinesia in children as African-American children appear to be more prone to tardive dyskinesia when compared to European-American children.[23] Aripiprazole or clozapine are useful in those who require antipsychotics but have developed tardive dyskinesia.

(b) Weight gain and metabolic dysfunction

Weight gain is a serious side effect of SGAs and potential consequences of obesity include non-compliance with medication and significant morbidity and mortality.

(c) Risk for SGA-induced metabolic dysfucntion

High risk – clozapine, olanzapine

Moderate risk – risperidone, quetiapine, sertindole

Low risk – amisulpiride, aripiprazole, ziprazidone

(d) Baseline information before starting an SGA

Weigh the children and adolescents and track the BMI during treatment; get baseline personal and family history of obesity, dyslipidaemia, hypertension, and cardiovascular disease; children of parents with total cholesterol >24 mmol/l (parents with high BMI), and history of overt heart disease in parent or grandparent at age 55 or younger should be considered to be at high risk; get waist circumference at umbilicus, blood pressure, fasting plasma glucose, and fasting lipid profile.

(e) Monitoring after starting an SGA

BMI monthly for three months then quarterly with blood pressure, fasting plasma glucose, fasting lipids within three months and then annually but earlier and more frequently for patients with diabetes or who have gained greater than 5 per cent of initial weight; treat or refer for treatment and consider switching to another atypical antipsychotic for patients who become overweight, obese, prediabetic, diabetic, hypertensive, or dyslipidaemic while receiving an atypical antipsychotic. Elevated fasting plasma triglyceride or increased insulin levels may be important signals of potential insulin resistance. Increased low density lipoprotein (LDL) cholesterol and decreased high density lipoprotein (HDL) cholesterol is associated with increased adiposity, especially visceral adiposity. Even in patients without known diabetes, one should be vigilant for the rare but life threatening onset of diabetic ketoacidosis, which always requires immediate treatment by monitoring for the rapid onset of polyuria, polydipsia, weight loss, nausea, vomiting, dehydration, rapid respiration, weakness, clouding of sensorium, and even coma.

Treatment of SGA-induced metabolic dysfunction

Careful selection of treatments taking the metabolic-risk profile into account, preventative healthy lifestyle counselling, and regular monitoring of body composition and metabolic variables need to become clinical routine. Total caloric intake is more important than the content of the diet and motivational interviewing and cognitive behavioural techniques can be used to address unhealthy diet, physical inactivity and smoking. Clinically an effective method is to insist that the family (as opposed to only the child) go onto a healthy diet and activity schedule. In fact, informing the parents that they will also be weighed along with the child, helps address this issue effectively. Although preliminary, co-treatment with an SGA plus a mood stabilizer seems to be associated with a greater risk for age-inappropriate weight gain than treatment with one or even two mood stabilizers. A pilot study has shown Metformin therapy is safe and effective in decreasing weight gain, insulin sensitivity, and abnormal glucose metabolism in children aged 10 to 17 whose weight had increased by more than 10 per cent during less than one year of olanzapine, risperidone or quetiapine therapy.[24]

Developmentally appropriate monitoring guidelines[25] need to be implemented in routine clinical practice and the effectiveness of suggested behavioural and pharmacological interventions should be evaluated.

Mood-stabilizers

Mood stabilizers are usually antiepileptics (except SGAs and lithium) and are used to treat epilepsy or bipolar disorders and occasionally mood lability. Commonly used mood stabilizers include carbamazepine, sodium valproate, lamotrigine, and lithium carbonate. Lithium use warrants regular blood level monitoring, which is often a problem in children. *Sodium valproate or valproic acid* is the most used mood stabilizer and has a reasonable evidence base for its use in bipolar disorder. It however should be used very cautiously, if at all, in girls of child-bearing age due to its teratogenic effects, as well as possible side effect of polycystic ovarian disease. Lamotrigine is a more recent addition and hence being discussed.

Lamotrigine works by inhibiting effects on sodium channels and stabilizes neuronal membranes and thereby moderates the release of excitatory amino acids such as glutamate and aspartate. Lamotrigine is especially useful when significant depressive symptoms exist in bipolar disorder.[26] It has a moderately long half-life, especially as monotherapy and can be given once or twice a day. When used with hepatic enzyme inducing medications such as phenytoin and carbamazepine, the half-life is reduced to 12 h. Valproic acid markedly increases the half-life of lamotrigine to 48 to 72 hand the concomitant use of these medications increases the likelihood of developing severe drug rashes including Steven–Johnson syndrome. Lamotrigine is to be started at very low doses (as low as 5 mg/day) and increased slowly over a couple of months. In general, the initiation of this drug requires great patience. Evidence for anti-manic effects is limited.

Rapid tranquilization when managing severe maladaptive aggression in children and adolescents

Managing acute severe maladaptive aggression requires a quick functional analysis to identify the underlying cause – mood lability, mania, impulsivity, sensory trigger such as hypersensitivity to sound in autism, psychotic experience, autonomic arousal, anxiety or depression etc. The management involves:

- 'Talking down' as the first strategy.
- The patient should be offered the choice of having an oral medication before forcing parental medication.
- Oral medication – risperidone 0.5–1mg, or olanzapine 1–2.5 mg or lorazepam 0.5–1 mg.
- If the above fails, repeat giving either the antipsychotic or the benzodiazepine again in 30 min.
- If this fails, combine the antipsychotic and benzodiazepine after 30 min.
- If oral treatment is not accepted or has not helped, IV haloperidol 1–2 mg or IV diazepam 100–200 mcg/kg or IM haloperidol 1–2mg or IM lorazepam 50–100 mcg/kg can be used.
- The IV can be repeated after 10 min if not sufficient or the IM can be repeated after 30 min. Supportive staff and equipment are necessary as side-effects such as cardio-respiratory arrest can occur. *Maximum safe doses based on age and weight of the child or adolescent should not be exceeded over a 24-hour period.*
- Physical restraint and seclusion in safe environment may also be necessary.
- If **aggression is occurring in the context of a medical condition** (for example, cardiac delirium leading to pulling out catheters etc), midazolam 500 mcg/kg (max 15 mgs) can be used in the context of the medical setting.

Pharmacological preparation for medical or surgical interventions in children and adolescents with intellectual disability or severe behavioural disorders

Children with intellectual disability or mental health problems should receive the same level of medical care if they are physically unwell, as normal children. As they may not be co-operative, often, physical investigations (for example, MRI or dental X-rays) are avoided or postponed, often leading to late interventions and poorer medical outcomes. If the procedure is a minor one, lasting a few minutes, oral midazolam can be used; if it requires longer or for more serious procedures, an anaesthetist could administer oral ketamine or another general anaesthetic. Simple strategies such as anti-emesis prophylaxis, removal of cannulas before they recover consciousness etc., help minimize difficulties.

Conclusions

Paediatric pharmacovigilance for psychotropic agents are essential and more studies on efficacy in this population is necessary. Studies are urgently required in children and adolescents investigating the metabolic effects of individual medication, especially the SGAs and mood stabilizers, using sex and age adjusted measures of weight and body composition, including fasting blood work and blood pressure measurements, protective factors and long-term follow-up. Until such detailed data become available, it is safe to assume that paediatric populations are at least as or more vulnerable to adverse effects compared to adults. True long-term data is not available currently on most psychotropic agents. Evidence on treatment impact on co-morbid disorders, cost-effectiveness and impact on Quality of Life are sparse and urgently need to be addressed.

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9.5.6 Residential care for social reasons

Leslie Hicks and Ian Sinclair

Introduction

Residential care for the young is an elusive object of study. Provided in the past by establishments as diverse as workhouses, orphanages, and reformatories, it has no clear definition marking its boundaries with foster care or boarding education; at the same time it variously aims to shelter, classify, control, and reform and it has no agreed theory or body of values. The need for residential care, and the difficulties of providing it, vary with time and place; the issues it raises are quite different in Romania than they are in California, or were in Victorian England.

Given this diversity, any discussion of residential care needs to outline the context within which it was written. In the case of this chapter the context is provided by current British social policy. Although the focus is on residential care provided to young people by Children's Services in England for social reasons, the conclusions drawn are applicable to the rest of the United Kingdom. The issues raised by this provision have similarities in other parts of the developed world, in virtually all of which the use of residential care is declining.[1,2] This chapter is written against the background of this decline. Its aims are as follows:

- to describe the current characteristics of residential child care in England, and by extension in Great Britain
- to outline the problems that have led to its numerical decline
- to identify practices that should overcome or reduce these problems
- to discuss the role that residential care might play in future.

The characteristics of residential care

In 1979, official statistics for England (the figures for Wales, Scotland, and Northern Ireland are published separately) showed

that there were approximately 95 000 children in substitute care (mainly foster care and residential care). By 2006 this figure had dropped to 60 300. While for much of this time numbers in foster care were fairly constant, the numbers in residential care fell from 35 000 to 6 600.[3] The great bulk of residential care is directly provided by local authority social services departments (more recently Children's Services), although provision by the private sector is increasing.

These figures give, in some respects, a misleading impression of the numerical importance of homes. The turnover in them is quite rapid—roughly 60 per cent leave a home within 2 months of arrival and just under half the placements result from movements within the care system rather than the breakdown of community care. On average homes still see around three times the number of residents in a year than they accommodate at any one time.[4] A recent study found that residential care accounted for about 28 per cent of the time that children aged 12 or over spent in the care system.[5]

The basic characteristics of the local authority homes are well known. They are 'open' in the sense that the residents are expected to go out to school or work and are not restricted at other times. They are typically small, 50 per cent of them have five or fewer places and the average capacity is around six.[6] The buildings are not markedly institutional, although identifiable to the practised eye, are usually located near to where the residents are likely to live, and aim to take local young people. Staff are non-resident. Many care staff do not possess a recognized appropriate qualification and although government targets in the form of National Minimum Standards[7] aim to rectify this situation, progress towards these remains relatively slow. Homes have more staff than residents and research has shown substantial variation in the number of care hours delivered for each resident young person each week (between 37 and 254 h).[6]

Children enter the care system in Britain to be 'looked after' because of a temporary emergency (for instance hospital admission of the carer) or because of abuse, extremely problematic behaviour on the child's part, or a breakdown of family relationships. The main characteristics that distinguish residents from other users of substitute care are as follows:

- Age: 81 per cent of those who start their period of being 'looked after' in local authority homes were aged 10–15 over and about 15 per cent are aged 16 or 17. By contrast, 62 per cent of those entering foster care are under 10 years of age and only 2 per cent are aged 16 or 17.[8]
- Sex: the proportions of males and females in foster care are roughly equal, but nearly 60 per cent of those in residential care are male.
- Geographical location: some local authorities use residential care much more than others. In 2006 the proportions of 'looked after' adolescents in residential care varied from just under 3 per cent in one local authority to slightly over 24 per cent in another.[3]
- Behaviour: on average children in residential care exhibit more 'challenging' behaviour than do foster children, as reflected for example in educational performance, measures of psychiatric ill health, delinquency, and the likelihood of being imprisoned as an adult.[9,10]

One study[4] carried out as part of a major tranche of research on residential child care in England showed that residents had relatively high levels of school exclusion, truancy, delinquency, violence towards adults and children, running away, risk-taking sexual behaviour, self-harm, and suicide attempts. Around two-thirds of residents entered 'care' for the first time as teenagers, generally because family relationships had broken down, and a further one-fifth entered because of abuse. They were unwilling to be fostered or seen as too disturbed for this. The great majority had previous placements in foster care, residential care, or with relatives.

The role of the homes was to return some as soon as possible to their families, attempt to improve the behaviour of others, or prepare them for independent living, and to keep a minority (around one-fifth) for the foreseeable future. Although there was some specialization, most homes attempted to fulfil all these roles and took all types of resident.

Problems of residential care

Residential care lacks the moral basis that it formerly had; the disciplines and virtues required for successful group life are no longer seen as imperatives. The Children Act 1989 ensured that, except in rare circumstances, young people could no longer be 'looked after' simply on the grounds of delinquency when their own welfare and those of others are not at risk.

Theoretical uncertainty accompanies these moral shifts. The best-known texts on residential care are now around 25 years old and are dubiously relevant to the current situation where staff no longer live on the premises, and where there have been major changes in staffing, turnover of residents, clientele, and the size and purpose of homes. In any event, there was never a consensus about which theory should underpin treatment or training. Clear evidence for the efficacy of any approach has been lacking, whereas the evidence for the harmful effects of bad residential care has been clear—and frequently repeated on social work courses. Young people reach residential care as the culmination of a process that marks, and possibly even exacerbates, their social exclusion. Typically, they are disturbed, poorly supported by their families, and lack educational qualifications, a combination which makes it difficult for them to compete subsequently in the job market or to lead happy lives. Descriptions of residential care do not suggest that it mounts the determined attack on these problems that might have some chance of success.

There are particular problems for those young people who leave residential care to live independently. Residential homes have difficulty in retaining young people until such time as they are 'properly launched'. To do so would create problems related to cost and to creating a regime suitable at the same time for younger and older teenagers. This means that young people leave care when they are still vulnerable, to cope with lives that are lonely and difficult, at an age much younger than their better qualified and supported contemporaries leave home. Their transitions to the adult roles of getting an income, maintaining a home, and living with a partner are made earlier than those of others and compressed into a shorter period.[11] Unsurprisingly, they have a much higher chance than their contemporaries of becoming lone parents, unemployed, imprisoned, or homeless.[12,13]

In addition to these problems, there are often pragmatic difficulties.

- Residential care is very expensive; estimated costs are around £78 000 per place per year in local authority homes and around £87 000 in the non-statutory sector.[6] The system costs

considerably more than foster care or preventive work.[14] Around 14 per cent of those who enter stay for prolonged periods and take up half the beds.

- Residential homes are prone to scandals involving sexual abuse, outbreaks of disorder, and suicidal behaviour.[4]
- Delinquency and running away are widespread within residential homes.[4]
- There is widespread bullying, sexual harassment, and personal unhappiness.[4]

Official reports[15] have emphasized the need to recruit a better trained workforce, and there are considerable efforts being made to raise standards in this respect.[16] However, the proportions of qualified staff remain low and studies that have looked for a relationship between the proportion of qualified staff and a measure of performance found no association.[4,6,13]

Contrary to common belief high staff ratios do not in themselves lead to better control in homes or better staff morale or better outcomes, although they do increase costs.[4,6,13]

Improving the quality of residential care

If residential care is to overcome these problems, it will need to reduce the incidence of difficulties in the home and increase its influence after residents have left.

In terms of immediate impact, residential homes and schools vary widely in the morale of the staff, the incidence of delinquent behaviour and running away, the proportion of residents who avoid going to school, the relationships between staff and residents, and the degree to which the residents report that they are bullied, offered drugs, or feel part of a friendly establishment to which they are committed. These characteristics are far from fully accounted for by intake, and establishments which do 'well' in terms of one of them tend to do 'well' in terms of the others.[4,6,13]

These studies suggest the following conditions are required for managing homes that are successful in the short-term.

- The residential units are either small in size or consist of a large unit divided into small subunits. Such small establishments seem better able to combat the influence of delinquent residents.
- The units have clear aims, with which the manager of the home is in agreement.
- The manager of the home has a clear philosophy on how the young people can be helped, and the staff are in agreement with this outlook.
- The residential unit is not based close to the residents' homes. An emphasis on local placement makes it harder to combat the influence of local negative cultures or to maintain a clear focus.

The most recent of these studies[6] was able to demonstrate the link between these immediate outcomes for young people and the practice of managers of homes. Young people experienced better outcomes where managers were accepted as embodying good practice from within a clear ethos, had positive strategies for working with the behaviour and education of young people, and importantly, could enable staff to reflect and deploy these. Without this bedrock, the systems, procedures, and management targets advocated in reports on the residential system will fail. The basic principle is to establish a set of shared expectations and approaches and this is more easily done in a small well-led establishment with a cohesive staff group.

Unfortunately, the ability of homes to affect the behaviour of residents in the short-term implies that the environments to which residents go next are equally powerful. This has been shown to be the case and creates problems for homes in achieving long-term change. (Evidence on the short- and long-term impact of residential care is discussed in more detail elsewhere.[4,6,13]) Overcoming these problems requires the following:

- Reduction of delinquent and problematic behaviour in the home—as noted above these are influenced by the residential environment, and there is evidence that they lead to future delinquency.
- Encouraging educational achievement and those skills required for 'success' in subsequent life and that are relevant to the resident's subsequent environment—and are seen by the residents as being so.
- Working to improve family relationships so that the resident either goes back to an improved family situation or can turn for support to his or her family even though he or she is not living with them.
- Providing continuing back-up from the residential home or an aftercare scheme and practical support (e.g. relating to accommodation).

Success is probably more likely where the home operates on a number of fronts simultaneously: seeking to improve skills, learning and educational achievement, and the way the residents see themselves and also the environment to which the residents return.

Future of residential child care

The case for residential care is essentially three-fold. First, it is able to tolerate behaviour that leads to foster-care placements, the main alternative at the moment, breaking down. Second, a number of young people choose it in preference to foster care. Third, it offers a major resource for adolescents, in preparation for adulthood, in providing supported accommodation, and for those who make a later entry to the care system, such as unaccompanied asylum seeking young people. Its future will depend, in part, on the degree to which other provisions can be developed that can match these advantages.

It seems likely that more alternative provision will be developed. Some intensive American fostering schemes do appear able to contain young people who are as challenging as those in British children's homes, and these schemes are being adapted to conditions in the United Kingdom. Some forms of fostering with outreach support from respite residential units are in their early stages of development. Remand fostering, 'crash pads', and occasional beds in family centres might provide for some young people currently accommodated briefly in children's homes. Supported lodgings could provide for some, particularly older adolescents who avoid foster care because they feel they cannot live in a family, or that it is time that they started to move out on their own. Boarding schools might provide for younger children who do not want to enter foster care because they feel it invites disloyalty to their family, or who enjoy the company of other teenagers.

The comparative advantages of these differing kinds of provision have not been evaluated and it is important that they should be.

The growth of alternative provision will allow the continuing reduction of the more traditional residential sector. It is also likely that the sector itself will change, with the current all-purpose children's home being replaced by more specialist provision that can focus upon clear goals. Research that could determine the shape of such provision is lacking and should be carried out. Possible models of provision include the following:

- Secure (namely, closed) provision—public opinion will not allow young people who have committed very serious crimes to remain in open conditions.
- Brief- to medium-term provision designed to allow time for young people to consider their situation and move on to new placements in a planned way—there is evidence that fostering placements made from residential care are less likely to disrupt, although it is not yet clear why this is.
- Medium-term provision designed to provide treatment—the case for such provision is that treatment involving a group can be more effective. Evidence for this is lacking, although the variations in the behaviour of residents in different establishments is evidence of the power of the group.

In addition to these kinds of residential care, there is a case for some long-stay accommodation in which groups of residents live together. Such provision would be similar to a large foster home or the former family group homes. There seems no reason why it should not work well. It would, however, need to be less generously staffed than current residential homes, otherwise there would be pressure to move residents on for purely economic reasons.

Conclusions

Residential care is very different in different parts of the world. In the United Kingdom it is provided by small community-based homes, who work with an extremely complex and challenging clientele, and face problems associated with high costs, scandal, and frequently a lack of a clear rationale. Despite these difficulties there are wide variations in the performance of such homes, particularly in terms of their immediate impact. Outcomes after the residents have left are harder to influence. However, an approach which combines attention to the residents' education, behavioural problems, and family environment seems most likely to be effective. In the longer-term, the residential sector is likely to continue to shrink and the part that remains may well need to become more specialized. Research that could guide such developments is presently lacking and should be undertaken.

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9.5.7 Organization of services for children and adolescents with mental health problems

Miranda Wolpert

Introduction

This chapter aims to guide the thinking of practitioners who might be involved in developing services to meet the needs of children and young people with mental health difficulties. Anyone involved in this challenging but vital endeavour will need to address the following questions:

- Who should the service be for?
- What sort of interventions should be provided?
- How should the service be structured?

- Who should the staff be?
- How can the service be made most accessible?
- How can service quality be ensured?

This chapter will look at each of these issues in turn to explore how each might best be approached.

Who should the service be for?

Ever expanding conceptualization of mental health needs has meant that at least four groups of children are routinely referred to in the discussions about service development in this area:

1 Children and young people in difficult (and often terrible) circumstances
2 Children at risk of developing diagnosable mental health problems
3 Children with diagnosable mental health problems
4 Children with levels of impairment due to mental health issues that make it difficult for them to function within their community/culture

Lack of clarity when discussing the needs of the different groups can confuse service planning. In particular, it can result in the range of agencies that are all increasingly involved in collaborating in planning provision, talking at cross purposes. When resources are limited (as they generally are for these populations of children, even in the most economically developed countries of the world) it is likely that choices will need to be made about prioritizing between and within these groups. It is therefore vital to be clear at the outset which groups are seen as the priority for a given community and to achieve multi-agency agreement on this.

The needs of each of these groups in relation to service provision in this area is considered in turn below.

Children in difficult circumstances

Definitions of 'difficult circumstances' vary with national context. The estimated 14 million AIDS orphans (concentrated largely in Africa) would fall into this category as would the 12 million children in the United States living below the poverty line along with all those children who are in contexts of war, famine, or abuse.[1] This group is likely to include those children with high and complex mental health needs who are the hardest to reach and who are a policy priority in many areas. However it may also include children who do not have specific mental health needs. Services need to ensure they are accessible to these groups where they do have mental health issues but specialist mental health services are unlikely to be the main provider of care for the majority of children in such circumstances.

Children 'at risk'

Risk factors for developing diagnosable mental health problems include some aspect of difficult circumstances (such as violent environments, lack of warm family environments) but risk is also heightened by other individual and interpersonal factors such as: brain injury, low birth weight, poor parental mental health, low IQ, irritable temperament, family dysfunction, and the lack of a key supportive relationship with an adult. For children identified as 'at risk' the focus for mental health provision is likely to be on how to enhance resilience. There may be key opportunities for intervention, such as when the child is born or at key transition points (such as starting school, changing school, leaving school, etc). There is an argument for targeting particular groups such as children of parents with mental health problems. However, evidence for the effectiveness of health promotion and prevention initiatives is still limited and there is some evidence that the promotion of resilience may best be achieved by agencies other than child mental health specialists, such as welfare sectors creating greater neighbourhood cohesion, perinatal services reducing risk of low birth weight and educational services implementing appropriate programmes to promote emotional well-being and support children in times of stress. It is likely that services should only put major resources into targeting 'at risk' children if they have sufficient resources to meet the needs of those children with existing problems.

Children with diagnosable mental health problems

Epidemiological data from Europe and the United States suggests that around 10–20 per cent of children suffer from diagnosable mental health problems (using ICD-10 or DSM-IV criteria). There are indications of differences between countries, with slightly lower rates found in India and Norway, for example, and slightly higher in Brazil, Bangladesh, and Russia. However, not all of these children need direct service provision. Some diagnosable mental health problems may get better without intervention (such as depressions in mild form). For these reasons, amongst others (in particular the fact that not all current treatments are proven to do more good than harm—as will be discussed below), it is not advised to go down the route recently suggested by the American Psychiatric Association of screening all children in schools and treating all diagnosable difficulties.[2]

Children with impairment due to mental health difficulties

It is children in this category who are likely to be the main target for specialist child mental health provision. The impact of significant and impairing mental health difficulties if not effectively treated can be substantial. Worldwide, suicide is the third leading cause of death amongst adolescents; major depressive disorder, often starting in adolescence, is associated with substantial psychosocial impairment; conduct disorders amongst children tend to persist into adult life and are reflected in later drug abuse, antisocial behaviour, and poor physical health. This group covers a wide variety of children and young people with problems ranging from bed-wetting to psychosis. It includes those with chronic and multiple difficulties and those with discrete and defined difficulties. It encompasses those with difficulties where there are known to be effective interventions and those presenting with problems where the best course of action is much less clear (as will be discussed below). It is this very mixed range of problems that child mental health services must address.

Prioritizing needs

The way needs are prioritized in planning service provision will be heavily influenced by the context in which services are located. In some countries independent child mental health provision and unaffordable luxury when pitted against other basic needs. The World Health Organisation[3] has suggested that where resources

are particularly limited, priority for funds for child mental health provision should be given to those children with existing difficulties which are:

- occur frequently (and/or have highest cost implications)
- cause a high degree of impairment
- have the greatest long-term care/cost consequences
- have an evidence base for treatment and (particularly in those countries with the most limited resources)
- where the difficulties can be dealt with in primary care or universal services such as schools or GPs.

In countries with greater resources, child mental health professionals' input can be conceptualized as being provided at universal, targeted, and specialist levels. This involves supporting and working alongside universal provision to promote emotional well-being whether in schools or via primary health care workers. Targeted provision aims to promote emotional well-being in those deemed either at most risk (group 2 above) or in most need (group 1 above) with the groups being determined by policy imperatives. Here specialist mental health professionals will work alongside workers from other sectors who take a lead in relation to the needs of these groups as a whole, such as social welfare workers and primary care staff. Specialist provision aims to intervene primarily with those with existing impairment due to mental health difficulties (group 4 above) and can be provided at a local community level, though for more rare and specialized resources may be provided at a regional ore even at a national level.

Weighing mental health promotion initiatives against interventions for those with existing impairments requires particularly careful thought. Whilst there is evidence that promotion programmes may sometimes promote emotional well-being, research to date has not proved that this will reduce levels of significant disturbance and thus impact on specialist services, nor that such programmes are necessarily the most effective approach. It therefore does not seem warranted at this stage to assume that investment in prevention can be done at the expense of investment in services for those with existing impairing mental health difficulties.

In planning response to 'need', it is also important to consider the potential negative and even harmful impact of increased specialist mental health services. Mental health professionals are frequently in danger of assuming that more specialist mental health provision is unquestionably an unalloyed good. The need for more provision must be set in the context of other (sometimes competing) 'needs', such as: the primary need of children to be nourished, sheltered, and protected; their need not to be stigmatized or miss education; and their need not to receive inappropriate, ineffective, or harmful treatment. At times an inappropriate mental health focus can be an unhelpful drain on resources. One documented example is when well-meaning voluntary groups entered a country following a disaster to provide 'interventions for PTSD' that were not linked to other relief efforts and actually interfered with and undermined key initiatives.[1] Whilst the costs of not providing effective specialist mental health inputs can be high, it is important to remember there are also costs to providing unhelpful services.

What sort of interventions should be provided?

As yet, few services have been developed on the basis of considered evidence. Systems of care in CAMHS (as for many areas in health care) have historically been developed on the basis of beliefs, assertion, and innovation within the limits of given structures but with little reference to the slowly and tentatively emerging evidence base. The arguments for trying to promote evidence-based service development are compelling. Natural biases in reasoning mean that people tend to make decisions based more on things that fit their assumptive world view than those that challenge it and are more influenced by the charisma of those promoting a particular approach than by evidence for its effectiveness. When the evidence base is not used as the basis for service development, it makes it more likely that seemingly plausible but ineffective and/or harmful interventions may be introduced or continued and that new interventions that have been shown to do more good than harm may never be introduced.[4]

The evidence base in relation to child mental health interventions, whilst growing, is still limited both in extent and quality. Shortcomings include the sheer paucity of studies, the fact that most research is conducted in United States and there is lack of agreement over appropriate outcome measures. Even where interventions have been found to work in academic studies they are generally not as effective when applied in 'real life' settings. This difference may be due to differences in the populations of children seen, types of interventions made, and/or outcomes assessed. There is increasing evidence of the necessity of carefully implementing all aspects of a particular intervention if it is to be as helpful, and that lack of 'fidelity to model' may account for the lack of generalizability of some of the interventions. On the other hand, the role of non-specific factors such as therapeutic engagement, expectations of change and therapist warmth may need to be taken into account.[5]

Whilst bearing these limitations of the research evidence base in mind there is an emerging consensus in relation to some key interventions[6–8] and increasing attempts to develop guidance for practitioners based on the evidence in this area are being developed (e.g. National Institute of Clinical Excellence guidelines in England, American Psychiatric Association Practice parameters in the United States). Those interested in the detail of the sorts of interventions currently found by the evidence base to be most efficacious should go to these guidelines and may also be interested in attempts to summarize the evidence base[8, 9] (condenses current findings to inform the work of practitioners and others and is freely available via the internet).

It is hoped that as the evidence base develops, so should the sophistication with which it might be interrogated. For every intervention listed above as having 'good evidence' to support it, the following questions should be asked (modified from Kazdin[10], p. 113):

- What are the costs, risks, and benefits of this intervention relative to no intervention?
- What are the costs, risks, and benefits of this intervention relative to other interventions?
- What are the key components that appear to contribute to positive outcomes?
- What parameters can be varied to improve outcomes (e.g. including addition of other interventions, non-specific clinical skills, etc)?
- To what extent are effects of interventions generalizable across (a) problem areas, (b) settings, (c) populations of children, and (d) other relevant domains?

We are a long way from being able to answer these questions in relation to CAMHS currently but it is to be hoped that this will develop with time.

However, even as the evidence base grows from academic studies this must still be treated with caution. It is not suggested that findings from even the most rigorously undertaken randomized controlled trials can necessarily be applied wholesale to all individuals with similar problems. The full context of an individual's range of needs and circumstances must be taken into account and it is also hoped that understanding of the academic literature will be supplemented with a growing evidence base emerging from practitioners own routine evaluation of their own work (see discussion in 'ensuring service quality' section below).

How should the service be structured?

If the evidence base for types of intervention is limited, this is even more so for types of organization.

The 'Fort Bragg Study' (and the subsequent Stark County Study) conducted by Bickman and colleagues in the United States warrants particular attention as they generated such high levels of interest and controversy. This study evaluated a large-scale system change project designed to improve outcomes by providing an unrestricted set of coordinated inputs from a range of services. Results were compared with other sites using traditional services. Information was collected on service use, cost, satisfaction, clinical, and functional data over the course of the 3-year study and at follow-up since. The study found greater access and increased rates of satisfaction and less use of inpatient services, but no differences in behavioural–emotional functioning overall and the cost was much greater at Fort Bragg. The subsequent Stark County Study also found that a multi-agency system for care led to no significant difference in clinical outcomes when compared with routine services. Bickman concluded 'the current national policy of large investments in systems of care infrastructure is unlikely to affect children in the manner intended . . . we need to focus on the services or treatments themselves to improve outcomes'.[11]

This conclusion has been hotly contested. Flaws in the study design, implementation, and interpretation have been highlighted. It was argued that services were overwhelmed in the early months and provided less effective interventions, that the researchers failed to take into account some of the positive outcomes on some of the measures used and so on. It has been suggested that there is evidence that 'wrap around services' (whereby a range of services are provided in a coordinated fashion to children and families based on their needs and not those of the services) do produce better outcomes and that the poor results above may have been due to lack of fidelity to this model.[12]

There is some evidence that shared understandings at the highest level and pooled budgets seem to be crucial to positive multi-agency collaboration. 'MHSPY' in the United States is an initiative that involves five public and two private agencies who have come together with blended funding to provide a coordinated focus on a group of 'at risk' and ill children and where outcomes look positive though lacking a control group or clear cost-effectiveness data as yet.

There is little evidence available currently to guide service developers as to the best way to structure services in relation to a number of other key issues, such as: what age of children should be seen by different services? Is it best to structure services around particular problems or around different age groups? What should be the balance between inpatient and outpatient provision? Whilst it is hoped that with time, more evidence will emerge in relation to these issues, it may be hypothesized that, here too, non-specific factors, may be relevant. Thus the leadership skills of the clinicians who establish a given service, the strength of their commitment in a particular shape of service, and the ability of key individuals to collaborate across whatever boundaries are inevitably created, may impact as much, if not more, than the specifics of different forms of service structure.

Who should the staff be?

In terms of who the key staff should be who provide specialist mental health services for children, whilst there is much assertion and rhetoric as to what is the ideal workforce composition and an emphasis on multi-disciplinary and multi-agency team development wherever possible, there is, in fact, very little hard evidence as to what an ideal workforce should look like.

In many parts of the world specialist mental health professionals are in short supply, and there is an increasing focus on capitalizing on opportunities for mental health provision to piggyback on other sectors with more provision (such as HIV programmes in Africa or schools in many areas of the world). The potential contribution of paediatricians, primary care workers, and teachers is increasingly recognized, though care must be taken not to overload already stretched systems and research highlighting the importance of 'fidelity to model' (as discussed above) suggest that there may be a necessary threshold of amount of training combined with ongoing organizational support, for subsequent interventions to be effective.

How can the service be made most accessible?

Attempts to make provision more accessible as well as less stigmatizing can include use of education and primary care workers, community and religious leaders, and family networks. There is evidence that linking with relevant belief systems of the community, such as ayurvedic treatment and yoga in parts of India, may be crucial, and that providing training for traditional healers may help increase child mental health capacity. The work of voluntary organizations can be a major source of innovation and energy and communities need to find ways for statutory services to learn from them. For example the walk in community centre, Empilwent in South Africa pioneered a more accessible drop in approach.

It could be argued that child mental health services have been slow on the whole to 'get out of the clinic' and in particular to embrace the impact of new technologies that might increase access. However some interesting innovations are developing. These range from mobile services such as the peripatetic child mental health team which travels across Germany providing follow-up on previously hospitalized young people, providing new consultations, and supervision of institutions for children[13] to increased use of telephone and text contact generally, and telephone helplines and websites specifically.

Telephone helplines such as 'Kids Help'[14] in Australia, 'Childline'[15] in the United Kingdom, and 'Parents Information

Service' (YoungMinds Parent Information Service)[16] in the United Kingdom, are often run by charities independent of statutory provision but it may be in the future that greater links can be made by such resources and more traditional services. 'Telefono Azzuro' of Italy shows the way these can be combined with other provisions.[13]

A range of websites have been developed by and for young people some of which allow for discussions between young people and others offer access to specialist advice and support (e.g. 'ru-ok.com').[17] The use of emails and text messaging to communicate between professionals and young people is now increasing as are the use of IT aids to assessment and treatment and the pace of innovation in this area is likely to accelerate sharply in the future.

How can service quality be ensured?

The implementation of routine outcome evaluation is likely to be a crucial factor in ensuring service quality. There are increasing attempts to ensure routine outcome evaluation is in place. The CAMHS Outcome Research Consortium (CORC)[18] (chaired by the author) is collaboration between over half of all services across the United Kingdom who are implementing an agreed model of routine outcome and agreeing jointly ways to present the data in order to inform service providers and users and to help inform service developments (CORC).[8] The approach has parallels with that being employed in the United States and Australia. In all cases a small suite of measures is completed by service users and providers at initial contact and at some time(s) later. Increasingly models are being developed whereby individual practitioners evaluate outcomes over the course of treatment which can be immediately compared with outcomes from others with similar difficulties at the outset, allowing practitioners to get feedback about progress relative to others.

For child mental health service to institute routine outcome evaluation and to develop into self-reflective and learning organizations, requires functioning IT systems and an agreed core dataset.[19] There are now datasets for child mental health emerging in some countries.[20] But even in the most developed countries, the promise of coherent IT systems, remains more a hope than a reality. It is key that IT systems develop that are capable of supporting decision-making by providing easy access to information for practitioners so that they can get feedback on their work and learn accordingly.

There is an increasing emphasis on the importance of involvement of service users to inform service development priorities and to be part of any evaluation of services, to ensure quality. Some innovative models have been tried such as in Ohio which has created outcome systems from service user perspective and in parts of the United Kingdom where young people are trained as service evaluators though again the impact of these on quality of services in the future is yet to be researched.

Conclusion

There is no one ideal model for organizing services, however some key principles can be identified:

1 It is helpful to start with an analysis of the range of anticipated needs, and to seek clarity as to the current priorities for a given community.

2 Where resources are limited, specialist mental health provision should be targeted foremost on those with greatest levels of impairment for whom there are the most effective interventions.

3 Implementation of effective models of prevention and promotion should be supported so far as resources allow and not at the expense of provision for those with existing difficulties.

4 The existing evidence base in relation to effective interventions and models of delivery should be seen as a starting point for practice, though the limitations of the evidence base should be held in mind.

5 Successful service development is likely to rest on collaboration across health, social care, and education—using pooled budgets where possible.

6 Good information gathering systems are needed that allow individual practitioners and managers to audit and review work.

7 Staff members should be encouraged to adapt their practice in the light of the emerging evidence from literature and their own outcomes rather than retaining allegiance to any one theoretical model or framework.

8 Collaborations between service providers that focus on developing best practice may be of help.

9 The input and involvement of service users may be of relevance in helping develop accessible and acceptable services.

10 Remember not all service developments are benign—always weigh up possible harm that may be caused as well as potential for good.

Further information

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9.5.8 The management of child and adolescent psychiatric emergencies

Gillian C. Forrest

'Child' is used throughout this chapter to refer to anyone aged under 18, to avoid the repetition of 'child or young person'. 'His' and 'her' are used interchangeably.

Introduction

This chapter provides a practical approach to the management of psychiatric emergencies in children and adolescents. Such emergencies are challenging for a number of reasons. The professional resources available are usually very limited, and there is often confusion or even disagreement between professionals over what constitutes a psychiatric, as opposed to a social emergency. The parents or carers play a key role in the situation and need to be engaged and involved appropriately in the assessment and management; and issues of confidentiality and consent need to be taken into account. In addition, the psychiatrist may find himself or herself working in a variety of settings—the child's home, a hospital emergency department (A and E), a police station, a children's home, or residential school—where the facilities for assessing an angry, disturbed, or upset child may be far from ideal.

Most emergencies occurring in community settings involve externalizing behaviours: aggression, violence; deliberate self-harm, or threats of harm to self or others; or extreme emotional outbursts.[1,2] Some will involve bizarre behaviour which could be an indication of serious mental illness or intoxication by drugs or alcohol, or a combination of both. The emergency situation often arises in the context of acute family conflict or distress.

Frequently other agencies are involved before the psychiatrist is called in (for example, emergency room staff, social workers, or the police). The on-call psychiatrist needs to be familiar with or able to obtain immediate advice about his or her local child and adolescent psychiatric services, the local child protection and child care procedures, and with the relevant mental health and child care legislation.

Vignette 1:

A 12-year-old boy is in the police station, after attacking a neighbour and smashing a window. He punched a police officer when they tried to pacify him. He has refused to talk to the police, and is sweating and dishevelled, pacing up and down and muttering to himself. The police think he is psychotic. The neighbour is in the waiting room; his father has been called back from work.

After obtaining the history of the incident from the neighbour and the police, the psychiatrist interviewed the father. The boy had been diagnosed with an autistic spectrum disorder when he was 5. He was easily upset by any change of routine. His mother had gone away for a few days, leaving him in the care of a neighbour while his father was at work. This aggressive outburst was precipitated by the neighbour refusing to let him watch his favourite video. The boy calmed down with his father and a mental state assessment confirmed autistic features but no psychotic symptoms. The psychiatrist persuaded the neighbour and the police to drop charges and the boy was allowed to return home. The family were offered an out-patient appointment but declined this, and arrangements were made for his General Practitioner (GP) to review the situation in a few days' time.

Vignette 2:

A 15-year-old girl has locked herself in the bathroom at home with a carving knife and is threatening to cut her wrists. Her parents have been unable to persuade her to come out and are now distraught. The GP was called but she refuses to speak to him.†

When the psychiatrist arrived, he asked the family to withdraw so that he could talk to the girl through the bathroom door. She was very upset and described how she had been dumped by her boyfriend earlier that day, had had a row with her step-mother about a large phone bill, and now felt that she didn't want to live any more. The psychiatrist persuaded her to come out to talk things over with her parents. There was no evidence of a depressive disorder and after the psychiatrist helped the girl share her distress about her boyfriend with her

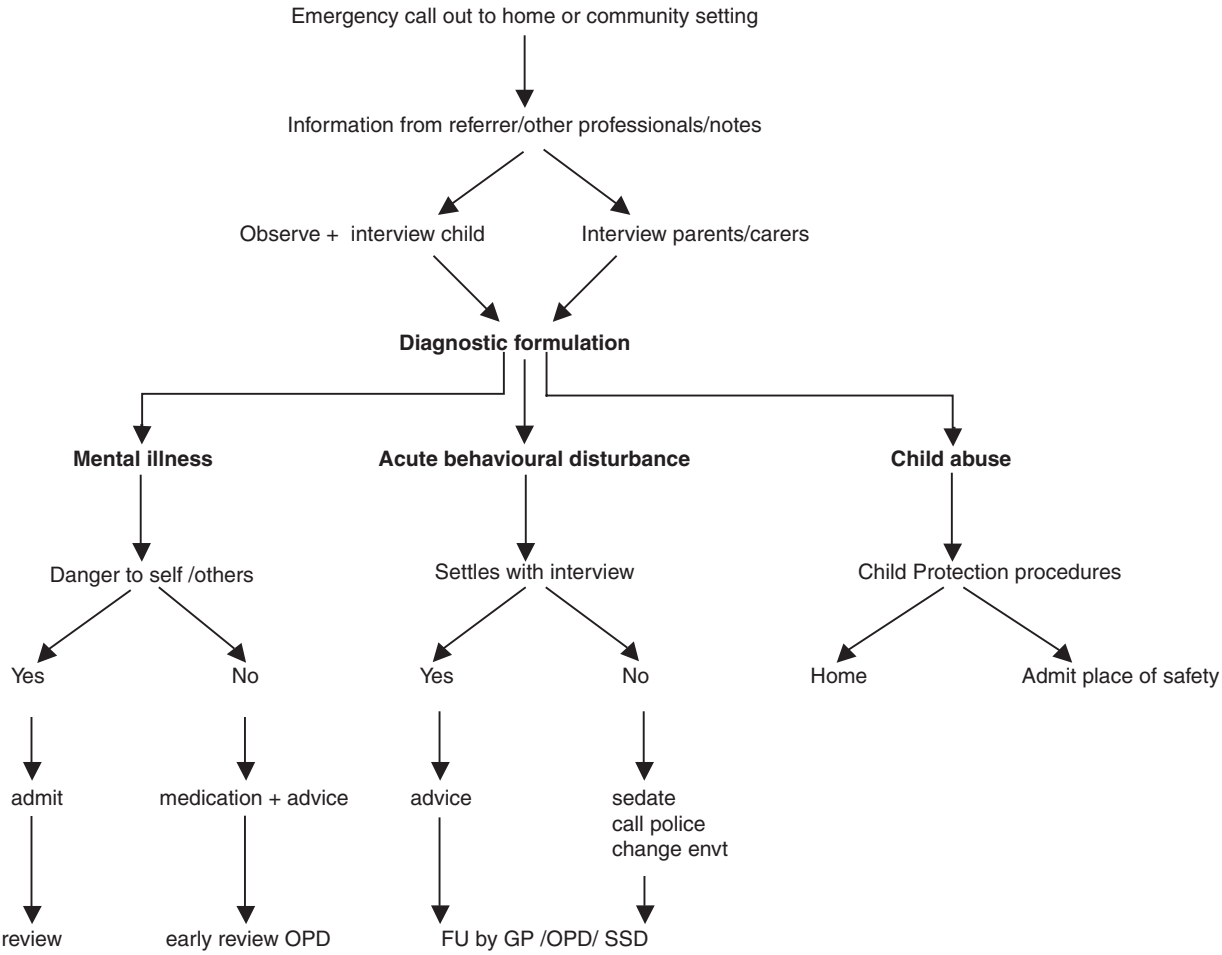

Fig. 9.5.8.1 Flow chart of management of child and adolescent psychiatric emergencies.

parents, she calmed down. There was a history of one previous attempt at self-harm, also connected with peer relationship difficulties, and the psychiatrist felt that there were some underpinning issues around family communication. He offered the family an out-patient appointment to explore these further.

In the emergency situation, the role of the psychiatrist is to:

- assess the disturbed behaviour
- make a diagnostic formulation which distinguishes behaviour associated with mental illness from that which is a reaction to an upsetting event or situation
- produce a care plan to manage the situation safely and effectively in the short-term.

A step-by-step approach to the management of child and adolescent emergencies is described here. The management of deliberate self-harm is dealt with in Chapter 9.2.10.

Step 1. Gathering information

Before attempting to assess the child and his or her family, it is very important to gather as much background information as possible about the incident or behaviour from any professionals who have been directly involved. They may be able to give an accurate description of the child's behaviour and any incident which preceded it; or useful background information about the family. Take time to read any notes that are available; and talk to those involved (GP, police, paediatric staff, A and E staff, etc.).

Step 2. Interview with the child/young person

Although this is described first, parents may expect to be seen before the child, or it may be preferable to talk to them first to gather background information (see Step 3).

A disturbed or upset child in this situation will be wary of any stranger, and the more out of control they are, emotionally and behaviourally, the more likely they are to be uncooperative initially. The psychiatrist needs to calmly establish that they have come to try and help the child, that they are non-judgemental and that they can be trusted. She should always ask to see the child alone, to establish a confidential relationship, and to be able to assess the child without the influence of parents.

Honest and open communication is essential, especially if there is any possibility of child protection issues. Often the child is terrified

that they are going to be forcibly taken away from their home or family (a desperate and angry parent will often threaten this), and this fear may need to be addressed before the assessment can begin.

A useful approach is to say to the child: 'I'm Dr ….. I'm a psychiatrist who sees children and young people with all sorts of worries and troubles. I've come to see you now, to try and understand what this is all about, and see if I can find a way to help'.

If the child refuses to talk at this stage, they may be angry, mistrustful, or psychotically withdrawn. Patience is required to see if this can be overcome by convincing the child that you have come to listen to them and not to 'take them away'. Reflecting back to the child how you perceive their emotional state may also help. For example, the psychiatrist might say: 'You are obviously very upset and angry. I wonder what it is that's made you feel this way'. Sometimes children will choose to communicate by writing notes or drawing, rather than by using speech, and if the child is mute, the psychiatrist should try offering paper and pencil.

If the child remains violent and out of control and the behaviour is being reinforced by attention and anxiety, it will be necessary to ask everyone to withdraw for a while to give the child the chance to calm down on their own. If this still does not help to calm the situation, the psychiatrist will need to set some limits, which could include calling the police to regain control of the behaviour. Medication should only be considered as a last resort when all other strategies to deescalate the situation and calm the child have failed (see Step 5: Management of emergencies in community settings section).

When the child starts to be able to communicate with the psychiatrist, they can then be encouraged to give their account of what happened to precipitate the behaviour. As this is explored, it will be possible to assess the child's mental state (see Chapter 9.1.3) and to form an impression about whether this is mental illness, or whether the disturbed behaviour is the result of emotional turmoil secondary to relationship problems, family conflict, or other psychosocial issues. Further information from the parents or carers will be needed to clarify this.

If child abuse is suspected, an interview with the child alone is crucial. The psychiatrist will need to enquire about the possibility of physical and sexual abuse without using leading questions[3] (see Chapter 9.3.2). A good starting point is 'Has anyone ever hurt you or made you do anything you didn't like?' The child needs to understand that you may need to breach confidentiality in order to protect them from further harm. (see Consent and confidentiality section).

Step 3. Interview with parents/carers

It is essential to see the parents or carers as part of the assessment, in order to fully understand the context of the crisis, and identify any relevant contributory factors. They may be seen first, or after the child's assessment, depending on the situation. It is often helpful to see the parents or carers on their own so that they can speak freely about their child.

These are the areas that will need covering:

- History of current episode/disturbance. Current medication?
- Previous emotional or behavioural problems.
- Brief developmental history: normal or unusual features? Learning difficulties? School and academic progress.
- Temperament; relationships with family/extended family members, other adults, peers.
- Family composition and history (parental separations/divorce; relationships mental and physical illness; recent life events).

During this interview, an assessment can be made of the family functioning; the parent's or carer's mental health, and their attitude to the child, and whether this seems to be playing a part in the current problem (e.g. high levels of family discord; rejecting, neglectful or hostile attitudes to the child; harsh parenting practices; parental depression, anxiety, drug or alcohol abuse).

Vignette 3:

> A 10-year-old has been raging around his house for several hours, following an argument with his mother about going out with his friends. He has been smashing toys and kicking in doors and wrecking his bedroom. His single mother and 6-year-old sister are terrified and cowering in the sitting room.

The psychiatrist spoke calmly to the boy through the door, stating that he wanted to talk to him. The boy continued being aggressive and abusive and the psychiatrist then said he would give him 5 minutes to calm down and come out, otherwise the police would have to be called in to help. The boy did then manage to calm down and talked about his resentment of his mother's rules, and his angry feelings about the loss of his father, who had had little contact with him since leaving the family.

His mother told the psychiatrist that she and the boy's father had divorced a year ago, and there had been on-going disputes with her exhusband about money and his erratic contact with the children. Recently she had become depressed, and her son had become increasingly irritable and oppositional at home. She felt unable to control her boy's temper outbursts.

The mother agreed to see her GP about her depression, try and talk to her ex-husband about seeing the children on a regular basis, and attend the clinic with the children for some family counselling sessions. The boy appeared relieved that his mother was going to seek some help.

Step 4. Making and sharing the diagnostic formulation

There should now be sufficient information available to make a diagnostic formulation. This will include the diagnostic category for the disturbed behaviour, the factors which led up to the acute disturbance; the factors which precipitated it, and the risk factors for continuing or recurring problems.

The Three Ps:

Predisposing Factors

Precipitating

Perpetuating

These factors may be present in the child herself (temperament, illness); in the family environment; at school or college; in the child's wider social environment (friends, neighbours; clubs, etc.); or any combination of these.

Apart from acute intoxication with drugs or alcohol, and deliberate self-harm, most child and adolescent psychiatric emergencies in the community fall into three categories:

- mental illness
- acute behavioural disturbance due to family/psychosocial factors
- child abuse

The psychiatrist should share his formulation with the child and parents/carers, using age-appropriate, and jargon-free language, to help their understanding of the events. Sometimes the parents or the child will need further discussions before a shared understanding between the psychiatrist and the family about the problems can be reached, and an acceptance of the proposed management/care plan by the family.

The formulation should also be shared with any other professionals present, as they may be needed to contribute to the immediate management plan, or even to take over the care of the child.

Step 5. Management of emergencies in community settings (see Fig. 9.5.8.1)

Mental illness

The emergency management of a child diagnosed with a mental illness will depend on the risk assessment. Is the child a danger to himself or others? If the psychiatrist considers this to be a high risk, then referral to inpatient care will be necessary to allow further assessment and treatment in a safe setting. Compulsory admission, using mental health legislation, may be required if the child or the parents refuse to cooperate with this plan (see Consent section). In some places, it may be possible to admit younger children to a paediatric ward for further observation and treatment. Where there are medical complications of a mental illness (for example, low output cardiac failure in severe anorexia nervosa), admission to a paediatric or medical ward will be needed.

If there is a low risk of any danger, and the child and family are cooperative, the psychiatrist can prescribe appropriate medication for the mental illness (see Chapter 9.5.5), give advice on the management of the symptoms in the short-term, and arrange for early follow-up and review in the outpatient clinic.

Acute behavioural disturbance (no mental illness)

Very often, the situation is defused by the psychiatrist's assessment, and the child's behaviour settles. In this case, the psychiatrist will need to decide whether there is a high risk of further episodes. If not, she may simply give advice to the family about how to avoid or deescalate future situations, and inform the child's GP about the incident. However, if she has identified significant ongoing issues underpinning or precipitating the child's disturbed behaviour, she will need to give the family advice about how to try and deal with these. For example, if the child has difficulties with family conflict, the psychiatrist could recommend referral for outpatient family therapy or anger management; if stress at school was linked to the outburst, she might advise the parents to arrange a meeting with the child's teachers; or she could advise a parent to seek help for their own mental health problems (such as depression or addiction) or social problems such as overcrowding or financial difficulties.

If the disturbed and violent behaviour continues in spite of the psychiatrist's intervention, a decision must be made about how best to regain control of the situation. The police may need to be called in, to provide sufficient manpower to safely control the violent behaviour. It may be possible to change the environment by calling in significant other people (for example, another relative) who can calm the child. The use of medication needs very careful consideration.[4] Children may react paradoxically to sedatives and tranquillizers, and the use of medications with the risk of respiratory suppression is not recommended unless life support equipment (and staff) is available. However, sometimes it may be possible to persuade a child to accept an appropriate dose of oral medication such as lorazepam or midazolam, and this may be beneficial.[5]

Child abuse

Although it is not common for child abuse to present as a psychiatric emergency, nevertheless it is vitally important for any psychiatrist on call to be able to recognize this, and manage the situation according to local child protection procedures. If the child is making allegations of abuse, these will need to be discussed with the parents/carers, and then it will be necessary to consult with other professional colleagues (including the police, social services, or paediatric staff) to decide on how best to care for the child. The welfare of the child must always be paramount, even if this involves the child being taken to a place of safety such as a paediatric ward or to alternative accommodation while further investigations are carried out. Clear and accurate notes of all aspects of the assessment are vital as they may be needed for legal proceedings later. (The assessment of possible child abuse is considered further in Chapter 9.3.3.)

Emergencies in paediatric/medical wards

Psychiatric assessment and advice is needed at times for children showing acutely disturbed behaviour on paediatric wards, as well as for deliberate self-harm.

The child may be acting bizarrely or aggressively. The psychiatrist will need to make a thorough assessment of the situation, including the background of the child and family and the medical aspects of the case. He will then need to assess whether the behaviour is part of the physical illness, (e.g. hypoglycaemia, hypoxia, delirium, pain), a primary mental illness (e.g. psychosis, somatoform reaction), the side effects of medication, or an acute behavioural reaction. Management will need to be discussed and agreed with the paediatric staff, and shared with the family if they are available. The psychiatric assessment and care plan should be carefully written up in the medical file to ensure continuity through shift changes of the ward staff.

The use of sedation needs to be given careful consideration, and used only if other strategies fail (such as finding out from the child the cause of any upset and talking it over; moving the child to a quieter setting; calling in the parent to help reassure or pacify the child). Oral lorazepam or midazolam are useful drugs of first choice.

If the primary problem is one of mental illness and the child cannot be cared for effectively and safely in a paediatric ward setting, bearing in mind the needs and safety of the other patients, then transfer to a psychiatric ward will need to be arranged as soon as possible.

Follow-up visits or phone calls to the ward will be needed, to make sure the child is improving or to reconsider the diagnosis or treatment.

Psychiatric problems in paediatric or medical wards are considered further in Chapter 9.3.4

Step 6. Follow-up and communication with other professionals

Effective management of the emergency situation can be undermined by inadequate follow-up arrangements or poor communication between professionals and agencies. It is therefore very important

that the on-call psychiatrist communicates with the child's general practitioner, the team providing routine care for the child, and any other relevant professional involved as soon as practicable, usually the following day. This will hopefully ensure that there is a seamless provision of care for the child and the family, and prevent or forestall future emergencies.

Consent and confidentiality issues

(a) Consent

Psychiatrists who assess and treat children and adolescents, like all mental health professionals, have a duty of care to ensure that the welfare of children is protected. They must also work within the limits of the laws of the land. This means that care and attention must be paid to the rights of children to consent to their treatment, while at the same time taking into account the circumstances, their mental capacity, their parents' views and the risks and necessity for treatment. In many cases, the sharing of information with the child and the family, and their involvement in the decision about the best care will ensure that an agreement can be reached. However, there are other occasions when either the child or their parents refuse to cooperate with treatment felt to be essential by the psychiatrist. If the child is refusing but the parents consent, it is usually possible to treat the child under parental consent, or for adolescents, to use other legal means (for example, the mental health legislation to allow the hospitalization and treatment of a mental disorder). If the child is consenting to treatment, but the parents refuse, care proceedings may need to be considered. If both child and parents are refusing treatment, the situation is very difficult and a second opinion on the need for treatment and legal advice may have to be obtained before anything further can be done.

(b) Confidentiality

The confidentiality of the doctor–patient relationship, as mentioned in the Hippocratic Oath, is a fundamental prerequisite for patients being able to trust their doctor with sensitive information. However when working with children and adolescents, confidentiality is complicated by the need to share information with parents, in order for them to fulfil their responsibilities to their child, and also with the other agencies which may be involved in providing services for the child (education, social care, etc.). Children and their families should be informed of the scope of any promises of confidentiality at the beginning, and it is always good to ask for the child's or adolescents' consent to share information where this is necessary and in the best interests of the child. Breaches of confidentiality may be clinically or legally necessary (such as where an adolescent with a mental illness is exposing them self to risk, or when child abuse is suspected).

Tan *et al.*[6] provide a full discussion of these issues, and decision-making algorithms. Effective management of an emergency should result in the child receiving appropriate care and treatment, the parents having a greater understanding of the issues associated with the disturbed behaviour, and any other professionals feeling supported by the psychiatrist's advice and intervention. This will all reduce the likelihood of further emergency situations arising for that child and family.

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9.5.9 The child psychiatrist as consultant to schools and colleges

Simon G. Gowers and Sian Thomas

Introduction

Those who provide public services for children and young people may have a role in the identification, prevention, and within reasonable parameters, the treatment of mental health problems. Social services and education in particular have a key responsibility to safeguard the physical and psychological health of children and identify potential areas of avoidable harm, including those which may develop within their institutions.

There is a well-recognized mismatch between the rates of child mental health problems identified in epidemiological studies and the number of children referred to child and adolescent mental health services (CAMHS). School staff will often be in the best position to identify unrecognized difficulties and also to help children and their families address prejudices associated with referral to CAMHS, though they may need training and help to do so.

The responsibilities of teachers have been confirmed by schools' inclusion within the broad concept of CAMHS in a number of countries. In the United Kingdom, the Health Advisory Service (now the Health and Social Care Advisory Service—HASCAS), proposed a model, subsequently adopted by the Department of Health of a tiered approach to service provision, in which schools, alongside primary medical care and social services formed the first Tier.[1] Within this model schools have been seen as offering unique opportunities to identify problems, provide simple assessments and refer up to more specialized tiers as judged appropriate and in negotiation with caregivers. Teachers though, often feel inadequately trained to fulfil this role and look to other professionals, including psychiatrists to advise and support them. Fortunately there are a number of professional roles, some employed within

education and some outside, forming a bridge between education and mental health services. Some of these roles vary in their detail between countries, but most developed countries will have professionals (possibly with different titles) filling roles comparable to those in the United Kingdom.

It is important to note that CAMHS generally work as multidisciplinary teams, hence any support and liaison may be offered by a range of professionals and not exclusively by psychiatry. One of the HAS recommendations was the creation of a new professional group—the primary child mental health worker—with the particular aim of liaising between Tier 1 and Tier 2 services. The following are some of the professionals involved in the interface between child mental health and education:

Primary child mental health worker

A practitioner, often with a mental health nursing background, employed either by education or CAMHS with the specific brief to liaise between the two in identifying children with mental health needs.

Special educational needs coordinator (SENCO)

Employed by the school as a teacher, the role of the SENCO is primarily to develop effective ways of identifying and removing barriers to learning, which may result from intellectual retardation, physical, or mental health problems. Alongside primary child mental health workers they have a role in the identification, management, and referral of children as well as a responsibility to contribute to in-service training for teachers.

Educational psychologist

Educational psychologists (EP) provide assessments of special educational needs. In the United Kingdom a consultation model has been adopted, whereby the Educational Psychologist meets with the person who has raised concerns as they are likely to be the person most motivated to bring about change. The new model recognizes that teachers are often skilled in assessing pupil attainment, learning styles, behaviour, strengths, and weaknesses. EPs have an important role in early identification and intervention and aim to promote child development and learning through the application of psychological theory using information gathered within a wider ranging context.

Education welfare officer (EWO)

In the United Kingdom, each school has an EWO assigned, to provide a support service to families and schools to help them meet legal obligations related to a child's education. They work with parents/carers to monitor attendance, with schools to consider courses of action of benefit to poor school attendees and with other agencies (e.g. health, social services, police, and youth offending teams) to provide a suitable programme that will help the child return to full-time education.

School nurse

An integral part of the school health team, the school nurse's responsibilities include supporting children with complex health needs, running immunization programmes, providing drop-in clinics, parenting programmes and bed-wetting clinics, assessing the health needs of every child on starting school, and providing health schemes for young people.

Learning mentor

Learning mentors have a broad remit including supporting the safe and effective transition from primary to secondary school, supporting provision for pupils with special educational needs and developing a relationship with identified pupils, based on a trusting individual relationship.

Connexions advisors

Primarily concerned with those in the 13–16 age range, connexions advisors offer one-to-one support and guidance similar to that previously carried out by careers officers. There is a strong emphasis on surveillance and monitoring. Young people who are seen to be 'at risk' of dropping out of education or who present behavioural problems are a priority for intervention. Personal advisers act as advocates, especially for those who are vulnerable or who have special needs.

Teachers' training in mental health

Despite their responsibility for identifying mental health problems, teachers in many countries are offered little specific training in this area. In the United Kingdom, most post graduate certificate of education courses offer only a very small amount of time, perhaps as little as a half day to the teaching of special educational needs. A survey of SENCO'S training and their wish for further teaching about mental health issues[2] revealed a significant lack of training. Many had no training in 3 years. In contrast they showed a great willingness to receive more and welcomed liaison from CAMHS professionals. There have been a number of useful initiatives to improve teachers' experience including the National Healthy Schools Programme[3] which aimed to improve learning by reducing emotional and health inequalities using a whole school approach; this involved improving the emotional literacy not only of pupils but of staff and parents too.

Developing a school liaison service

Establishing a liaison service between CAMHS and a school can have a number of benefits including:

- Early identification of child mental health problems
- Information sharing
- Monitoring and evaluation of treatment e.g. for attention-deficit hyperactivity disorder (ADHD)
- Establishing pathways of referral to higher tiers of service
- Offering school-based interventions for common problems
- Promoting the development of social skills and positive self-esteem.

The majority of CAMHS services do work with schools, the nature of the intervention ranging from consultation and support for school staff to direct work with children, including observation and assessment. However, joint working between CAMHS and schools has a record of patchiness across the United Kingdom, with a lack of key personnel often leading to a fragmented service.

Good examples of joint practice are characterized by secondments between organizations, shared working environments, a clear understanding of the different roles and expertise of team members, and a shared vision of joint working.

Where good practice is operating, schools are often faced with anxieties around short-term funding for specific projects, for example, the recent initiatives 'City Education Action Zones', 'Health Action Zones', and the 'Healthy Schools Standards'.

Schools in the United States tend to operate within multidisciplinary settings and research suggests that these are effective in breaking down professional barriers and also addressing the stigma associated with a young person being referred to external agencies such as CAMHS.

The provision of a key mental health worker within the school facilitates better communication between services and helps develop a greater understanding of how the culture of a school operates. Integrated links between CAMHS and the local authority, educational psychologists, behaviour and emotional support teams, and education welfare promotes a cohesive and collaborative service for children.

Practical issues

In order for a CAMHS service to establish an effective working link with a school, there are several issues to address:

(a) Gaining the cooperation of all the staff

Commitment of all staff (and indeed parents) rather than just one interested teacher is crucial. Effective prevention, treatment, and referral pathways require a 'whole school' approach.

(b) Negotiating realistic aims

Child mental health problems are common and often long-lasting; a realistic balance should be struck between prevention and management.

(c) Establishing a level of service

Both the school and CAMHS should be clear about who is providing the service, at what frequency and the expected level of commitment on both sides. There should be perceived benefits to the school and CAMHS. Does the service provide an urgent referral component or not? Who is the named contact?

(d) Confidentiality

Schools say that policies around sharing information act as barriers to effective joint working and so there is a need to determine a process whereby a joint strategy on confidentiality is agreed. At an individual level, young people can expect that private discussions on personal matters should be kept confidential unless they are told otherwise. However, teaching staff should not give unreserved assurances on confidentiality as these may have to be breached if the young person discloses information which leads an adult to believe that they or others are at risk. Sometimes the teacher will need to share information with others in the staff team in the young person's interests, or for supervision purposes. On occasions, (for example, where there is a serious risk of self-harm) it will be necessary to contact parents, but in these circumstances the young person should be told explicitly what has been shared with whom and why. Confidences should not be breached to other pupils.

(e) Pitfalls

There are several dangers of providing a mentoring/counselling service for the inexperienced teacher. Some of the commonest are: becoming over involved (emotionally and with time), giving unconditional guarantees of confidentiality, and dealing inappropriately with pupils concerns about another pupil. Obtaining advice or supervision from a more experienced member of the team or a CAMHS liaison worker is the most effective way of addressing these difficulties.

What do teachers need to know?

(a) Education about mental health problems

Surveys suggest teachers want to understand the common presentations of mental health problems in childhood, how they affect children's behaviour, and their impact on learning. They are often uncertain about aetiology and prognosis. Where disorders have a genetic component to their aetiology, teachers often mistakenly believe that this diminishes the potential impact of school-based interventions. They like to understand the distinction between generalized and specific learning difficulties.

Distinguishing disorder from bad behaviour is especially complex, particularly when attempting to differentiate between what a child can't or won't do. This is commonly an issue with hyperactive children whose attention in school is poor.

(b) Identification/detection

Teachers benefit from guidance on the detection of disorders, by learning about common symptoms and behavioural phenotypes. They can be helped in this by being aware of groups at risk and by the use of screening instruments designed to be used in school. Commonly used measures include the Conners Teacher Rating scale (CTRS)[4] and the Strengths and difficulties questionnaires.[5]

(c) Interventions/treatments teachers can deliver in school

These may include simple counselling interventions, e.g. to address anxiety at exam times or supporting a child during parental separation or after a bereavement. The teacher's role may involve supporting the administration of medication within school (e.g. for hyperactivity). This treatment (and the child's motivation to take it) can be severely undermined if teachers do not support its use.

(d) Knowledge/understanding of treatments given by CAMHS

Many myths about child mental health problems and their treatment may be shared by teaching staff. These include the aims, benefits, and likely adverse effects of medication. Some will mistakenly believe, for example that drugs for hyperactivity are sedative and will turn a child into a 'zombie'. Stigmatizing attitudes to inpatient child psychiatry units may impair a child's rehabilitation after admission.

(e) Pathways of referral

Schools should be clear about which problems should be referred to which agency. For example, acute self-harm should be referred to the accident and emergency department of a general hospital, whilst child protection issues should be referred to social services.

Specific issues posing a challenge for schools

A major concern for a school is how to manage disruptive disorders and their impact on other children. A disorder such as Tourette's syndrome will excite younger children and they will be easily distracted. Those whose attention is poor, who are more impulsive or who are low achievers are particularly vulnerable, whilst the subject may find their behaviour reinforced by their unexpected celebrity and status as the 'class clown'.

Appropriate behavioural management can be difficult to institute without it being punitive or unwittingly reinforcing. This applies to

conduct problems and for example where teaching staff may offer lunchtime supervision of a child with an eating disorder.

It is often difficult to address minor self-harm sympathetically without reinforcing the behaviour. Similarly the wish to be sympathetic towards those with eating disorders may be tempered by concerns to avoid 'epidemic' dieting.

The school's role in child protection

Schools in the United Kingdom have a responsibility to safeguard and promote the well-being of pupils under the Education Act of 2002[6] and, where appropriate, under the Children Act 1989.[7] Each school has a designated lead for child protection and if staff have concerns about the safety and well-being of a child they should report their concerns to them. The child protection lead will refer to the school Child Protection Policy and then directly to Children's Social Care services as necessary.

A guidance document 'safeguarding children and safer recruitment in education' was produced in the United Kingdom in 2006[8] and is a consolidated version of earlier guidance material. It focuses on the recruitment and selection processes, vetting checks, and duties for safeguarding and promoting the welfare of children in education. The document also forms a guide to inter-agency working. The guidance explains that a school should 'create and maintain a safe learning environment' and have the appropriate arrangements in place. Child protection arrangements, pupil health and safety, and bullying are all subject to statutory requirements. The guidance directs that if a child is the subject of an inter-agency child protection plan, the school should be involved with the preparation of that plan.

Through the delivery of personal, health, and social education (PHSE) the school may provide opportunities for children and young people to learn about keeping safe. Pupils should be taught to:

- Recognize and manage risk in different situations and then decide how to behave responsibly
- Judge what kind of physical contact is acceptable and unacceptable
- Recognize when pressure from others (including people they know) threatens their personal safety and well-being and develop effective ways of resisting pressure.

School-based intervention strategies

Primary prevention

A healthy school promotes physical and emotional health by providing accessible and relevant information and equipping pupils with the skills and attitudes to make informed decisions about their health. It understands the importance of investing in health to assist in raising levels of pupil achievement and improving standards. It also recognizes the need to provide both a physical and social environment that is conducive to learning.

The National Healthy School Standard was part of the Healthy Schools programme, led by the DFES and the Department of Health.[9] Launched in October 1999, it offered support for local programme coordinators and provided an accreditation process for education and health partnerships. It provided a model of partnership working between the health service and schools, with the aim of promoting a coherent and holistic message about the importance of a healthy lifestyle.

The standard covered four key themes:

- Personal, Health, and Social Education (PHSE).
- Healthy eating
- Physical activity
- Emotional health and well-being (including bullying)

PHSE is now included in many countries' teaching curricula. This provides education on social and emotional development and citizenship, including the individual's place in society, responsibility, and rights. In The United Kingdom, the PHSE syllabus now has sessions on mental health issues including the use of drugs and alcohol and the links between drug misuse and mental illness. It also covers self-harm and suicidal behaviour.

Social and Emotional Aspects of Learning—(SEAL) is a whole-curriculum framework for teaching social, emotional, and behavioural skills to primary school children in five areas: self-awareness, managing feelings, motivation, empathy, and social skills. In 2004, the scheme was piloted in 250 schools in 25 authorities in the United Kingdom with a subsequent planned extension to high schools.

Secondary prevention

Solution Oriented Schools (SOS) is an approach used in many United Kingdom local authorities, comprising training and resources to support the whole-school promotion of positive behaviour. The focus is on establishing small steps that can be taken to resolve conduct problems, attendance issues, poor peer group interactions and negative attitudes to learning.

The approach invites staff to consider: 'What works in school?'. It encourages them to take a pragmatic approach; learning from what is working, leaving behind practice that is failing to pay-off, recognizing 'the problem' as the problem (not the child, teacher, or professional), and building on strengths that each individual brings. It stems from the principles of solution-oriented brief therapy which focuses on finding and creating solutions to a problem whilst spending little time on the problem itself.

Circle time was developed in the 1930s particularly for primary schools, as a forum for children to share views and concerns about issues arising in school (such as bullying) or outside (within the family or neighbourhood). It is still widely practised.

Mobile phone, text, and Internet-based initiatives

A number of education authorities have developed pilot schemes using new technologies such as online counselling and support services. Various websites offer qualified counsellors and other support services. Services are confidential and young people book in for their session of online chat. In addition the young people can often access a Frequently Asked Questions area. Some sites have counsellors who can make referrals to CAMHS for the young people who access them.

Other initiatives include a pilot scheme in Wales in which pupils used mobile phones to text their school nurses for health advice. This short-term project ran in office hours, offered students instant help and provided those who might be wary of approaching adults with their problems face-to-face the chance to do so anonymously.

In Liverpool, *The Health and Education for Life Project (HELP)* was set-up in 2003, as an action research project that worked in

schools to change pupils' attitudes towards mental health issues and to provide coping strategies.

Meeting the needs of children with special educational needs

While most pupils with complex needs are educated in special schools, where the special needs of children can be met by a mainstream school, they are often taught in this setting. Attitudes to inclusion have changed over time. The latest draft guidance in the United Kingdom moves away from the inclusion drive of 2004 and advises councils that they should provide a 'range of provision' for children with special educational needs. The national curriculum requirements may often impact negatively on the experiences of a young person with specific learning needs; the challenge for teachers is to create a stimulating, engaging programme of study whilst still meeting the national requirements. The use of teaching assistants in a creative and well-planned way can facilitate the delivery of lessons in an inclusive setting. Ultimately, the needs of the individual child should be of primary concern in the inclusive–exclusive decision-making process.

Some special schools offer outreach to the mainstream, so that expertise can be shared and support given for inclusive practice. Some local authorities name specialist schools for each area to meet the needs for example, of autistic young people. Where a young person lives too far from the locality of the specialist school; the specialist school may adopt an advisory role.

Special schools attempt to offer a tailored and focussed response to the needs of specific groups of young people; it is arguable that a young person who has for example, high functioning Asperger's syndrome will feel more 'included' in a special school setting where his differences are less noticeable. The social exclusion that such a young person may experience in a mainstream setting can have detrimental effects on their progress. Successful inclusive education relies on a school approach that creates an inclusive culture; develops inclusive policies, and evolves inclusive practices.

Children and young people with emotional and behavioural difficulties (EBD) present a major challenge to schools attempting to become fully inclusive organizations. Through emotional literacy programmes such as SEAL, anger management groups, social skills, and self-esteem groups, schools can offer a variety of behavioural interventions that support an inclusive experience.

Hospital schools or Medical Pupil Referral Units offer education to young people who are unable to attend school because of medical needs. The term 'medical needs' includes those with mental illness; anxiety, depression, and school phobia. There is a strong emphasis on a strategic planning framework which ensures a continuum of provision; a focus on close liaison with all parties and the development of a robust reintegration plan.

The role of CAMHS within special education has a higher profile than in many mainstream educational provisions; children should all have statements of special educational need and therefore should be reviewed regularly. The reviews of children with a statement of emotional and social need or with autistic spectrum disorders (ASD) tend to require the involvement of a CAMHS worker; subsequently good relationships are developed through close and regular interactions with educationalists. CAMHS may offer consultation and advice or direct intervention with the child or group of children in a special educational setting.

The psychiatrist as advisor to higher/further education

Recent years have shown a growing awareness of the unmet needs of students in higher education.[10] Although higher education institutions often have quite sophisticated pastoral and counselling provision in place, they may need to consult with mental health services regarding issues with specific students. The psychiatrist should be aware of the following particular issues in relation to this group:

- The vulnerability of young people living away from home for the first time.
- Recent expansions in student numbers. In a number of countries, greater training opportunities for young people have resulted in access to higher education no longer being restricted to those from privileged backgrounds. One adverse consequence of this otherwise desirable state of affairs is that those with greater risks (or indeed histories) of mental health problems may take up places at colleges and universities.
- Vocational courses. Some courses (such as medicine, nursing, and social work) may restrict those suffering with particular mental health problems. The psychiatrist may have a duty to report such issues as, for example, drug dependence. Related to this a student may be reluctant to disclose a problem which would have implications for continuing on their course.
- Interface/communication issues. Young people living away from home at college may be vulnerable to falling into any gap that might exist between child and adult services and between services local to their home and those at their college. Effective communication between service providers is of paramount importance.
- Confidentiality. Students are often concerned about confidentiality from their parents, their peers, and their college's academic staff. As with the examples given earlier in this chapter, confidences should only be breached on a strictly 'need to know' basis.

Conclusions

As participation in education is almost universally compulsory for children, schools are in a unique position to offer prevention and identification of child mental health problems. Effective practice requires good liaison with CAMHS. There are a number of obstacles to effective working but recent times have seen a number of examples of good practice and policies to support these.

Further information

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The National Healthy Schools Programme. www.healthyschools.gov.uk

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SECTION 10

Intellectual Disability (Mental Retardation)

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10.1

Classification, diagnosis, psychiatric assessment, and needs assessment

A. J. Holland

Introduction

The general principles developed during the latter part of the twentieth century and continued into the twenty-first century guiding support for people with intellectual disabilities remain those of social inclusion and the provision of services to enable people to make, as far is possible, their own choices and to participate as full citizens in society. These are articulated in national policy documents, such as the White Paper for England, 'Valuing people[1] and also at an international level in the UN Declaration on the rights of people with disability.[2] However, given that people with intellectual disabilities represent a highly complex and heterogenous group with very varied needs, in order for such objectives to be achieved, a range of community based support and interagency and interdisciplinary collaboration is required. It is acknowledged that people with intellectual disabilities experience considerable health inequalities with the presence of additional disabilities due to the presence of physical and sensory impairments and co-morbid physical and mental ill-health, much of which goes unrecognized, and also the occurrence of behaviours that impact on their lives and the lives of those supporting them.[3,4,5] In the twenty-first century, few would now challenge the objectives of social inclusion and community support. The tasks for Government and society are to provide special educational support in childhood and also support to the families of children with intellectual disabilities, and the necessary range of services to meet the social and health needs of this diverse group of people in their adult life. This includes enabling adults with intellectual disabilities to gain meaningful support or full employment and to exercise their rights as citizens and to participate fully in society. To achieve such objectives there is a need to be able to characterize the nature and level of need, to establish the presence and significance of co-morbid illnesses and/or challenging behaviours, and to organize and provide support and services to meet such identified needs.

This complexity of need has meant that no single 'label', such as 'intellectual disability', can adequately describe this group of people. What individuals have in common is a difficulty in the acquisition of basic living, educational, and social skills that is apparent early in life, together with evidence of a significant intellectual impairment. However, for some this may be of such severity that, for example, meaningful language is never acquired and there are very substantial care needs. For others, there is the presence of subtle signs of early developmental delay, and evidence of learning difficulties that only becomes clearly apparent at school when there is an expectation that more sophisticated skills will be acquired. The nature and extent of disability and of any functional impairments in general, distinguishing those people with intellectual disabilities from those with specific learning difficulties, such as dyslexia.

In infancy and early childhood, the reason for any apparent developmental delay needs to be established. This is primarily the responsibility of paediatric and clinical genetic services. Such information helps parents understand the reasons for their child's difficulties and may guide, in a limited way, an understanding of future needs and potential risks. Later in childhood, the nature and extent of a child's learning difficulties and a statement of special educational needs is the main task and later still, the main focus may be the assessment of longer-term social care needs. Throughout life, there may also be questions about a child's or adult's behaviour or mental state or the nature and extent of physical or sensory impairments and disabilities. The role of assessment is essentially to determine need and to inform the types of intervention and treatments, whether educational, medical, psychological, or social, which are likely to be effective and of benefit to the person concerned. Systems of classification provide useful frameworks for such assessments.

Classification

The term 'classification' is unfortunate as it carries with it the stigma associated with previous legislation (e.g. Mental Deficiency Act, 1913) and the associated history of institutionalization consequent upon the eugenics movement at the beginning of the twentieth century.

However, systems of classification are an important way of organizing information and thereby enabling the reliable passing of that information to others and providing a framework to guide intervention. Whilst there are clear strengths to this process, any system of classification has serious limitations. It will tend to focus on a few particular characteristics to the potential exclusion of others, and none can impart a truly comprehensive picture. Methods of classification have inevitably changed over time in an attempt to better clarify the key issues and to minimize stigma that might be associated with any given label. However, the central principle of any system of classification is to bring order to knowledge in a manner that may then enable further advances or the instigation of interventions that previous research has shown to be effective. There is no single universal system—the system of classification used depends on the reasons for its use. These may be as diverse as being predominately administrative or for the purposes of guiding intervention and the use or not of specific treatments.

Classification systems also differ with respect to whether they are dimensional or categorical in nature. Intellectual disability illustrates this difference in that measures such as those obtained from IQ tests are clearly dimensional and continuous whereas labels such as 'intellectual disability' or the identification of particular syndromes are categorical. More recently such obvious categorical distinctions have begun to break down as the genetic basis for syndromes are more clearly elucidated. For example, in fragile X syndrome there is variation in the extent of the number of repeat sequences in the FMR-1 mutation, both within carrier and affected individuals that influence whether or not an intellectual disability is likely to be present.[6] Various different systems of classification are examined below and the relationship between assessment and classification is considered.

Mental retardation (DSM-IV)

From January 1st 2007, the previously named American Association on Mental Retardation changed its name, replacing 'mental retardation' with the term 'intellectual and developmental disabilities'. This followed similar changes in other organizations. However, in DSM-IV[7] the term 'mental retardation' remains for the moment. This standard diagnostic system provides a framework for multiaxial diagnosis with Axis II for personality disorders and mental retardation. Table 10.1.1 summarizes the DSM-IV criteria for mental retardation. The focus is not primarily one of aetiology but rather of quantifying the extent of 'mental retardation' through defining the level of intellectual impairment and listing the range of possible adaptive functions that might be impaired. The definition makes explicit that the onset is in the developmental period and that mental retardation is the final common pathway of a number of potential aetiologies. Significant sub-average intellectual function is defined as an IQ of 70 or below (using standard IQ tests). The IQ is also used to help determine the level of mental retardation (mild, moderate, severe, or profound).

The use of such a multi-axial system recognizes the fact that intellectual disability is a disorder of development, which is separate from other mental disorders, such as mental illness (Axis I), general medical conditions (Axis III), and which may be associated with particular psychosocial and environmental problems (Axis IV). Thus, the process of formulation requires that all these broad domains be considered in arriving at an understanding of an individual's particular difficulties.

International Classification of Functioning, Disability and Health (ICF)

In 2001, the World Health Organization published the International Classification of Functioning, Disabilities and Health (ICF).[8] This is a complete revision of the International Classification of Impairments, Disabilities, and Handicaps.[9] The latter classification was an advance at that time in that it had attempted to overcome the limitations of other methods of classification (particularly with respect to chronic disability) and, most importantly, aimed to guide intervention at several levels and in a more holistic manner than classification systems that were primarily focused on diagnosis, had been able to do. In this context, intellectual disabilities could be conceptualized at different levels. In the case of impairment, the organ system involved is that of the central nervous system. It is the impairment of this system for genetic, chromosomal, or environmental reasons that have primarily affected the acquisition of developmentally determined skills and the ability to learn. The associated disability is the effect of the impairment on a person's ability to learn and acquire new skills that come with development. The exact nature and extent of the disability may not only include the impact of an intellectual disability but also physical and sensory disabilities. The extent to which a given

Table 10.1.1 Summary of the diagnostic criteria for mental retardation (DSM-IV)

A. Significant subaverage general intelligence
B. Significant limitations in adaptive functioning in at least two of the following:
- Communication
- Self-care
- Home living
- Social/interpersonal skills
- Use of community resources
- Self-direction
- Functional academic skills
- Work
- Leisure
- Health
- Safety

C. Onset before age 18 years of age

Note

- Significant subaverage intellectual functioning is defined as an IQ of about 70 or below. The choice of testing instrument should take into account the individual's socio-economic background, native language, and other associated handicaps
- Adaptive functioning refers to how effectively individuals cope with common life demands and how well they meet the standards of personal independence expected of someone in their particular age group, sociocultural background, and community setting. Adaptive behaviour may be influenced by individual and/or environmental factors including the presence or not of additional mental or physical disorders. Information on adaptive behaviour should be gathered from one or more independent sources
- The degree of severity of mental retardation may be specified on the basis of intellectual impairment taking into account other aspects of functioning
  - Mild mental retardation: IQ level 50–55 to approximately 70
  - Moderate mental retardation: IQ level 35–40 to 50–55
  - Severe mental retardation: IQ level 20–25 to 35–40
  - Profound mental retardation: IQ level below 20 or 25

impairment results in a loss of function (disability) may well be influenced by the extent and nature of interventions such as special education, or the correction of hearing loss through the use of a hearing aid. The final level, that of 'handicap', is a consequence of an interaction between the disability and the extent to which support is available or environmental adjustments made. It is a measure of disadvantage that can be ameliorated through, for example, the presence of carers to enable individuals to go out, or environmental modifications (e.g. wheelchair ramps) that diminish the impact of physical disabilities.

The ICF attempts to take classification further and to conceptualize 'disability' within the context of society as a whole, recognizing that everyone can experience disability at one time or other—the stated aim of the ICF is to 'mainstream the experience of disability and recognize it as a universal human experience'. The intent is to encourage those using the ICF to take into account more fully the social aspects of dysfunction and not to see disability as only a medical or biologically determined dysfunction. This means of classification aims to enable the recording of the environmental effects on an individual's functioning. The ICF itself is divided into two Parts. Part 1 is concerned with 'Functioning and Disability' and Part 2 is concerned with 'Contextual Factors'. Each of the two components are expressed in both positive and negative terms in order to emphasize what a person is able to do as well as what he/she is not able to do. The ICF is more complex and more comprehensive than the 1980 WHO system of classification attempting to provide a conceptual framework that forces a much wider understanding by bringing together more comprehensively social and biological models of disability. In doing so, it does what a sound formulation should do, moving from the limitations of a diagnosis to an understanding of the individual within a biological, social and environmental context. This is illustrated in the diagram in Fig. 10.1.1 taken from the ICF.

As with the 1980 'Impairments, Disabilities and Handicaps' means of classification, the ICF is seen as complementing other WHO classification systems, such as the ICD-10.[10] The ICD-10 is focused on disease and the ICF on 'components of health'. The latter, in doing so, provides an appropriate means for characterizing need and for ensuring that people with chronic disabilities have such needs met in the context of their individual human rights and also based on rights established through national legislation. However, as knowledge has increased, for example, about the nature and extent of physical and psychiatric co-morbidity affecting people with intellectual disabilities, so then has the need to use different and other relevant systems of classification increased.

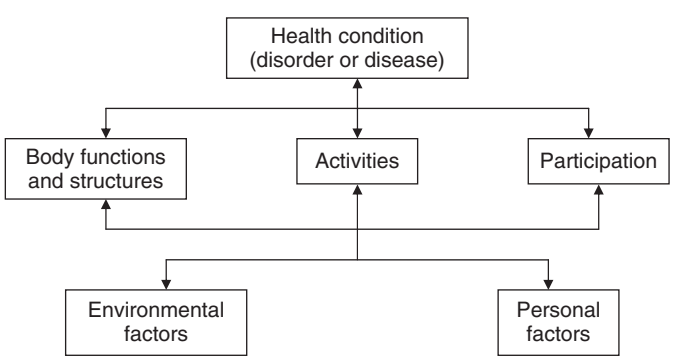

Fig. 10.1.1 Interactions between the components of ICF.

Such different approaches provide the necessary frameworks for a rigorous and comprehensive formulation of a person's needs and to guide treatment of any identified co-morbid illness.

Assessment

Assessment is a task that is undertaken to address specific questions and is informed by the relevant theoretical knowledge and conceptual framework. The type of assessment undertaken therefore, depends on the issue in question and in turn, on both the relevant theoretical background and the appropriate systems of classification, where they are required. The above systems of classification provide a broad framework for considering need. However, in the field of intellectual disabilities a truly holistic assessment will frequently require different theoretical perspectives because of the variability in and complexity of need. The precise form of any focussed assessment will depend on the reasons for undertaking the assessment. The nature of the assessment will vary considerably if it is primarily to determine a person's social care needs, as opposed to the reasons for a particular problematic behaviour. Even with the context of challenging behaviour, the assessments undertaken will vary. Increasingly, the skills expected of those working in community teams supporting people with intellectual disabilities is to be able to recognize what assessments are relevant and required and to be able to undertake such assessments in the community setting where the person with intellectual disability lives. For the sake of clarity, a distinction is made below between those assessments that are fundamentally directed at characterizing the persons, intellectual disability and those whose main focus is on psychiatric and behavioural aspects.

Intellectual disability, its characterization and causes

The term intellectual disability is not in itself a diagnosis as it does not inform in any reliable way about aetiology, prognosis, or specific treatments. Rather, it refers to a clinical state that is developmental in origin and affects intellectual and social functioning. The diagnosis is the identification of the underlying cause for the observed developmental delay. The extent of early developmental delay can be measured against standardized developmental scales (e.g. Bayley or Griffiths Developmental Scales), and during childhood and adult life. There are also well-established specific assessments of intellectual, language, and functional abilities. These assessments provide a profile of a person or group of people that can be compared against the norms for age and a given population. Adaptive functioning has to be measured against what would be expected for a person of that age, and the social and cultural experiences of the person have to be taken into account. The Wechsler Scales for IQ, and the Vineland Adaptive Behaviour Scales[11] or the revised Adaptive Behaviour Scales of the American Association for Intellectual and Developmental Disabilities[12] for characterizing functioning are established instruments for the measurements of these abilities and for which there are normative data for comparison.

Possible single major causes for an intellectual disability are covered in more detail in a separate chapter and are mainly the province of paediatrics or clinical genetics. However, where there is clear evidence for developmental delay in childhood and for the person having an intellectual disability and no obvious cause has been previously established, further investigation is indicated. This will be informed by the clinical history and by physical

examination (e.g. presence of a family history of similar disorder or not, evidence of the physical characteristics of a particular syndrome). As part of this process, the developmental history should also establish whether the developmental profile is not only characteristic of an intellectual disability but also whether the person meets criteria for having an autistic spectrum condition. This is important because of the high rates of autism within the population of people with intellectual disability and the implications such a diagnosis may have for the type of support.[13] Particularly where the person has evidence of dysmorphic physical characteristics, a moderate, severe or profound intellectual disability, and/or a family history of disability, chromosomal, biochemical, and/or molecular genetic studies may be indicated. With improving knowledge about the developmental differences and the different predispositions to specific co-morbidities that exist between genetically determined syndromes, establishing the cause of a person's intellectual disability is becoming increasingly important and may help inform psychiatric and psychological assessments.[14]

(a) Assessments of index problem and psychiatric diagnosis

There have been significant advances in the conceptual models used to help understand the occurrence and maintenance of problem behaviours or abnormal mental states in people with intellectual disabilities. The following four approaches are particularly relevant and each of these perspectives needs to be considered in any assessment. First, applied behavioural analytical studies have demonstrated how the occurrence of behaviours, such as self-injurious behaviour, can be shaped in the context of particular environmental or individual setting conditions.[15] This approach attempts to identify the 'functions' of behaviours. Specific behaviour (e.g. self-injurious behaviour, aggressive outbursts etc.) may, for example, be identified as being attention-maintained or demand avoidant. Secondly, some behaviours may be a consequence of arrested development. In such cases the behaviour itself (e.g. repetitive checking) may be similar to that which occurs as part of typical childhood development but it has continued into adult life. Thirdly, the behaviours may be a manifestation of co-morbid physical or psychiatric disorder. For example, as a consequence of increasing irritability and agitation associated with depression[16] or consequent upon pain or some other physical distress. Fourthly, the abnormal mental state or behaviour may be associated with the cause of the person's developmental disability. This is increasing referred to as 'the behavioural phenotype' of a particular syndrome.[14] These different models of understanding are not necessarily mutually exclusive. For example, the behaviours and psychiatric problems that commonly affect people with Prader Willi syndrome includes examples of each of the above. The increased propensity to temper outbursts and to repetitive and ritualistic behaviours are likely to be partly a consequence of arrested development and to be partly re-inforced and modified depending on environmental contingencies, the over-eating behaviour is a direct consequence of the syndrome and, if obesity is not prevented, it can lead to physical illness that may present with changes in behaviour (e.g. sleep disorders), and those with one genetic form of the syndrome have a high risk for developing co-morbid affective psychotic illness.[17,18] In case of each of these examples, rather different approaches will be required, varying from environmental change to the possible prescription of medication to treat co-morbid psychiatric illness. Assessments should therefore be informed by these different theoretical perspectives recognizing that similar 'behaviours' may have different aetiologies and that what predisposes to, precipitates, or maintains a particular behaviour and/or mental state may each be different. The challenge of assessment is to identify the developmental, biological, psychological, and social factors that relevant and the treatment/management implications.

In psychiatric practice, the referrals and the assessments are usually to determine the reasons for the occurrence of a particular maladaptive/challenging behaviour and/or apparent change in mental state or in cognitive and functional ability. The focus of the assessment is therefore to address these issues but, in doing so, it invariably requires an assessment of the developmental profile of the person concerned and consideration of the cause of any such disability. In principle, however, the psychiatric assessment of people with intellectual disability is not dissimilar to that undertaken in general child or adult psychiatry. The main differences are as follows:

1 Special care may have to be taken in assessing an individual's mental and cognitive state and whether this has changed over time and, where language development is impaired, a greater reliance may have to be placed on information from an informant;

2 a good developmental history is essential to map early development and the potential developmental origins of the individual's present state;

3 it is important to investigate the enabling and constraining aspects of a person's environment that facilitate or impinge on a person's life and thereby how environmental change might bring real benefit;

4 the possibility of multiple physical and mental health problems and the potential for complex interactions between the individual and his or her carers and the immediate and distant environment should be appreciated.

5 where co-morbidity is a possibility, evidence of a change in physical and mental state or behaviour or an exaggeration of previous states (e.g. obsessional behaviours) that might be indicative of the development of a physical or psychiatric illness must be enquired about. The development of physical illness may also manifest as behavioural change.

Psychiatric assessment is therefore invariably an iterative process, especially so with the uncertainties associated with the assessment of a person with an intellectual disability and the different conceptual models. In undertaking an assessment it can be helpful to draw a distinction between:

1 the characterization of the nature and extent of the person's intellectual disability and the identification of its aetiology;

2 the identification of the onset, nature and extent of additional problem behaviours or abnormal mental states;

3 the determination of the possible aetiological factors of the person's behaviour, such as whether there is a co-morbid psychiatric diagnosis or whether there are particular factors that might have predisposed to, precipitated and are now maintaining a particular behaviour or abnormal mental state.

A detailed history taking from both the person him- or herself and an informant covering both childhood development and

the presenting problem, a mental state examination and where indicated a physical examination, direct observation, and often detailed record keeping of particular behaviours (depending on the reason for referral) by care staff over time are the key components of a comprehensive assessment. Where referrals are about the occurrence of problem behaviours, the assessments should include a description of the behaviour as well as an attempt to identify those factors that might increase or decrease the likelihood of the behaviour occurring. These will include the identification of those factors affecting the person him- or herself and those that are particular to the environment. Specifically from a psychiatric perspective, this includes the identification of psychiatric or physical illnesses, the relationship of the index behaviour to any change in mental state or, for example, the occurrence of seizures, as well as the possible contribution of the developmental disability to the problem. For example, the presence of autism may account for observed ritualistic and obsessional behaviours and might also help to make sense of a person's aggressive outbursts that staff observation and record keeping over time has shown occurs at times of unexpected change in routine or activity. Knowledge about the chronicity of a particular behaviour might influence the understanding of the likely contributory factors that give rise to that behaviour. If the behaviour is of recent onset then the role of recent life events or the possibility that the person has developed a mental or physical illness should be investigated. However, if the behaviour has been present since early childhood, psychological models of understanding may be more relevant.

In terms of assessment instruments, a clear distinction needs to be drawn between those that are essentially descriptive in nature and those that are investigating the potential aetiology of the index behaviour. The former include, for example, the Aberrant Behaviour Checklist[19] and the latter, which are based on particular theoretical models, may include, for example, the Psychiatric Assessment Schedule for Adults with Developmental Disorders[20] (a structured assessment of mental state) and the Motivational Assessment Scale[15] (a structured assessment of the possible 'functions' of particular behaviours).

Mental state examination

Central to the practice of psychiatry in particular is the identification of specific mental phenomena that, when clustered together, are indicative of a specific psychiatric disorder. The gold standard for diagnoses are the diagnostic manuals ICD-10 and DSM-IV. Diagnostic criteria have also been modified to better fit what is observed when assessing people with intellectual disabilities. These have been based on the ICD-10, (DC-LD).[21] The process is two-staged and includes first a detailed history and mental state examination that establishes whether there has been a change in a person's mental state and if so, the characteristics of the change, and secondly, a comparison of these changes against diagnostic criteria to establish the presence or not of specific psychiatric disorders. Investigations may be necessary where there is uncertainty or other possible causes for what is observed need to be ruled out as part of the differential diagnosis.

For people with intellectual disabilities with spoken language, disorders such as schizophrenia present in similar ways to the general population, with the onset of the characteristic mental phenomena. [22] Similarly, disorders such as Alzheimer's disease can be diagnosed using very similar criteria, although the early features may be different.[23] In each of these examples, greater emphasis may have to be placed on informant observation, as some people with intellectual disabilities may have difficulty with concepts such as mood and with being able to describe the presence or not of abnormal mental phenomena.

The key to mental state examination and psychiatric diagnosis in people with intellectual disabilities is to be able to characterize the nature of any observed change. The assessment of mood, sleep, appetite, and concentration may be relatively easy as carers observe increasing distress, tearfulness, and agitation over time in the context of sleep and appetite changes, therefore giving rise to the suspicion of the presence of an affective disorder. Similarly, carers may observe personality and memory changes suggestive of dementia. The presence of hallucinations or delusions may have to be inferred by the development of odd behaviour that might reasonably be interpreted as responses to abnormal mental experiences, such as appearing to respond to auditory hallucinations. Family or other carers who have known the person for some time may well be able to describe more subtle mental state abnormalities such as a deterioration in a person's ability to express his or her thoughts indicating the possibility of thought disorder, or increasing perplexity or evidence of paranoid ideas. The interpretation of cognitive findings is more difficult because of the pre-existing intellectual impairments, but clear documentation of cognitive abilities is important as further deterioration or evidence of improvement over time can be very informative.

Needs assessment

A full needs assessment brings together the identified social, emotional, and health needs of an individual, including an understanding of the wishes of the person and views of other people who are concerned for and involved in the support and care of that individual. This whole process has, over the last few years in both mental health and intellectual disability services, become more formalized through 'needs-led assessments' and sometimes through the 'care programme approach'. Person-centred planning is the process by which this information is brought together in a manner that directs the allocation of support and any necessary treatment interventions.[24]

Social services are in general responsible for undertaking assessments at key transitions (e.g. prior to the end of statutory education) or if there is evidence that need has changed. If the person lacks the capacity to express his or her view then the question of what is in the person's best interests should be determined. The balance that this process is attempting to achieve is to respect the wishes of the person concerned and to balance an adult's right to autonomy, on the one hand, versus the need for care and support, on the other. More recently, the critical importance of assessing a person's ability to make decisions about his or her own life and the pivotal role of a person's 'decision-making capacity' in achieving the correct balance between the respect for autonomy and the need for care and support has received more attention. Legislation has been enacted first in Scotland and more recently in England and Wales that provides the framework for intervention where the person concerned lacks the capacity to make the relevant decision for him/herself. The assessment of a person's decision-making capacity requires an evaluation of a person's ability to understand and use information relevant to the decision, retain and balance the necessary information and then to communicate a choice.[25]

Although there is clearly an overlap, it can be helpful to distinguish between social care, educational, and health-care needs. In the case of children, the need for special education and for family support are likely to be central, depending on the extent of the disability and any other associated disabilities. For adults, accommodation with an appropriate level of support, meaningful daytime occupation, and companionship and practical help according to need will be key. Systematic assessments provide a structured way to determine whether a low, medium, or high service environment will best meet an individual's needs. The types and range of services that need to be provided are covered in other chapters. Health-care needs will include the same as those of the general population but high rates of physical and sensory impairments, mental health and behavioural problems, and impairment in communication are all likely to give rise to additional needs. Particularly for those people with more severe intellectual disabilities and limited and/or no language, there is also a clear responsibility for carers to ensure a healthy lifestyle, health screening, and to help identify health-related problems when they do occur. Special help may be required to ensure access to both primary and secondary health-care services and to ensure the person understands what is happening to them. This will include questions of consent to health treatment and how a person with intellectual disabilities can be best helped to give maximally informed consent or at least be able to assent when it is in his or her best interests to do so. A structured process of assessment, paying attention to the cause of the intellectual disability and its associated problems and the extent and nature of other impairments, disabilities, and handicaps will minimize the risk that health-related problems might go unnoticed or that social care needs might be ignored.

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10.2

Prevalence of intellectual disabilities and epidemiology of mental ill-health in adults with intellectual disabilities

Sally-Ann Cooper and Elita Smiley

Prevalence of intellectual disabilities

If intelligence quotient (IQ) was normally distributed in the population, with a mean of 100 and standard deviation of 15, then about 2 per cent of the population would have an IQ below 70. However, reported rates vary widely depending on the definition of intellectual disabilities used, the country and region of study, the time of the study, the age range and ethnicity of the population, and the method of population ascertainment.[1–3]

Definition

ICD-10 and DSM-IV-TR definitions of intellectual disabilities are similar:

- significantly sub-average intellectual functioning (IQ below approximately 70; mental age less than 12 years);
- concurrent impairments in present adaptive functioning; diminished ability to adapt to the daily demands of the social environment;
- onset before the age of 18 years.

IQ is a continuous measure, so when basing a definition of intellectual disabilities upon it the threshold is arbitrary, and changes in threshold can have a large impact on prevalence. For example, in the past the American Association on Intellectual and Developmental Disabilities' criteria had an IQ threshold of 84. This was subsequently changed to 70 in 1973, and then to 70–75 in 1992. ICD-10 and DSM-IV-TR definitions are not statistical constructions; the requirement of impaired adaptive functioning may half estimated prevalence rates compared with a statistical definition. Furthermore, different methods to assess intelligence and adaptive behaviour can lead to different prevalence rates.

Country and region

Prevalence of intellectual disabilities is reported to be much higher in developing than developed countries, due to socio-economic factors, although mild intellectual disabilities may possibly be less disadvantaging in non-literate societies. Iodine deficiency is the most common preventable cause of intellectual disabilities worldwide, and is indigenous in some regions of Asia and Africa. Exposure of populations to heavy metals and toxins can lower the average population IQ by a few points, hence some people who would otherwise have had low-average ability move into the intellectual disabilities range. Regions with a high level of consanguineous marriages can also have higher prevalence. For example, Tay-Sachs disease is prevalent amongst Ashkenazi communities, although premarital genetic counselling has markedly reduced it in the United States. The availability and sophistication of antenatal, perinatal, and neonatal care account for some differences between countries. Some studies show differences in ethnic groups, although the cultural suitability of the measures used may have contributed to these findings.

Time

Prevalence varies with time, due to preventative measures, and social developments. In developed countries, an increase in prevalence was seen in the early 1960s, with falling prevalence thereafter, due to developments in neonatal care with increasing survival of very low birth weight infants. Down syndrome is the most common chromosomal disorder causing intellectual disabilities, and survival rates of neonates and children with Down syndrome have increased substantially in recent decades, primarily due to access to surgery for congenital heart disease. The widespread introduction of antenatal screening for Down syndrome might have been expected to reduce the population prevalence of Down syndrome through lowering birth rate, but rising maternal age at birth, and increasing life expectation counter this, and there appears to be little change in the population prevalence. The widespread introduction of antenatal screening for phenylketonuria in the 1960s, and congenital hypothyroidism in the 1970s has virtually eliminated intellectual disabilities due to these conditions in

developed countries. Better living conditions, with individualized packages of support, and a political agenda for social justice and equality of access to health care and supports may all have contributed to the increasing life expectation for people with intellectual disabilities, although this is still lower than that of the general population. Increasing maternal age and increasing maternal alcohol consumption are expected to lead to higher birth rate of infants with foetal alcohol syndrome, and genetic causes of intellectual disabilities. Overall, there appears to have been little change in prevalence compared with 50 years ago.

Age

Prevalence is higher in child than adult cohorts, and lower in older than younger adult cohorts, with the highest prevalence at around age 10 years. This is due to intellectual disabilities having been identified by this age, combined with an earlier age of death for persons with intellectual disabilities compared with the general population. Children with the mildest intellectual disabilities are likely to benefit from additional support for learning at school, but will develop skills and experience over time, such that some no longer meet criteria for intellectual disabilities in adulthood.

Ascertainment

Reported prevalence varies with the methods of population ascertainment. For children, the ascertained prevalence doubled compared with case registers, when record linkage to education department data on educational attainments was included (giving an estimated prevalence of 1.4 per cent).[3] There was a disproportional increase in indigenous Australian children, who were possibly false positives. For adult populations, only a proportion with intellectual disabilities will be in contact with specialist health services for adults with intellectual disabilities. Ascertainment is higher when data is combined from primary health care, specialist health services, and social services, if the provision of day opportunities, supported work, respite care, funded support packages, and direct payments is considered. This is likely to identify almost all persons with moderate to profound intellectual disabilities, and adults with mild learning disabilities receiving support; it will not identify adults with IQ below 70 who no longer have impaired adaptive functioning (who therefore do not meet ICD-10 or DSM-IV-TR criteria for intellectual disabilities) and do not need support, or some people who receive all their support exclusively from unpaid carers. The assessment of IQ (culturally sensitive), adaptive functioning and support needs, plus medical assessment, of all individuals within a whole population or a representative sample would provide accurate prevalence data for that time point and area. However, this would be a substantial undertaking, in view of the tens or hundreds of thousands of participants required.

Prevalence

There have been many studies of prevalence of intellectual disabilities. For the reasons above, there is substantial variation in reported prevalence in developed countries, varying from 2 to 85/1000 general population, and there are few robust studies in developing countries. Less variability is found between studies of moderate to profound intellectual disabilities. Given the variation, it is inappropriate to provide average figures from across the studies. Interpreting the literature, we suggest that prevalence of intellectual disabilities in the United Kingdom may be in the order of 9–14/1000 childhood population, and 3–8/1000 adult population, varying with time and geography. However, it should be noted that the figure of 2 per cent is frequently assumed. Intellectual disabilities are more prevalent in males than females, particularly amongst children, young- and middle-aged adults: the reported ratio varies between 1:1 and 2:1. At older age, the gender ratio equalizes due to greater life expectancy of women compared with men (mirroring the general population), and at extreme old age, women may even outnumber men. The distribution of level of intellectual disabilities varies with age, due to the shorter life expectancy of people with more severe intellectual disabilities. Mild intellectual disabilities are associated with socio-economic status. These issues are explored in greater depth elsewhere.[1,2]

Prevalence and incidence of adult mental ill-health

Some genetic causes of intellectual disabilities have specific behavioural phenotypes. For example, Down syndrome confers protection from mania, and problem behaviours, whilst increasing risk for dementia, Prader Willi syndrome is associated with affective psychosis, and velo-cardio-facial syndrome increases risk for psychosis. Behavioural phenotypes are considered in greater depth in Chapter 10.4. In this section we consider mental ill-health of adults with intellectual disabilities of all causes.

Study methodologies

Mental ill-health is thought to be commonly experienced by adults with intellectual disabilities. Many of the existing prevalence studies have methodological limitations, accounting for the wide discrepancy in reported prevalence which ranges from 7 to 97 per cent. Limitations have included biased sampling; reliance upon existing case-note information, or instruments designed as screening tools only; lack of information on the extent of detail within assessments, the instruments, or diagnostic criteria used; and population-based studies limited by small cohort sizes. Other limitations include failure to indicate whether rates are lifetime, point, or period prevalence; reporting combined prevalence for children and adults; reporting mental ill-health in total, but not describing nor being comprehensive as to what is, and what is not, included (particularly with regards to problem behaviours, autistic spectrum disorders, attention-deficit hyperactivity disorder, and anxiety disorders); and studying selected subgroups such as only adults with verbal communication skills. All of these points must be carefully considered when interpreting and drawing conclusions from the existing literature.

Diagnostic criteria

Prevalence of mental ill-health varies, depending upon the diagnostic criteria employed. This is because many of the diagnostic categories within *The ICD-10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research* (DCR), and the DSM-IV-TR contain criteria that cannot be met due to the person's degree of intellectual disabilities and communication skills, and do not include other criteria that are important in this population. For these reasons, *Diagnostic Criteria for Psychiatric Disorders for Use with Adults with Learning Disabilities/Mental Retardation* (DC-LD) was developed for use specifically with this population. These, and

other important diagnostic issues are explored in further depth elsewhere.[4]

Prevalence

Population-based studies where participants received a psychiatric assessment are shown in the Table 10.2.1. [5–9] A high prevalence of mental ill-health was reported in all but Lund's study, which used assessment methods which would today be considered limited.[6] Point prevalence is higher than that observed in the UK general population. Specific types of mental ill-health with a higher prevalence compared with the general population including problem behaviours, autism, dementia, bipolar disorder, and psychoses. Dementia is part of the behavioural phenotype of Down syndrome, and also occurs three to four times more commonly amongst people with intellectual disabilities of other causes.[10,11] Bipolar disorder occurs at about double the prevalence of that reported for the general population. This is despite a high proportion of people (about 25 per cent) taking mood-stabilizing drugs (typically for epilepsy management). Depression is either more prevalent, or occurs at the same rate, depending upon the criteria used. Prevalence of non-affective psychotic disorders (including schizoaffective disorders) has consistently been reported to be higher than for the general population; a recent study found a point prevalence of 4.4 per cent including schizophrenia, in remission, or 4.0 per cent for psychosis, currently in episode. Problem behaviour is the most prevalent type of mental ill-health at 22.5 per cent (Consultant psychiatrist's opinion) or 18.7 per cent (DC-LD) in the most recent of the studies.[9]

Given the numerous biological, psychological, social, and developmental disadvantages experienced by adults with intellectual disabilities compared with the general population, a higher prevalence of mental ill-health is expected. The higher prevalence of bipolar disorder and of psychosis points to biological origins yet to be determined, and likely to be of relevance to the whole general population.

Incidence

There has been limited study of the incidence of mental ill-health in adults with intellectual disabilities. Longitudinal studies in the general population have not included persons with moderate to profound intellectual disabilities, but have demonstrated the higher prevalence of symptoms of depression and anxiety in adults with mild intellectual disabilities compared with the general population.[12,13] A recent study of a population-based cohort of 651 adults with intellectual disabilities found that incidence varied, depending upon the criteria used.[14] According to the Consultant psychiatrists opinion, the 2-year incidence of mental ill-health (of all types, except specific phobias) was 16.3 per cent (12.6 per cent excluding problem behaviours, and 4.6 per cent for problem behaviours), giving a standardized incident ratio for common mental disorders of 1.87 (95 per cent CI = 1.51–2.28). The comparison general population data was broadly similar, but not identical in its classification. The episodes with most frequent incidence were affective disorders at 8.3 per cent, followed by problem behaviours. The incidence of episodes of psychosis over the 2-year period was 1.4 per cent, of which 0.5 per cent were first episodes of psychotic disorders, giving a standardized incidence ratio of 10.0 (95 per cent CI = 2.1–29.3). The rate of full remission from an episode of psychosis within the 2-year period was low, at 14.3 per cent. The authors concluded that the high point prevalence of mental ill-health is explained by both a high incidence and high level of enduring mental ill-health, with slightly more enduring than incident cases.

Protective and vulnerability factors

Many of the factors that might afford protection from or increase vulnerability for mental ill-health are interrelated, for example level of ability, age, gender, and epilepsy. Few studies have attempted to tease these apart. Further work in this area is important, particularly as the pattern of related factors appears to differ from those found in the general population, suggesting that inferences cannot necessarily be drawn from general population data.

Ability

The relationship between ability level and mental ill-health has variously been reported to be absent, present with higher prevalence of mental ill-health at lower ability levels, or present with higher prevalence of mental ill-health at higher ability levels; these differences are explained by the limitations in the previous literature, as described above. Recent reports suggest lower ability is associated with mental ill-health in general, and specifically with

Table 10.2.1 Prevalence of mental ill-health among adults with intellectual disabilities

Study	Sample size	Ability	Diagnostic criteria	Prevalence (%)*
Corbett (1979)[5]	402	Borderline-profound intellectual disabilities	ICD-8	46.3†
Lund (1985)[6]	302	Borderline-profound intellectual disabilities	Modified DSM-III	28.1
Cooper and Bailey (2001)[7]	207	Mild-profound intellectual disabilities	Modified DCR	37.0
Deb *et al.* (2001)[8]	101	Mild-moderate intellectual disabilities	ICD-10	14.4‡
Cooper *et al.* (2007)[9]	1023	Mild-profound intellectual disabilities	Psychiatrists opinion DC-LD DCR DSM-IV-TR	40.9 35.2 16.6 15.7

*Excluding specific phobias.

†Excluding dementia.

‡Excluding problem behaviours, personality disorder, dementia, autism, alcohol problems, schizophrenia not in episode, and bipolar disorder not in episode.

problem behaviours, but not depression nor psychosis; and that lower ability also predicts incident problem behaviours.[6,7,9,14] However, diagnostic complexities may contribute to the lack of association between lower ability and some disorders.

Age

The prevalence and incidence of dementia is higher with increasing age. Whilst it has been suggested that problem behaviours are more prevalent at younger ages, there is no consistent evidence to support this. Of the population-based studies quoted above, no relationship was found between age and mental ill-health.

Gender

Autism and attention-deficit hyperactivity disorder are more common in males. Most studies have not otherwise found any association between gender and mental ill-health in this population, unlike the general population. A higher prevalence of problem behaviours has been reported in women,[8,9] but aggression has also been reported as more common in men[15]; these differences might possibly be due to whether the independent effects of gender and autism are investigated. Women have been reported to score higher than men on the 'affective/neurotic disorders' subdomain of a screening tool,[16] and to have a higher prevalence of mental ill-health,[9] and specifically depression; however, within the same cohort gender did not predict incident mental ill-health.[14]

Epilepsy and other physical ill-health

There are conflicting findings regarding whether there is a relationship between epilepsy and mental ill-health in this population, with the interaction between level of ability and epilepsy and use of antiepileptic drugs possibly contributing. It remains unclear whether physical and mental ill-health are related.

Life events

Associations have been demonstrated between preceding life events in adults with intellectual disabilities and scores for 'affective/neurotic disorders', and between life events and scores on the *Developmental Behaviour Checklist for Adults*.[17] A relationship has been reported with mental ill-health in general, and specifically with depression, but not psychosis, nor problem behaviours, nor incident mental ill-health (excluding problem behaviours),[9,14] but has been reported for incident problem behaviours.[14]

Accommodation/support

Both prevalence and incidence of mental ill-health, and of problem behaviours, is related to living in a setting other than with family carers, and this is independent of past psychiatric history. This highlights the need for engagement between professionals, service managers, and paid carers.

Deprived localities

Unlike for children, no relationship had been found between area-based measures of deprivation and mental ill-health in the adult population with intellectual disabilities, although there have been few investigations of this. It is possible that adults with intellectual disabilities may not have the same lifestyle characteristics as the general population living in the same area, due to being 'placed' in areas dissimilar from those they originated from and within which they acquired life-long habits and preferences, and through ongoing important relationships with family members whose own views and actions may be of greater influence than those of their paid carers or local community. It may be that the biological, social, and developmental causes and consequences of intellectual disabilities far outweigh some of the factors of relevance to the general population, in the aetiology of mental ill-health. This requires further study.

Smoking

As for the general population, smoking is associated with mental ill-health, and specifically with depression and psychosis.

Other factors

Other factors which may be independently related to mental ill-health in this population include urinary incontinence (as in the general population), not being immobile, visual impairment (for psychosis, as for the general population), abuse, neglect and exploitation, and parental divorce during childhood. However, few studies have investigated these factors. Few people with intellectual disabilities use alcohol or cannabis, so it is not known if there is any relationship between these behaviours and mental ill-health in this population.

Conclusions

- The prevalence of intellectual disabilities varies, depending upon definition, country, time, age range, and methods of population ascertainment. Reported rates vary substantially, and may be in the order of 9–14/1000 childhood populations, 3–8/1000 adult populations in developed countries, and higher in developing countries.
- Mental ill-health is more commonly experienced by adults with intellectual disabilities than the general population. Point prevalence is about 40 per cent, with problem behaviours being the most prevalent type.
- Dementia, problem behaviours, autism, bipolar disorder, and psychoses are more prevalent than for the general population.
- Incident mental ill-health is also greater than for the general population, at about 8 per cent per year. Common mental disorders and psychoses both have higher incidence than that for the general population.
- There is limited information on the protective and vulnerability factors for mental ill-health.
- Some factors related to prevalence and incidence of mental ill-health are similar to those found in the general population suggesting similar underlying causative mechanisms, but other factors differ, suggesting that inferences cannot necessarily be drawn from general population data and applied to the population with intellectual disabilities.
- Identifying high-risk groups within the population may allow for the provision of early interventions and supports, whilst some causative factors may be amenable to interventions to prevent or improve mental ill-health in this population. We need to gain a better understanding of these issues.

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10.3

Aetiology of intellectual disability: general issues and prevention

Markus Kaski

Causation

The complexity of causes

Intellectual disability can follow any of the biological, environmental, and psychological events that are capable of producing a decline of cognitive functions. Some factors do not directly or inevitably cause intellectual disability but add to the effects of a previous primary cause. Genetic causes may be hereditary or non-hereditary, and may or may not produce specific syndromes. Some lead to inborn errors of metabolism.[1]

Neurological symptoms during the neonatal period are strongly associated with prenatal developmental disturbances. For example in maternal pre-eclampsia, placental insufficiency may lead to malnutrition, foetal asphyxia, intrauterine growth retardation and prematurity, and subsequently to perinatal problems including asphyxia, intracranial haemorrhage, hyperbilirubinaemia, and hypoglycaemia. It is important to detect these coexisting conditions, because their effects may add to or interact with those of the primary cause.[2]

The biomedical cause of intellectual disability may lead to *additional disorders or disabilities*, or may itself be progressive.[1–3] In fact, intellectual disability exists commonly with many other symptoms in a patient, for example with sensory problems, dysphasia, cerebral palsy, epilepsy, or autism. These additional factors affect *opportunities for gaining experiences necessary for development*. Activity may be restricted by sickness, or the effects of medication. Motor disability may reduce mobility, or cause dysphasia. Sensory impairment may restrict vision or hearing. These restrictions add to the effects of the primary cause and interact with environmental and emotional factors to retard the development of the individual.[1–4]

How often do we know the cause(s)?

The various concepts of intellectual disability and its causes have led to *different epidemiological estimates*. The population at risk consists of survivors, but some figures include also all live born. Differences in the definition and detection of cases, the classification system used, the population studied, the timing of the study in relation to measures of the general population, the resources available for the study, and sources of the data make it difficult to compare both the frequency of persons with intellectual disability and the frequency of various causes obtained in different studies. However, a specific cause for intellectual disability can be identified for approximately 80 per cent for persons with severe intellectual disability (IQ <50, includes the groups of moderate, severe, and profound intellectual disability in ICD-10) and 50 per cent of persons with mild intellectual disability. The principal cause of intellectual disability is estimated to be prenatal in 50 to 70 per cent, perinatal in 10 to 20 per cent, and postnatal in 5 to 10 per cent of persons.[3–7] In general, knowledge of aetiologies is more accurate for people with severe intellectual disability than for those with mild intellectual disability.[8, 9]

Classifying causes

The causes of intellectual disability may be classified according to the particular clinical entity, the causative agent or presumed cause, or the *timing of the causative factor*. The newer and more successful classification systems are based on timing.[10] The principle is biomedical in nature and it is intended to elucidate the *earliest factor* that has affected the development of the central nervous system. The discovery of the primary cause (or sequential causes) aids family counselling and may lead to identification of a preventable general risk.

Viewed in this way, diagnoses can be divided into six main groups according to the probable cause and the timing of damage to the central nervous system (Table 10.3.1) [11]:

- genetic causes;
- central nervous system malformations of unknown origin;
- external prenatal factors;
- disorders acquired in the paranatal* period;
- disorders acquired postnatally; and
- untraceable or unclassified causes.

*The paranatal period is defined as the period from 1 week before birth to 4 weeks after birth.

Table 10.3.1 Aetiology based on the time and mechanism of the injury to the central nervous system and the history helping identification, timing, and diagnosis of intellectual disability

Aetiology	History[a]
Genetic causes Chromosomal disorders Malformations due to microdeletion Single-gene disorders Multifactorial intellectual disability Mitochondrial disorders	*Family history* Family tree, intellectual disability, learning disabilities, neurological diseases, congenital anomalies, psychoses, consanguinity of parents, recurrent abortions, previous stillbirths, low parity or infertility, and parental ages
CNS maformations of unknown origin Isolated malformation or malformation sequence of the CNS Multiple malformations	*Family and gestational history*
External prenatal factors Infections Physical, chemical, and toxic agents Maternal and gestational disorders Other	*Gestational history* Maternal infections of the TORCH group, HIV, radiation, trauma, chronic maternal diseases, drugs, pre-eclampsia, severe malnutrition, alcohol, bleeding, and abnormal intrauterine growth or fetal movements
Paranatally acquired disorders Infections Delivery problems Other newborn complications	*Birth and neonatal history* Gestational age, multiple pregnancy, birth order, placental abnormalities, labour or delivery complications/ mode and duration, asphyxia, intracranial haemorrhage, trauma, 5-min Apgar scores, newborn weight, length and head circumference, infections, hypoglycaemia, hyperbilirubinaemia, neurological problems, and weight gain
Postnatally acquired disorders Infections Other damage to CNS Psychosocial problems Psychoses without family history	*Childhood history* Feeding and sleeping patterns, nutrition, growth charts, developmental milestones, infections of CNS, head injuries, submersions, metabolic and endocrine disorders, vascular accidents or thromboses of cerebral veins, cerebral tumours. toxic agents, and psychosocial environment
Untraceable or unclassified causes Pure non-familial With CNS symptoms Not classified	*History of associating conditions* No history of adverse events or signs or history with CNS symptoms such as cerebral palsy, epilepsy, or autism in addition to mental retardation

CNS, central nervous system; TORCH, toxoplasmosis–other infection–rubella–cytomegalovirus–herpes.

[a]Gives the time of the risk event or cause, or appearing time of the sign whose cause(s) may also be earlier.

(a) Genetic causes

Chromosomal disorders include all intellectual disability caused by a proven chromosomal aberration or a clinically obvious chromosomal syndrome such as Down syndrome. However, chromosome analysis should be performed in Down syndrome because translocation, mosaicism, or other abnormalities are found in 5 per cent of cases. Chromosomal anomalies associated with intellectual disability account for up to 40 per cent of severe cases, and 10 to 20 per cent of mild cases.[3,6,12,13] However, the detection rates for chromosome abnormalities with the novel molecular karyotyping methods, such as micro**array**-based **c**omparative **g**enomic **h**ybridization (**array CGH**), range from 5 to 17 per cent in individuals with normal results from prior routine cytogenetic testing.[14] Array CGH has the ability to detect any genomic imbalance including deletions, duplications, aneuploidies, and amplifications.

Malformations due to microdeletion include many malformation syndromes whose causative agent is obscure. A new method of using DNA probes and fluorescence in situ hybridization has increased understanding of the causes of syndromes such as the Angelman, Cornelia de Lange, CATCH 22 (**c**ardiac defects, **a**bnormal face, **t**hymic hypoplasia, **c**left palate, and **h**ypocalcaemia) (velocardiofacial syndrome), Miller–Dieker, Prader–Willi, Rubinstein–Taybi, Smith–Magenis, Sotos, Williams, and Wolf–Hirschhorn syndromes. Parental imprinting modifies the expression of the genes involved in the Prader–Willi and Angelman syndromes.[12, 14–16]

Subtelomeric deletions or **chromosomal rearrangements** have been found in some persons with intellectual disability of hitherto unknown aetiology. Subtelomeric aberrations may explain up to 5 to 10 per cent of previously unknown causes.[13,15]

Single-gene disorders include states with intellectual disability in which the pedigree is highly suggestive of a single-gene origin. Some are caused by a mutant gene with simple Mendelian inheritance. Single-gene mutations may increase or diminish in frequency in areas with long-standing populations of the same origin, or in populations isolated by language or culture. For example, the so-called Finnish *disease heritage* includes 36 disorders, from which 10 manifest with central nervous symptoms and some others may have them.[17] Most of the specific disorders due to mutant gene have characteristic clinical phenotypic features, but there are a considerable number of non-syndromic individuals, especially in early infancy.[12,18,19]

Autosomal dominant inheritance causes tuberous sclerosis, myotonic dystrophy, Gorlin syndrome, neurofibromatosis I, Apert syndrome, Menkes syndrome, and Huntington's disease.

Autosomal recessive inheritance is the cause of most metabolic diseases with intellectual disability. These diseases include phenylketonuria, homocystinuria, maple syrup urine disease, aspartylglucosaminuria, mannosidosis, Salla disease, I-cell disease, mucopolysaccharidoses (except type II), neuronal ceroid lipofuscinoses, Tay–Sachs disease, metachromatic leucodystrophy, Smith-Lemli–Opitz syndrome, and Joubert syndrome.

X-linked inherited disorders include the fragile X, Aicardi, Lesch–Nyhan, Lowe, Norrie, and Coffin–Lowry syndromes, mucopolysaccharidosis II, Duchenne muscular dystrophy, α-thalassaemia intellectual disability syndrome, and Rett syndrome. The most

common intellectual disability syndrome caused by mutation of a single gene is fragile X syndrome. The pattern of its inheritance is X-linked dominant with decreased penetrance.[17,19,20] The prevalence of the 24 other genes identified to date in the X chromosome is low.[20] Dystrophic myotony, fragile X syndrome, and Huntington's disease are caused by so-called *dynamic mutation* in which the length of the repeated sequence of three DNA bases can vary from generation to generation increasing the variability in the phenotype.[21] In Rett syndrome female inactivation of X chromosome may be skewed. It explains the existing of the syndrome in a male or the very mild phenotype in a female.[22] Epigenetic regulatory factors are also involved in the aetiology of Rett syndrome.[23]

Mitochondrial disorders are inherited in most cases due to mutations in the *nuclear genes* encoding proteins targeted to this organelle. Autosomal dominant, recessive, or X-linked inheritances are possible. In addition, mitochondrial dysfunction is shown among others in patients with fragile-X, Rett, and Wolf–Hirschhorn syndromes or autism. [24–27] *Mitochondrial DNA* (mtDNA) is inherited maternally. Sporadic deletions and duplications are also found (Kearns–Sayre syndrome, sporadic deletion or partial duplication in mtDNA). Examples of the maternally inherited (mtDNA) syndromes with central nervous symptoms are the MELAS (mitochondrial myopathy, encephalomyopathy, lactic acidosis, and stroke-like episodes), MERRF (myoclonus epilepsy with ragged red fibres), and NARP (neurogenic muscle weakness, ataxia, retinitis pigmentosa), FSFD (facio-scapulo-femoral muscular dystrophy, familial cerebellar ataxia, recurrent Reye syndrome, cerebral palsy with intellectual disability), and cytochrome c oxidase (COX) deficiency (deafness, myoclonic epilepsy, ataxia, and intellectual disability) syndromes. Nuclear genes are often involved in mitochondrial DNA depletion and Leigh syndromes, which are severe progressive diseases in early childhood.[11, 18, 21]

Multifactorial intellectual disability may be a state of *pure familial* intellectual disability or associated to some *multifactorially inherited* conditions, for example neural-tube defects. One or more first-degree relatives are also affected. Similar pervasive developmental disorders or childhood or other psychoses in one or more of first-degree relatives or otherwise strong family background suggest a polygenic component of intellectual disability.[12, 21]

(b) Central nervous system malformations of unknown origin

Approximately 30 to 40 per cent of all malformations are *genetic* and 10 per cent have *exogenous* causes. The aetiology of the rest is unknown. The number included in this aetiological group decreases with more accurate diagnosis of genetic malformations, intrauterine infections, other teratogenic agents, or deficiencies of essential ingredients needed for the normal development.[28,29] The development of the central nervous system may be disturbed at the following stages[2,12,21,28,29]:

1 dorsal induction at the 3rd to 7th weeks of gestation, leading to anencephaly, encephalocele, meningomyelocele, or other neural-tube-closure defects;

2 ventral induction at the 5th to 6th weeks of gestation, causing prosencephalies and other faciotelencephalic malformations;

3 proliferation of the neurones at the 2nd to 4th month of gestation, leading to microcephaly or macrocephaly;

4 migration of the neurones at the 3rd to 5th month of gestation, causing gyrus anomalies and heterotopias;

5 organization of neurones from the 6th month of gestation to a year postpartum, leading to disturbances in the formation of dendrites and synapses;

6 myelination from 6th month of gestation to a year postpartum, disturbing the proliferation of oligodendrocytes and the formation of the myelin sheets.

The **malformation sequence** is a type of multiple malformation, which includes secondary anomalies caused by an earlier anomaly, for example equinovarus with meningomyelocele. **Multiple malformation syndromes** are caused by the disturbances in blastogenesis or organogenesis. Multiple malformation syndromes of unknown origin include some whose causes are unknown such as the Goldenhar and Kabuki syndromes, and research will show that some of these aetiologically unknown syndromes have a genetic cause.[30,31]

(c) External prenatal factors

The nature of the impairments or malformations, and the severity of resulting intellectual disability appear to relate, at least partially, to the timing of the causative factor as discussed earlier. Also dosage may be important. Effects are most serious when the cause acts *early in embryonic development*; during blastogenesis or organogenesis, when it may result in *multiple malformations*. Effects on the central nervous system of *causes acting later* may be severe even though outward signs may be lacking. These causes include congenital infections such as rubella, cytomegalovirus, herpes simplex type 2, parvovirus, and HIV infection, as well as toxoplasmosis and syphilis. Exposure to medication and other substances such as hydantoin, lipid solvents, alcohol, cocaine, and other drugs can affect the developing foetus.[2,3,28,32,33]

Maternal disorders that may contribute to the causes of intellectual disability include maternal diabetes, arterial hypertension, placental insufficiency, pre-eclampsia, pre- and postmaturity, multiple pregnancy, and foetal growth retardation. In other cases no specific causes can be identified with certainty but available data strongly suggest a prenatal external cause of central nervous system impairment such as exposure to ionizing radiation or trauma.[2, 34]

(d) Disorders acquired in the paranatal period

The effects of the last week of pregnancy extend to the neonatal period and are very important for the outcome of the newborn,[11,35] and combinations of the prenatal and postnatal factors are not rare. *Infections* are transmitted via placenta or the birth canal. They include neonatal septicaemia, pneumonia, meningitis, and encephalitis, which may lead by several mechanisms to neurological deficits, intellectual disability, and sometimes microcephaly, or in bacterial meningitides also to hydrocephalus. *Congenital infections* of herpes simplex and HIV as well as tertiary syphilis may manifest later. *Problems during delivery* may lead to asphyxia, intracranial haemorrhage, or other birth injuries and cause various symptoms of cerebral palsy and epilepsy. *Other newborn complications* include hypoglycaemia, hyperbilirubinaemia, and respiratory distress. Paranatal aetiologies may cause disorders of cognitive functions, as well as motor and sensory impairments.[2,35,36]

(e) Disorders acquired postnatally

Improved postnatal care has reduced the frequency of these causes, which include *infections* such as meningitides and encephalitides.[37] Other causes of postnatal damage to the central nervous system include *toxic agents*, *vascular accidents*, *brain tumours*, *hypoxia*, *and traumas*. Traffic accidents, other traumas, submersions, and cerebral tumours are common causes of disability in childhood. Lead poisoning has been a problem in the United States, iodine deficiency in some regions of the world, and malnutrition almost worldwide.[38] *Psychosocial problems* causing intellectual disability are not as common as was thought in the past, partly because of better identification of medical factors.[39] Severe maternal mental or chronic physical illness, parental alcohol or drug abuse, and some consequences of poverty may be contributory causes leading to inadequate care and stimulation. *Deprived environments* are linked to other risks such as malnutrition, poor medical care, child abuse, usage of alcohol and other substances, and teenage pregnancies.[40]

(f) Untraceable or unclassified causes

The aetiology of intellectual disability can be classified as unknown if the causative factor or timing of the brain damage cannot be established. ***Pure non-familial*** intellectual disability is the term used when there is no family history of intellectual disability and no signs and symptoms suggesting brain damage. It represents the extreme of normal variation. In the untraceable group, the second category is ***intellectual disability of unknown aetiology*** with other symptoms and signs of the central nervous system suggesting brain damage, but with no family history of intellectual disability and no identified malformations or dysmorphic features (see Table 10.3.1). Common examples are intellectual disability associated with cerebral palsy, epilepsy, or autism. Patients should not be assigned to the untraceable group if the diagnostic work-up is incomplete.[11] If so, the aetiology is still ***unclassified***.

How to assess causes

A comprehensive history and a *careful physical examination* are essential for identifying and timing the causative factor(s). In everyday practice it is appropriate to assess the family history, embryological, and postnatal development, possible pathogenetic mechanisms, and the time of the exposure to the supposed agent (Table 10.3.1). The finding of *more than three minor malformations* suggests genetic or early developmental disorder provided that the same dysmorphic features are not found in close relatives.[41] Infants at risk for external prenatal causes, perinatal causes, or postnatally acquired disorders should be examined carefully for dysmorphic features which may indicate alternative or additional cause. The diagnosis usually becomes evident by working out the history and clinical signs. *Databases for analyses of dysmorphology*, symptoms, or other findings are useful aids in the search of an aetiological diagnosis.[42,43]

Ophthalmological and audiological *examinations* can be arranged and other necessary investigations carried out when suggested by the history and physical examination (Table 10.3.2).[2,4,11,12,18,21,37,38,41–43] *If the findings are in accordance with history* and a person has no congenital anomalies, further examinations are seldom needed. However, the possibility of a metabolic disorder should be kept in mind, especially as congenital anomalies or dysmorphic features may occur in people with metabolic disorders. *Metabolic studies* should be performed for every patient with *progressive symptoms*. *If the history and physical findings do not match*, or if there are congenital anomalies or more than three minor dysmorphic features, additional studies are needed.[41] Because more accurate diagnostic methods are being developed, it is useful to keep for each person *a dated chart of examinations* performed.[11]

Prenatal diagnosis may be indicated when there is a known parental balanced translocation, chromosomal aberration of a sibling, a known hereditable disorder in family, a multifactorial disorder such as neural-tube defect in the family, or the mother is elderly.

The gravidity can be detected by *ultrasound examination* at 6th to 8th gestation weeks. Many structural changes can be found from 11th to 15th weeks and confirmed by repeated examinations to 22nd gestation week, for example neural-tube defects. The *nuchal translucency in relation to crown-rump length and gestation week* can be measured during the 9th and 14th for detecting Down syndrome. Other general screening methods for Down syndrome are based on the *applicable markers from the serum of the mother* to the 10th to 14th and 15th to 18th gestation weeks.[44,45] According to the positive screening result or abnormal morphology finding in the ultrasound examination foetal karyotype or some other further examination may be indicated. The age-specific screening of foetal chromosomes is based on the significantly increased probability of a Down syndrome child among mothers aged 35 years or older.[12]

The karyotype of a foetus can be identified after the 10th week of gestation from a *chorionic villus sample*, or after 15th week gestation from the *cells of amniotic fluid*. A known single-gene disorder can also be searched from the chorionic villus sample by *DNA*,

Table 10.3.2 Diagnostic examinations of intellectual disability

- Neurological, ophthalmological, audiological, cardiological, neuropsychological, etc., assessments
- Blood count, vacuolated lymphocytes, and thyroid function
- Antibodies, serology, and urine (TORCH, HIV)
- Radiographs of skull, vertebral column, chest, hands, feet, and long bones, and bone age
- Chromosomes: G-banding, high-resolution banding, and FISH array CGH
- FraX DNA, specific DNA tests, and other molecular genetic techniques
- Blood/urine: amino and organic acids, muchopolysaccharides, oligosaccharides, long- and very-long-chain fatty acids, Astrup, glucose, ammonia, lactate, pyruvate, uric acid, phytanic acid, carnitine, lead, copper, ceruloplasmin
- Fibroblast culture or white blood cell sample; specific enzymes
- Biopsies: muscle, skin, rectal
- Neurophysiological: EEG and evoked potentials
- Neuroimaging: cranial ultrasound, CT, MRI, MRS, functional MRI, SPECT, and PET
- Neuropathological examinations

TORCH, toxoplasmosis–other infection–rubella–cytomegalovirus–herpes; FISH, fluorescence in situ hybridization; MRI, magnetic resonance imaging; SPECT, single-photon emission CT; PET, positron emission tomography; MRS, magnetic resonance spectroscopy.

enzyme, or other specific methods. Cell cultures of few amniotic fluid cells can be used for karyotyping or diagnosing metabolic diseases, but it needs more time. Neural-tube defects, also the small ones, can be seen as elevated levels *of α-fetoprotein* in amniotic fluid. Sometimes a blood sample from the umbilical cord is needed after the 18th gestation week for confirmation of karyotype.

Prenatal diagnostic methods and identification of parental balanced translocations or aberrations in single gene are increasingly available. Microarray-based comparative genomic hybridization (array CGH), examination of foetal DNA or cells derived from maternal blood circulation, and preimplantation diagnoses becoming available for some diseases will change the prenatal diagnosing practice considerably.[12,21,46,47]

Why knowledge of causation is important

Intellectual disability is a confusing concept. The people with intellectual disabilities have more differences than common features. Developmental delay may appear in different ages and with different degrees of severity in different children. The development of a child can come to a stop or can even regress. There is a multitude of confirmed causes of intellectual disability. Single aetiologies are rare and the clinical picture within the same aetiology and between different aetiologies can vary greatly. It is now possible to detect causes that until recently were unknown. Associated disabilities and chronic diseases are common and modify further the complex interplay of individual and environmental factors.

The factors believed to be related to the incidence and prevalence of intellectual disability, such as personal history and gender, the age and the marital status of the parents, the number of siblings, and the living conditions and the social situation of the family, as well as the neighbouring community, vary in persons with intellectual disability. *Attitudes to disabilities* may differ in different families and societies. The permanence of the cognitive impairment is difficult to accept. Insufficient or inadequate information or a prolonged diagnostic process may lead the family or the child to become fearful about the cause of the condition or to try and identify some reason for it. The way is then open for misunderstandings, feelings of guilt, or projections.[4,11,48]

For the person with intellectual disability a confirmed aetiology is the basis of a correct awareness of his or her own disability; the limitations set by the disability and the possibilities for learning and development. The clinical manifestations of some developmental disorders, such as phenylketonuria, galactosaemia, or hypothyroidism can be prevented or arrested by dietary management or hormonal replacement therapy. Knowledge of the prognosis increases awareness of associated disease and disabilities such as sensory impairments, communication disorders, motor and joint problems, epilepsy, and behavioural or psychiatric problems. Thus, aetiology aids the planning of follow-up, rehabilitation, education, and living arrangements.[4,11] Knowledge of aetiology is particularly important at the time of transition from childhood to adult services, helping to ensure continuity of provision and to avoid drop-out.

For the family, knowledge of causes helps to dispel wrong beliefs, self-blame, and anxieties. The parents and siblings may change their preconceived ideas about the disability. It helps the parents to adopt appropriate standards for bringing up their child, and for life as an adult. It helps them to become aware of the child's special needs.[4,11] Aetiologic diagnosis is the necessary basis of reliable genetic counselling and helps the parents and siblings in family planning.[12,48]

In society the knowledge of the aetiologies of intellectual disability increases the likelihood that its people will adopt positive attitudes towards the disabled. Both the society and its service providers need understanding of the causes of intellectual disability, their prevalence, and their prognoses when planning primary prevention, organizing services and education, optimizing environmental factors, or preparing relevant legislation. Society needs experts continuously alert to advances in scientific research to keep this knowledge up to date.

When the causes are unknown, prognosis is uncertain and the planning and provision of the services is difficult. The risks of discontinuities in service provision and of drop-outs increase. Because families have limited information they are more likely to develop wrong beliefs, self-blame, and projections. They have unrealistic expectations about alternative therapies.[12, 48]

Prevention

Primary prevention

The identification of factors that contribute to intellectual disability, their removal or avoidance, and the protection of the population or individuals against them are the main principles of primary prevention. Immunization and other measures to prevent rhesus incompatibility, congenital rubella, measles encephalitis, tuberculosis and other bacterial meningitides, prionic diseases, and the provision of folic acid around the time of conception to prevent neural-tube defects have been successful preventive measures.[2,37]

Primary prevention includes *good medical follow-up, the identification and prompt removal of, or effective intervention in*, at-risk situations during pregnancy, delivery, the neonatal period, and childhood. Avoidable causes include intrauterine and perinatal infections due to many sexually transmitted diseases, and fetal alcohol spectrum disorders (FASD).[49] Lead intoxication, iodine deficiency, accidents in the home, and traffic accidents are preventable.[2,36,38] The primary prevention of genetic disorders has not been possible, although the disorder, the chromosomal aberration, or some associated abnormality can sometimes be identified.[21,29,44] However, all the means of the secondary prevention are available, and pregnancies may be terminated after counselling. Screening tests can identify some parents who are carriers.[48] The preimplantation diagnosis could give a new way for the early prevention of known genetic disorders in some cases.[47,50]

Secondary prevention

Early recognition and diagnosis, good medical care, and rehabilitation of injuries or diseases can avoid or reduce permanent damage which could lead to intellectual disability. Examples include the screening for and early treatment of congenital hypothyroidism and phenylketonuria.[2] Secondary prevention also includes planning or *genetic counselling* after the birth of a child with a genetic disorder.[48] The examination of asymptomatic parents and close relatives in order to detect carriers is part of genetic counselling. As yet there are no measures to prevent the underlying biological processes in genetic disorders.[12,18,21]

Once children with intellectual disability have been identified, accurate assessment of aetiology and associated conditions, therapy,

and rehabilitation lessen the risk of so-called **caused learning disability**, that is leaving the person with intellectual disability without the possibility of developing further because he or she is unable to share in the learning experiences of the peer group.[2,4,38]

Tertiary prevention

The aim of tertiary prevention is *to help an individual attain his or her full developmental potential.* Therefore tertiary prevention partly overlaps with secondary prevention. It encompasses all measures, which prevent or lessen persistent hindrances to the development of functional ability or social competence in people with identified damage or disease. It includes medical, psychological, social, and family support, environmental adjustments, aids, and education. In the ideal state, individual supports are so good that the mentally retarded can live a life similar to that of people without intellectual disability. However, even when this can be achieved, problems may reappear when tasks of life increase, or the supporting systems fail.[4,10]

Ethical problems of prevention

Usually there are no ethical problems. There are hardly any issues surrounding the prevention of causative diseases or injury, the attempt to reduce the prevalence of inheritable diseases by family planning or genetic counselling, the screening that leads to the treatment of the foetus or the newborn, preparation for the birth of a new infant, improving the care and rehabilitation, and planning a safer society.

Ethical questions arise if there are no measures to prevent the effects of an identified genetic or other cause of intellectual disability identified during pregnancy. Disordered genes, other genetic rearrangements, or accidental mutations cannot be removed from a population. Primary prevention of an intellectual disability becomes the prevention of childbirth or selective abortion. The latter is usually on the grounds that the burden of caring for the child would be too great for the parents, an indication that is more social than medical.[51,52] In general, the prevention of a disease does not exclude good therapy for it. However, the increasing trend to accept selective means of prevention may be a result of cultural change and increasingly negative attitudes towards disability.[53–56]

Prenatal diagnosis, detection of carriers and screening for disorders, which cannot be prevented or treated, usually lead to decisions about more or less harmful consequences. Ethical questions include *choices between knowing and not knowing*, between not knowing and worry or anxiety in risk situations, and between the need to take difficult decisions and to avoid them. It is very difficult to predict accurately all the long-term effects of these decisions. Parents have the right to be able to make voluntary decisions, but they need *accurate information* to be able to bear the burden of responsibility.[51,56–58]

Prevention requires a coordinated programme

On the basis of the aetiologies defined according to the timing principle, it is possible to search for epidemiological, psychological, sociological, and other explanations to answer the question of why some causes are more common in certain populations or subgroups than in others. *The starting point of prevention is knowledge of causation* and identification of factors that subject individuals to these causes. With better understanding of the causes of intellectual disability, attitudes towards it change. With knowledge, individuals needing support can be identified earlier, their development and life made easier, and the burden of care on the family lightened.

Public policy, education, public health, obstetric services, neonatal intensive care, general practice services, etc. have an important role in reducing and avoiding risk factors. Education, planning of a safe environment and alleviation of poverty has a general preventive effect on predisposing factors. *The preventive aspects should be taken into account in all general and specific legislation, in operating procedures, and professional practice*. Because multiple agencies are involved preventive measures need to be coordinated at both local and national levels.

The day-to-day prevention of infections, accidents in the home and traffic, exposure to toxic substances, drowning accidents, malnutrition, or child abuse is both *a general and a multiprofessional task*. Parents and educators should transfer their wisdom and experience to the next generation so that they will make better choices and avoid risks.

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10.4

Syndromes causing intellectual disability

David M. Clarke and Shoumitro Deb

Introduction

Psychiatrists working with people who have intellectual disability (mental retardation) need expertise in the diagnosis and treatment of associated neuropsychiatric disorders. This entails knowledge of the causes of intellectual disability, and especially knowledge about those syndromal (often genetic) causes that are associated with neuropsychiatric manifestations. Such manifestations include vulnerability to behavioural and emotional disorders, epilepsy, and particular patterns of cognitive strength and weakness. This chapter provides an introduction to some such disorders and a discussion of the concept of behavioural phenotypes. For a detailed account of conditions causing intellectual disability texts such as Jones[1] should be consulted. The concept of behavioural phenotypes is discussed in detail in O'Brien.[2]

The genetic aetiologies of intellectual disability include chromosomal abnormalities (trisomy, deletion, translocation, etc), single-gene defects, and the effect of interactions between several genes. The last is thought to account for a substantial proportion of people with mild intellectual disability by setting a ceiling on possible cognitive attainment (life experiences, nutrition, education, and other factors then determining the extent to which potential is fulfilled or thwarted).

This chapter discusses the concepts of syndromes and behavioural phenotypes, then describes the clinical features of a number of syndromes that cause intellectual disability. Down syndrome, fragile-X syndrome, sex chromosome anomalies, and foetal alcohol syndrome are described in some detail. This is followed by a briefer alphabetical list of less common conditions.

Syndromes

A syndrome is a characteristic pattern of clinical features, including signs (that can be observed) and symptoms a patient may experience. They may be causes of intellectual disability (Down syndrome), associated with intellectual disability (syndromes of epilepsy, such as West syndrome), or coincidental (polycystic ovary syndrome). This chapter deals with some of the syndromes that increase vulnerability to intellectual disability. The vulnerability may be increased so much that all affected people have intellectual disability (Angelman syndrome) or increased to the extent that many, but not all, have intellectual disability (velo-cardio-facial syndrome).

There can be disadvantages to the labelling of people with disability, but the identification of a syndromal cause may have benefits for the affected person and for their families and carers. Benefits include an explanation of the cause of the person's disabilities or of unusual cognitive strengths and weaknesses, better understanding of risk of recurrence of the disorder among relatives, and the identification of complications or associated features.

Identification of a syndromal cause may give access to support organizations. A list of such organizations is given in the CaF (Contact a Family) Directory (www.cafamily.org.uk).

Behavioural phenotypes

Behavioural, social, linguistic, or cognitive aspects of a syndrome may be so striking and characteristic as to prompt diagnosis. Examples include the severe self-injury associated with Lesch–Nyhan syndrome and the combination of appetite abnormality, ritualistic behaviours, sleep abnormalities, skin-picking, repetitive speech, and vulnerability to psychiatric disorder associated with Prader–Willi syndrome. Such patterns of vulnerability to particular emotional or behavioural problems or peculiarities associated with biologically determined syndromes have been called behavioural phenotypes. Environmental factors may interact with a genetically determined vulnerability to a behaviour to determine whether or not it occurs in a given setting. Knowledge of the nature of this interaction may be important in order to determine effective treatment or management strategies. In Lesch–Nyhan disease, for example, all affected men self-injure, but whether a man with the syndrome injures himself at a particular time is influenced by environmental and internal psychological factors such as anxiety. A careful assessment of the causes and consequences of behavioural problems is essential before interventions are planned, particularly the use of psychotropic medication to influence behaviour.[3]

Specific conditions

Down syndrome

(a) Prevalence and genetics

J. Langdon Down originally described the syndrome in 1887. Trisomy 21 is associated with Down syndrome, and was first reported by Lejeune and colleagues in 1958. About 1 in 600 live

born children have Down syndrome. The rate increases with increasing maternal age, being about 1 in 2000 at maternal age 20 years and 1 in 100 at maternal age of 40 years.[4] There are three types of abnormalities affecting chromosome 21. In about 94 per cent of cases, Down syndrome is caused by primary non-disjunction leading to trisomy 21. The risk of recurrence of this abnormality is low if maternal age is also relatively low. In about 2 per cent of cases Down syndrome results from an unbalanced translocation (when material from one chromosome is separated and attached to another with some duplication). This often involves chromosomes 21 and 14. In some cases a parent also has a balanced translocation (with no overall disruption or duplication of genetic material), and this raises the risk of recurrence. Chromosome 21 to 21 translocations can occur. Mosaicism occurs when there are two or more cell lines within the body. In Down syndrome there may be one cell line with trisomy 21 and one without. In about 2 per cent of cases the syndrome results from mosaicism. Some cases may not be diagnosed. The proportion of affected and unaffected cell lines varies, as does the intellectual impairment.

(b) Physical characteristics

Muscular hypotonia at birth usually improves with development. Most adults are of short stature and have a characteristic facial appearance. The eyes seem to slope upwards and outwards, the nose has a wide bridge and the head has an unusual shape (brachycephaly). Limb abnormalities include a single transverse crease on the palm, a large cleft between the first and second toes, and relatively short upper arms. People with Down syndrome are prone to thyroid abnormalities. About 25 per cent develop hypothyroidism during childhood or adolescence. About half of affected people have a heart abnormality. Abnormalities of the gastro-intestinal tract occur in a significant minority. Life expectancy has improved markedly over the past 50 years. Survival into the eighth decade is unusual but not extraordinary. Changes in blood cells are relatively common. Older texts reported an association between Down syndrome and leukaemia, but recent research suggests that leukaemia is rare, affecting less than 1 per cent of people with Down syndrome.

(c) Behavioural and psychiatric aspects

Adults with Down syndrome are much more likely to develop dementia than the general population. On post-mortem examination, the brains of almost all adults with Down syndrome over the age of 35 show changes characteristic of dementia of Alzheimer type. Only about 38 per cent of those aged 50 to 59 have clinically apparent dementia, with a mean age at diagnosis around 51 years.[5,6]

The stereotype of people with Down syndrome as happy, placid individuals with a gift for mimicry is not borne out by recent behavioural research. Stubbornness and obsessional features seem to be over-represented, and many people with Down syndrome react adversely in situations involving changes to expected routines or conflict. Autism seems to occur more commonly than would be expected, but few methodologically sound studies have been carried out.[7]

Most adults with Down syndrome have moderate intellectual disability. Almost all children with Down syndrome have some degree of specific speech and language delay. About 25 per cent have features of attention-deficit disorder. Cognitive abilities tend to be greater among people whose Down syndrome is caused by mosaicism for trisomy 21.

Further information: www.downs-syndrome.org.uk

X-linked intellectual disability

The prevalence of X-linked intellectual disability is around 0.18 per cent.[8] The majority of affected men have non-syndromic X-linked intellectual disability (usually referred to as X-linked mental retardation or XLMR in international literature), with no associated dysmorphology. The most common syndrome resulting in XLMR is fragile-X syndrome (described below). Coffin–Lowry syndrome (CLS) is also described below. It is increasingly accepted that there is a spectrum of disorders associated with XLMR genes, ranging from defined syndromes such as CLS to XLMR with no dysmorphology. For example, the gene RSK2 is usually mutated in Coffin–Lowry syndrome but a missense mutation in exon 14 of RSK2 has been found in a family in which males have intellectual disability but no associated features of CLS.[9] An interesting article described a woman with mild intellectual disability, epilepsy, and some minor dysmorphology whose karyotype was reported as normal in 1993. Repeated testing was carried out after she was found to have a more severely affected brother with a duplication affecting his X chromosome showed 46,X dup (X)(p22.13p22.31). The authors concluded that genetic testing for individuals with intellectual disability should be considered even when there was a low index of suspicion for an X-linked disorder.[10] About 200 XLMR conditions and 45 cloned genes have now been described.[11] At least eight genes have so far been implicated in non-specific XLMR: *Rab-GDI*, *PAK3*, *AGTR2*, *TM4SF2*, FRAXE (*FMR2*), *ARHGEF6*/*αPIX*, and *FACL4*.[12] Readers are referred to specialized texts such as Jaquemont *et al.* (2005)[12] and web resources such as xlmr.interfree.it/home for further details.

Fragile-X syndrome

(a) Prevalence and genetics

The syndrome was first described in 1943. All ethnic groups are affected equally, with a frequency of about 0.3 per 1000 in men. More recent investigations with modern diagnostic techniques show lower figures than earlier studies.[13]

Fragile-X syndrome is an X-linked disorder with a very unusual pattern of inheritance. It is characterized by a bias to affected men but with some affected women and some unaffected men who have daughters who then have affected sons. When peripheral blood lymphocytes from affected individuals are grown in certain culture conditions, including a lack of folic acid, a fragile site becomes evident on the long (q) arm of the X chromosome at Xq27.3 (fragile site A). Fragile sites may not be seen in some unaffected men who transmit the abnormality to their carrier daughters. These men were historically termed 'normal transmitting males'. The probability that a child with a fragile-X chromosome will have intellectual disability depends on the sex of the parent from whom the chromosome was inherited (higher risk when the chromosome is passed from the mother). The 'fragility' of the X chromosome is now known to be associated with an unstable region of DNA within the fragile-X mental retardation (FMR-1) gene, which was first described in 1991.[14] This region of unstable DNA gradually increases in length and degree of instability in successive generations (a pre-mutation) until a critical point is reached and the gene no longer functions (a full mutation). The instability is caused by an increase in CGG (cytosine-guanine-guanine) repeats from the 50 or so repeats that are usual to 50–100 repeats (pre-mutation)

to over 230 repeats (full mutation). The chance of a child inheriting a lengthened gene is proportional to the length of the unstable region in the carrier mother. The severity of intellectual disability and other fragile-X related phenomena in women probably depends mostly on the proportion of cells in which the abnormal chromosome is inactivated, X inactivation being random. Most women who have children with fragile-X syndrome are premutation carriers of normal intelligence.[15] Carriers of the premutation are intellectually unimpaired but are more vulnerable than other women to anxiety and depression.[16] Variants of fragile-X syndrome have now been identified, with DNA expansions nearer to the end of the long arm of the X chromosome. These include FraX-E and FraX-F.

(b) Physical characteristics

Physical features are variable. The most characteristic feature is that about 95 per cent of affected men have large testes, although macro-orchidism is not usually apparent until after puberty. Other features include a long face with a large forehead, large ears, a large lower jaw, and high-arched palate. There is a connective tissue disorder that may lead to tissue laxity with hyper-extensible joints, flat feet, heart defects (especially valve abnormalities), and ear infections (the eustachian tube closes easily). Cataracts and other eye abnormalities may occur, and lead to impaired vision. About 30 per cent of affected men have epilepsy. Life expectancy depends on the severity of associated features such as epilepsy and cardiovascular anomalies.

(c) Behavioural and psychiatric aspects

There is usually some degree of social impairment, with social anxiety and avoidance of eye-to-eye contact, but with social responsiveness. Men with fragile-X are usually affectionate, and do not have the aloof quality typical of autism. Self-injury is relatively common, especially hand biting over the anatomical snuff-box (between the bases of the thumb and index finger) in response to frustration, anxiety, or excitement. Stereotyped behaviours such as hand flapping are common.

The associated intellectual disability is usually mild to moderate. Verbal intelligence scores exceed performance scores among populations of affected men and non-disabled women carriers. Speech and language development is delayed. Speech is often disorganized, with rambling and circuitous conversation, incomplete sentences, poor topic maintenance, tangential comments, echolalia, and perseveration. It may be rapid, or include peculiar changes in pitch.

There may be problems with attention and concentration that are disproportionate to the severity of the associated learning disability. Hyperactivity may be the presenting feature among boys with fragile-X who do not have intellectual disability.

Further information can be obtained from Hagerman and Hagerman (2002)[17] and www.fragilex.org.uk.

Sex chromosome abnormalities

The Y chromosome is small and has been completely mapped.[18] The X chromosome is much larger, containing over 1000 genes. Abnormalities of the X and Y chromosomes are more prevalent than those affecting autosomal chromosomes. Many affected children are not significantly dysmorphic and do not have major developmental disabilities.[19] Some remain undiagnosed.

Klinefelter syndrome

(a) Prevalence and genetics

This is a disorder characterized by additional X chromosomes in phenotypic males. Two-thirds have a 47 XXY chromosome complement. Prevalence at birth is about 1 in 1000 live males, with a frequency in prenatally karyotyped male foetuses of 1 in 470.[19]

(b) Physical characteristics

Height, weight, and head circumference are below average at birth. Increased growth, especially of legs occurs from 3 years of age onwards. Affected men are usually taller than their fathers, and mean heights are around the 75th centile. Head size remains small. Puberty normally occurs, but testosterone production falls in early adult life. Affected adults have a normal-sized penis but small testes. About 60 per cent have some breast enlargement. Life expectancy is thought to be normal.

(c) Behavioural and psychiatric aspects

Boys with XXY are typically introverted and less assertive and sociable than other children, with poorer school performance (especially with regard to reading and spelling). Adults may have increased rates of antisocial behaviour and impulsiveness. The IQ distribution is skewed downwards, although measured full scale IQs run from the 60s to the 130s. Performance scores usually exceed verbal scores. Most affected children receive speech and language therapy, and expressive language deficits are often more pronounced than problems with receptive language. One follow-up study has been reported.[20] Further information: www.klinefeltersyndrome.org.

Turner syndrome

(a) Prevalence and genetics

The genetic abnormality in Turner syndrome is the loss or abnormality of one X chromosome in women. The 45,X karyotype is found in about 1 in 10 000 live female births. The abnormality is much more common at conception. About 99 per cent of affected foetuses are miscarried, and 45,X is the most common karyotype found in chromosomally aborted foetuses. About 50 per cent have a 45,X chromosome complement (a very small proportion of normal cell lines may be present). Most of the other cases are the result of mosaicism, some are the result of structural abnormalities of an X chromosome.

(b) Physical characteristics

Affected children have a short stature in childhood. Ovarian failure occurs before birth, and puberty does not usually occur naturally, although childbirth has, rarely, been reported. Dysmorphic features include a webbed neck, low hairline at the rear of the head, widely spaced nipples and multiple pigmented naevi. About 12 per cent have coarctation of the aorta or a ventricular septal defect.

(c) Behavioural and psychiatric aspects

Hyperactivity and distractibility are common in childhood. Poor social skills, with immature social relationships and low self-esteem in adolescence were reported in one study.[21] Women with Turner syndrome are usually of normal intelligence and verbal abilities are usually unimpaired or enhanced. Specific cognitive abnormalities including deficits in spatial perception, visual motor integration, affect recognition, visual memory, and attention have been reported.[22] The relative strength in verbal tasks may lead to an

overestimation of abilities. There is considerable variation in cognitive profile between affected women. Further information: www.tss.org.uk

XXX syndrome

(a) Genetics and prevalence

The 47,XXX syndrome occurs about 1 in 1000 female births.[23] Many are not diagnosed. There is a primary non-disjunction of a maternal or paternal X chromosome. The 48,XXXX chromosome complement is much rarer (about 40 cases have been reported so far).

(b) Physical characteristics

In 47,XXX syndrome newborn babies have a low birth weight and small head circumference. Height in adult life is usually increased, with a low body mass index. Fertility is not usually impaired, although there are reports of premature ovarian failure and recurrent spontaneous abortion. There may be deficits in balance or fine motor coordination. Life expectancy is thought to be normal.

(c) Behavioural and psychiatric aspects

Underactivity and withdrawal have been reported. Emotional development may be slowed. About a quarter of affected women in one follow-up study had repeated episodes of abdominal pain as teenagers for which no organic cause could be found.[24] Most appear to adapt to adult life without difficulties. Women with the syndrome usually have IQs between 80 and 90. Women with XXXX syndrome have lower IQs (55 to 75). An expressive language delay is typical. Some have a relatively poor short-term auditory memory. Further information: www.triplo-x.org.

XYY syndrome

(a) Genetics, prevalence, and physical characteristics

This karyotype is associated with 1 in 1000 live male births.[23] There is a primary non-disjunction of the Y chromosome. About 10 per cent have mosaic 46,XY/47,XYY chromosome complement. Offsprings rarely have two Y chromosomes. Affected individuals show increase in body and leg length between years 4 and 9. Most are over 10 cm taller than their fathers as adults. Sexual development and fertility are unaffected. Balance and coordination may be minimally compromised. Life expectancy is normal.

(b) Behavioural and psychiatric aspects

Early research found an increased frequency of XYY men among inmates of special prisons.[25,26] More recent studies examining the relationship between 47,XYY karyotype and behaviour have concluded that affected men have lower mean intelligence scores (with a large overlap with the normal range) and poorer social adaptation. Distractibility, hyperactivity, temper tantrums, and speech and language problems appear relatively common in childhood. There is little evidence to suggest a significant link with seriously aggressive criminal conduct in adult life.[27,28]

Foetal alcohol syndrome

(a) Classification and prevalence

Exposure of the developing foetus to significant amounts of alcohol leads to cognitive impairment. The effect can occur during any stage of pregnancy, because brain development continues during all three trimesters. Dysmorphology, including a facial dysmorphology, can also occur. Foetal alcohol spectrum disorder (FASD) includes a number of subtypes including foetal alcohol syndrome (FAS), and more subtle abnormalities subsumed under the terms possible foetal alcohol effects (PFAE), prenatal exposure to alcohol (PEA), or alcohol-related neurodevelopmental disorder (ARND).

Foetal alcohol exposure is thought to be a common cause of intellectual disability in the United States and other developed countries. In the United States, an estimate of 0.33 per 1000 births has been given for the prevalence of foetal alcohol syndrome.[29] Alcohol inhibits *N*-methyl-D-aspartate receptors, which mediate postsynaptic excitatory effects of glutamate, and this is thought to have an effect on cell proliferation.[30]

(b) Clinical features

A number of abnormalities have been linked to FAS. Facial dysmorphology commonly includes a thin upper lip and smooth philtrum. The jaw may be small. Low-set abnormal ears and palate abnormalities can occur. Other abnormalities include growth retardation, skeletal abnormalities (deformed ribs and sternum, spinal curvature, dislocated hips, fused or webbed or missing fingers or toes, limited joint movement, small head), heart abnormalities, and urinary tract anomalies.

(c) Neurological and behavioural aspects

Central nervous system abnormalities include a small brain with abnormally arranged cells. Intellectual disability is usually mild or moderate but may be severe. Other problems commonly include reduction in attention span, overactivity, irritability in infancy; and coordination problems.

Angelman syndrome

(a) Prevalence and genetics

The prevalence of this syndrome is around 1 in 10 000 births.[31] Most cases are sporadic, and associated with deletions within 15q11q13 of maternal origin (Prader–Willi syndrome). Angelman syndrome is occasionally associated with paternal uniparental disomy (both chromosome 15s are of paternal origin) but this is less common than in Prader–Willi syndrome. Other genetic abnormalities leading to Angleman syndrome are an imprinting centre defect (this incorrectly 'marks' the chromosome, through methylation, as being from a parent of the opposite sex) and mutations in the gene responsible for the Angelman syndrome phenotype (UBE3A, coding for a ubiquitin ligase enzyme).[32] UBE3A is expressed only from the maternal chromosome, and in Angelman syndrome expression in relevant brain areas is only around 10 per cent of normal.

(b) Physical characteristics

Physical characteristics include a small head, characteristic face with wide mouth, 'hooked' nose, prominent lower jaw, widely spaced teeth, and tongue protrusion. Many affected children are hypopigmented compared to first degree relatives due to deletion of a gene related to pigmentation.[33] Voluntary movements are jerky and the gait ataxic with stiff legs. About 80 per cent develop epilepsy, and the EEG is highly characteristic.

(c) Behavioural and psychiatric aspects

Behavioural characteristics, including sudden bursts of laughter and the jerky ataxic gait, led to the term 'happy puppet' syndrome

being used in the literature of the 1960s and 1970s. It is no longer considered appropriate. Affected children enjoy social and physical contact, and mouthing objects. Many are fascinated by water. Intellectual disability is severe, with markedly delayed motor milestones. There is little speech development (no person reported in the literature has more than a six word vocabulary), but understanding of language may be better. Overactivity is often associated with a short attention span in childhood, but may improve with development. Behavioural studies have been reported[34] and genetic aspects reviewed.[35] Further information: www.assert.dial.pipex.com/

Coffin–Lowry syndrome

(a) Prevalence and genetics

Coffin–Lowry syndrome is one cause of X linked intellectual disability. The syndrome has been ascribed to a locus in the Xp22.1-p22.2 region. More than 100 cases have been reported.

(b) Physical characteristics

Physical features include short stature, facial dysmorphology including slanting eye fissures, prominent forehead, short broad nose, forward facing nostrils, large ears, large mouth and small, widely spaced teeth. Increased fatty tissue is deposited in the forearms. Hands are often large, with tapering fingers. Ligament laxity may lead to flat feet. Spinal and chest abnormalities occur. Behavioural and emotional characteristics are largely unknown, although depression and schizophrenia have been reported in association with the disorder and in female carriers.

(c) Behavioural and psychiatric aspects

Affected men usually have severe learning disabilities. Drop attacks and sleep apnoea syndrome have been reported. Further information: www.clsf.info.

Congenital hypothyroidism

(a) Prevalence

Following the introduction of a neonatal screening programme in the United Kingdom, the incidence of congenital hypothyroidism (identified through heel-prick screening and further investigation for at risk infants) is about 1 in 4000, and occurs more commonly in girls. In many cases the deficiency of thyroid hormones is mild, and there are few symptoms.

(b) Physical and cognitive characteristics

Severely affected children have a distinctive appearance with a puffy face and a large tongue that protrudes from a mouth that is kept open. Other features include dry, brittle hair, a low hair line, jaundice, sleep disorders, low muscle tone, constipation, and failure of cognitive development leading to intellectual disability. If untreated, even mild hypothyroidism may lead to intellectual disability.

Cri-du-Chat syndrome (CDCS, 5p-syndrome)

(a) Prevalence and genetics

Cri-du-Chat syndrome was originally described as a syndrome of multiple congenital anomalies, intellectual disability, microcephaly, abnormal face, and a mewing cry in infants with deletion of a 'B group chromosome', later identified as a 5p terminal deletion. The prevalence is about 1 in 35 000 births. Deletions vary in size, but the critical region for Cri-du-Chat syndrome is thought to be 5p15.2. About 85 per cent of the deletions arise spontaneously and the majority are of paternal origin. About 15 per cent of affected people have an unbalanced translocation, and the clinical features depend on the other chromosome involved. Fewer than 1 per cent of cases are due to inherited deletions, which are usually very small.

(b) Physical characteristics

In infancy there are feeding difficulties and the cry is abnormally high pitched (cat-like, hence 'cri-du-chat'), but this is not an invariable feature. The gene causing the abnormal (cat-like) cry has been located at 15p13. It is possible for infants with small deletions to have a cat-like cry but no other features of CDCS, or features of CDCS without the characteristic cry. A round face with widely spaced slanting eyes, a small head, a broad flat nose, and small lower jaw are characteristic. Ear abnormalities may occur. Larger deletions, and some translocations, are associated with more pronounced clinical features such as lower intelligence, smaller stature, lower weight, and smaller head. The face often lengthens with development and may be asymmetrical. Cleft lip or palate, curved fingers, hernias, and orthopaedic abnormalities may occur. Older individuals often have premature greying of the hair.

(c) Behavioural and psychiatric aspects

Hyperactivity is a problem for a substantial proportion of children, but may improve with age.[36] Language development is often markedly delayed. The IQ associated with the syndrome in one study varied from 6 to 85. Further information: www.criduchat.asn.au.

De Lange syndrome (Brachmann–de Lange syndrome)

(a) Prevalence and genetics

This syndrome is considered to occur about once in 60 000 live births, although some authors believe it to be more common. Mutations in a large gene on chromosome 5, the Nipped B like or *NIPBL* gene (named because its function resembles that of a fruit fly gene that produces a nipped wing), have been shown in about 40 per cent of people with the syndrome.[37]

(b) Physical characteristics

Affected individuals show growth retardation; distinctive facial features consisting of well-defined arched eyebrows which meet in the middle, long curled eyelashes, small nose with forward-facing nostrils, and down-turned mouth with thin lips and limb abnormalities such as small or shortened limbs, especially arms. Hearing impairments, gut malformations, and congenital heart defects also occur. Early mortality is high because of feeding problems with regurgitation and vomiting leading to aspiration pneumonia in some cases.

(c) Behavioural and psychiatric aspects

Self-injury, autistic features, and pleasurable responses to vestibular stimulation, e.g. spinning in a chair have been reported as part of behavioural repertoire. The degree of learning disability is usually severe, and speech is often very limited. However, some affected people have IQs within the normal range. Clinicians should be alert to the presence of pain and discomfort resulting from gastro-oesophageal reflux and other gastro-intestinal abnormalities. Further information: www.cdlsusa.org.

Duchenne muscular dystrophy

(a) Prevalence and genetics

This is an X linked recessive condition in which deletions, duplications, and mutations at Xp21 lead to failure to produce dystrophin, a protein component of muscle tissue. New mutations account for about 30 per cent of cases. The prevalence at birth is about 1 in 4000 male births.

(b) Physical characteristics

The syndrome is characterized by progressive muscle weakness, affecting the pelvis, upper leg, and upper arm muscles first. The onset is typically between 2 and 6 years of age. Respiratory muscles are involved later in the disease process. Heart muscle abnormalities may also occur. The disease is usually more severe in the lower limbs and trunk initially, with later involvement of the arms and respiratory muscles. Affected boys often need a wheelchair by around 11 years of age, with death in early adult life (typically in the mid twenties).

(c) Behavioural and psychiatric aspects

Low mood, anxiety, and social abnormalities are often problems, and may become more prominent as the disorder progresses. These features may be reactions to a chronic and progressive physical disease. Specific learning disabilities are common, especially specific reading disorder. About 25 per cent of those affected have a learning disability. Performance IQ is typically higher than verbal IQ. Further information: www.mda.org.au.

Lesch–Nyhan syndrome

(a) Prevalence and genetics

This X-linked recessive disorder results from a deficiency of a purine salvage enzyme, hypoxanthine-guanine phosphoribosyl transferase (HGPRT) leading to hyperuricaemia, and neurological disorder. Partial HGPRT deficiency results in gout. HGPRT is a 217 amino acid peptide coded for by one gene divided into nine exons, located on the X chromosome at Xq26q27. Many different genetic lesions can cause HGPRT deficiency. Complete and partial deletions, insertions, and duplication of exons have been reported. Most lesions appear to be point mutations. Affected males may have had spontaneous mutations or inherited mutations from asymptomatic female carriers. Carrier detection and prenatal diagnosis are possible. The incidence is around 1 in 380 000 births.[38]

(b) Physical characteristics

Neurological features include athetoid and other abnormal movements and spasticity. Growth retardation is usual. The presentation is usually with hypotonia and motor delay at about 4 months. Extrapyramidal signs (such as spasticity and choreo-athetoid movements) develop at about 9 months. Hyper-reflexia and clonus appear at about 1 year. Dystonic movements may also develop. Dysarthria is common. Affected individuals may survive to the second or third decade. Death is usually due to kidney failure secondary to uric acid deposition or infection. The syndrome is associated with abnormal neurotransmitter turnover in the basal ganglia.

(c) Behavioural and psychiatric aspects

Compulsive severe self-injury is very prevalent and usually consists of finger and lip biting, with self-splinting in an attempt to prevent the behaviour.[39] Other compulsive behaviours occur; men with the syndrome are reported to hit, spit, and swear at caregivers while apologizing for their behaviour at the same time.[40] The mean age at onset of self-injury is 3.5 years, with wide variation. The IQ is usually between 40 and 80, but dysarthria and neurological problems limit the validity of standard IQ tests. Further information: www.lndinfo.org.

Mucopolysaccharidoses

(a) Classification, genetics, and prevalence

The mucopolysaccharide group of disorders have both names (Hunter syndrome, Hurler syndrome, Sanfillipo syndrome, Morquio syndrome, Schie syndrome, Maroteaux–Lamy syndrome, Sly syndrome) and numerical designations (MPS IIA/B, MPS IH, MPS IIIA/B/C/D, MPS IVA/B, MPS IS, MPSVI, MPSVII, respectively). The disorders result from deficiencies in enzyme systems involved in the degradation of glycosaminoglycans leading to the accumulation of abnormal metabolic products. The prevalences among live born children are approximately 1 in100 000 for Hunter and Hurler syndromes, 1 in 200 000 for all types of Sanfillipo syndrome and for Morquio syndrome, and 1 in 500 000 for Schie syndrome. Hunter syndrome is much more prevalent in Israel. The transmission is autosomal recessive except in Hunter (IIA and IIB) which is X linked.

(b) Physical characteristics

Physical features vary. Coarse facial features ('gargoylism'), hepatosplenomegaly, joint stiffness, eye abnormalities, and short stature occur in many of the disorders. Life expectancy varies from death in the first decade in Hurler syndrome through survival into second or third decade in Sanfillipo syndrome, to survival to adult life in Hunter syndrome and Schie syndrome.

(c) Behavioural and psychiatric aspects

Sleep problems and abnormal nocturnal behaviours such as staying up all night, night-time laughing and singing, sudden crying out, and chewing of bedclothes have been reported in association with Sanfillipo syndrome, and have been shown to respond to behavioural management strategies. Other problem behaviours reported in association with MPS disorders include aggression, overactivity, restlessness, and anxiety. Cognitive abilities vary from normal intelligence in Schie syndrome to severe learning disability and progressive cognitive deterioration in Hurler syndrome. Sanfillipo syndrome is associated with slower progressive cognitive impairment than that seen in Hurler syndrome, but often with marked behavioural and psychiatric abnormalities consistent with the diagnosis of childhood disintegrative disorder. The susceptibility to tooth decay in Morquio syndrome can lead to pain and problem behaviours. Further information: www.mpssociety.org.

Neurofibromatosis type 1

(a) Prevalence and genetics

This autosomal dominant disorder was first described by von Recklinghausen in 1882 and occurs about once in 3000 births. The gene responsible is localized to 17q11.2. The gene product, neurofibromin, regulates cell division and is thought to suppress tumour formation. A high spontaneous mutation rate means that about a half of all cases of NF1 arise in unaffected families.

(b) Physical characteristics

Tumours arise from the connective tissue of nerve sheaths. Two or more of the following features are usually required for diagnosis:

six or more cafe au lait (light brown) skin lesions more than 5 mm in diameter before puberty or 15 mm after puberty, two or more neurofibromas or one plexiform neurofibroma (tumours of the nerve sheath); freckling of the inguinal or axillary region; two or more lisch nodules (benign iris hamartomas); an optic nerve glioma (tumour); a bony lesion characteristic of neurofibromatosis (usually shin bowing or scoliosis), a first degree relative with the disorder. About 45 per cent of affected people will have non-enhancing hyperintensities (or unidentified bright objects 'UBOs') on magnetic resonance imaging. These are commonly seen in the cerebellum, basal ganglia, brain stem, and thalamus.[41]

(c) Behavioural and psychiatric aspects

About 50 per cent of children have speech or language abnormalities. Distractibility and impulsiveness may be problems. Learning disability is present in about 10 per cent of affected people. Specific developmental disorders such as difficulties with reading, writing, or numeracy affect about half of the children. Visuo-spatial abnormalities and lack of coordination have also been described. Further information: www.geneclinics.org/profiles/nf1.

Phenylketonuria

(a) Prevalence and genetics

Classical phenylketonuria affects about 1 in 10 000 live born children in the United Kingdom. Other hyperphenylalaninaemias also occur. The disorder results from a deficiency of the enzyme phenylalanine hydroxylase. The extent of the deficiency varies, with a spectrum of resulting clinical conditions from classical phenylketonuria to benign hyperphenylalaninaemia. The gene regulating phenylalanine hydroxylase is located at 12q22-24.1. It is subject to various mutations. The classical form is inherited in an autosomal recessive manner. Prenatal diagnosis and the detection of heterozygotes with one defective copy of the gene are possible. About 2 per cent of cases are due to a deficiency of tetrahydrobiopterin rather than phenylalanine hydroxylase.

(b) Physical characteristics

Physical features include blond hair, blue eyes, eczema, and microcephaly (in half the suffers), epilepsy (in a quarter) and tremor and movement disorders or spasticity. Untreated infants have an unusual mouse-like body odour. In the United Kingdom all neonates are screened for the disorder. A low phenylalanine diet is usually continued through childhood. There is debate about the age at which it is appropriate to lift or relax dietary restrictions. Amino-acid supplements may be used to block phenylalanine uptake. Dietary control is essential when affected women become pregnant, because hyperphenylalaninaemia is toxic to the foetus leading to learning disability, microcephaly, and facial and heart abnormalities. Theories about the toxic effects of hyperphenylyalaninaemia include direct toxicity, competition for transport across the blood–brain barrier and dopamine depletion.[42]

(c) Behavioural and psychiatric aspects

Untreated phenylketonuria is associated with a number of maladaptive behaviours and behavioural syndromes including overactivity, self-injury, and autism. Autism and many of the other features do not occur in children managed with low phenylalanine diets. Those who have not been treated may have moderate to profound learning disabilities, irritability, and marked social impairments. Inadequate dietary control is associated with deficits in mathematical, visuo-spatial, and language skills. Further information: www.nspku.org.

Prader–Willi syndrome

(a) Prevalence and genetics

The prevalence is around 1 in 40 000 live born infants.[43] About 70 per cent of those affected have a deletion affecting the long arm of chromosome 15 (del 15q11q13), the deleted chromosome always being of paternal origin. About 29 per cent have maternal uniparental disomy (MUPD) in which both chromosome 15s are inherited from mother, with no paternal chromosome 15. About 1 per cent have an imprinting error, in which the paternal chromosome is incorrectly methylated so as to resemble a maternal one.

(b) Physical characteristics

Infants are hypotonic or floppy and have feeding problems associated with a failure to suck. Many are tube fed. In early childhood there is a switch to marked overeating. Affected adults are of short stature, have small hands and feet and a characteristic pattern of facial appearance, and a lack of sexual development. Affected people were often obese, as a result of the impaired satiety leading to overeating, but modern dietary management and treatment with growth hormone in childhood may lead to near normal body size and shape. There is an increased prevalence of curvature of the spine or scoliosis and other orthopaedic abnormalities, and diabetes or heart failure may result from obesity. Life expectancy depends on severity of obesity.

(c) Behavioural, cognitive, and psychiatric aspects

Affected individuals have an almost insatiable appetite. They may steal food and consume 'unpalatable' food such as rotting or frozen food or pet food. A variety of sleep abnormalities and a lowering of the threshold for loss of temper may be associated. About 80 per cent pick or scratch their skin. Insistence on routines, and compulsive behaviours are commonly reported.[44] Severe psychiatric disorders including affective and psychotic states are more prevalent, especially among people with MUPD.[45] Anecdotal reports suggest the pain threshold may be raised.

About 5 per cent of those with this syndrome have overall cognitive abilities with IQs in excess of 85, 27 per cent have borderline cognitive abilities with IQs between 70 and 85, 34 per cent have mild learning disabilities, 27 per cent moderate, 5 per cent severe, and less than 1 per cent have profound learning disability. There are deficits in auditory information processing, and relative strengths in visuo-spatial tasks. Further information: www.ipwso.org.

Rett syndrome

(a) Prevalence and genetics

Rett syndrome causes severe intellectual disability in women. The prevalence in the United Kingdom is around 1 in 10 000 women.[46] The syndrome results from a mutation in the MeCP2 gene located at Xq28. The mutation was considered to be lethal in males but there are a small number of males with the syndrome. The severity of the syndrome in women depends on the percentage of cells with the normal MeCP2 gene active after X inactivation. If more of the X chromosomes with normal MeCP2 gene have been inactivated, the syndrome is likely to be more severe. MeCP2 acts as a mechanistic bridge between DNA methylation and histone methylation.[47]

(b) Physical characteristics

The affected child appears normal at birth. For the first 12 months no major abnormalities are apparent though the child may be placid, lack muscle tone, or be relatively immobile. She acquires skills to about 1 year with regression and loss of skills from around 18 months onwards. Speech and use of hands are particularly affected. Physical problems increase with age, and include scoliosis, spasticity, and leg deformities. Epilepsy is common. Pathological changes include a reduction in brain size with reduced cortical thickness, reduced neuronal branching, and depigmentation of the basal ganglia. Many affected girls reach adulthood, but about 1 per cent of them die each year with early death more likely with increasing physical disability.

(c) Behavioural and psychiatric aspects

Sleep disturbance, withdrawal and episodes of crying occur during the phase of regression around 18 months of age. This is followed by a phase in which development stops. Extreme agitation and over-breathing interspersed with episodes of cessation of breathing then become apparent. The most prominent feature of the behavioural phenotype is the presence of stereotyped movements, especially midline 'hand-wringing' movements. Affected women and girls usually have profound learning disability. Further information: www.rettsyndrome.org.uk.

Rubinstein–Taybi syndrome

(a) Prevalence and genetics

This syndrome is one of the 25 most common multiple congenital anomaly syndromes seen in genetic clinics in the United States and has an estimated incidence at 1 in 125 000 live born infants. Microdeletions at 16p13.3 have been described in some cases, and mutations in the gene coding for CREB-binding protein (CBP) found at this locus have been reported to cause the syndrome.[48] A few apparently familial cases have been reported, and four sets of concordant monozygotic twins have been reported.

(b) Physical characteristics

The affected individuals are usually short, have a small head, a beaked or straight nose and downward slanting eyes. They have a stiff gait. The thumbs and first toes have broad terminal phalanges, often with an angulation deformity. Other congenital anomalies are not uncommon. Inadequate weight gain in infancy, congenital heart defects, urinary tract abnormalities, and severe constipation contribute to morbidity reflected in a hospitalization rate 10 times higher than the general population.

(c) Behavioural and psychiatric aspects

Findings from postal questionnaire surveys in the United States and United Kingdom indicate that people with the syndrome have a friendly disposition, a propensity to self-stimulatory activities such as rocking and an intolerance of loud noises. Reduced attention span, rocking, spinning, and hand flapping were common in the UK survey.[49] Intellectual disability is usually moderate. Further information: www.rubinstein-taybi.org.

Smith–Lemli–Opitz syndrome

(a) Prevalence and genetics

This disorder is thought to occur about once in 30 000 live births. Mildly affected people may be undiagnosed. The syndrome is said to be one of the commonest autosomal recessive conditions affecting people of White European origin in North America, but rare among people of African or Asian origin. The male to female ratio appears to be around 3 to 1 but this may be due to the relative ease with which sexual abnormalities are detectable in men. Abnormalities in the *DHCR7* gene, located at 11q12-13 results in deficiency of the enzyme 7-dehydrocholesterol reductase results in elevated levels of a cholesterol precursor. Treatment with cholesterol supplementation has been attempted.[50]

(b) Physical characteristics

During pregnancy, the foetus may show growth retardation. Affected individuals may have a small head, drooping eyelids, squint, forward-facing nostrils, small lower jaw, and finger abnormalities such as extra fingers and syndactyly. Males have abnormalities of their external genitalia such as small testes or penis, hypospadias, undescended testes, and female type genitalia. Cleft palate and abnormalities of almost all major organ systems may also occur.

(c) Behavioural and psychiatric aspects

There is little information available about behavioural and cognitive characteristics. Intelligence varies from normal to severe learning disability. Aggressive and self-injurious behaviours and autistic spectrum disorders have been reported. Further information: www.geneclinics.org/profiles/slo.

Smith–Magenis syndrome

(a) Prevalence and genetics

This syndrome, affecting around 1 in 50 000 births, is associated with deletions at 17p11.2.

(b) Physical characteristics

Affected individuals have flattened mid-face, abnormally shaped upper lip, short hands and feet, single transverse palmar crease, abnormally shaped or placed ears and sometimes a high arched palate or protruding tongue. The facial features may coarsen with development. Ear and eye disorders such as otitis media and squint are relatively common.

(c) Behavioural and psychiatric aspects

Newborn babies with the syndrome are usually placid, 'floppy' and feed with difficulty. This changes to hyperactivity, self-injury (e.g. head banging, pulling out finger and toe nails and the insertion of objects into body orifices), from about 18 months onwards. Self-hugging and mid-line hand clapping have been reported. Sleep disorders are common with some children waking repeatedly in a state of agitation. An absence of rapid eye movement (REM) sleep has been reported in some patients. Many affected children appear to be relatively insensitive to pain. Behavioural studies have been reported.[36,51] The severity of the cognitive impairment correlates with the size of the 17p11 deletion. Moderate intellectual disability is common. Speech delay is more pronounced than delay in motor achievements. Further information: www.prisms.org.

Tuberous sclerosis

(a) Prevalence and genetics

About 1 in 7000 people are affected. It is an autosomal dominant condition but up to 80 per cent of cases arise as a result of spontaneous mutations. The disorder is genetically heterogeneous, with gene linkage to 9q34 and 16p13.

(b) Physical characteristics

Physical features are very variable. The previously used diagnostic triad of epilepsy, intellectual disability and a characteristic facial skin lesion is seen in only about 30 per cent of people with the disease. The disorder is a multi-system one, with hamartomatous tumours (arising from primitive cells) affecting the brain (in about 90 per cent), skin, kidneys, heart, eyes, teeth, bones, lungs, and other organs. About 80 per cent of affected people have epilepsy. Brain tumours and kidney lesions are common causes of death.

(c) Behavioural and psychiatric aspects

Tuberous sclerosis is associated with autism and related disorders, hyperactivity and attention-deficit disorder, obsessive and ritualistic behaviour, sleep problems, and occasionally self-injurious or aggressive behaviours. Less than half of affected people have a learning disability. Attention-deficit is common. Of those with learning disability, many have an IQ less than 30. Further information: www.tuberous-sclerosis.org.

Velo-cardio-facial syndrome

(a) Prevalence and genetics

First described in 1978[52] this condition is relatively common, affecting about 1 in 2000 people. The disorder has also been called Shprintzen syndrome, Digeorge syndrome, Cayler syndrome, Takao syndrome, conotruncal anomalies face syndrome, 22q11 deletion syndrome, and CATCH22. It is associated with microdeletions at 22q11. About 90 per cent arise *de novo*, with 10 per cent having an affected parent.

(b) Physical characteristics

Physical features include cardiac abnormalities including ventriculoseptal defects, pulmonary stenosis, and cardiac outlet abnormalities; facial dysmorphology with a prominent nose with broad bridge and squared tip, small head or small lower jaw; ocular abnormalities; cleft palate; short stature and long, thin, hyperextensible fingers. The clinical features associated with the disorder are highly variable in both type and severity.

(c) Behavioural and psychiatric aspects

Many affected individuals have difficulties with reciprocal social interaction.[53] A high prevalence of severe psychiatric disorders is reported in later life, including high rates of bipolar affective disorder[54] and schizophrenia.[55] Anxiety, social withdrawal and other disorders have also been described. Over 90 per cent have a learning disability. Speech and language problems are common. Further information: www.vcfsef.org.

Williams syndrome

(a) Prevalence and genetics

Also known as idiopathic infantile hypercalcaemia, the syndrome affects about 1 in 15 000 infants. Most cases are sporadic though a few familial cases have been reported where the transmission seems to be autosomal dominant. The syndrome is a contiguous gene deletion disorder in which there is variable loss of genetic material involving the Elastin gene at 7q11.3 and sometimes as many as 17 nearby genes in the Williams syndrome critical region.[56]

(b) Physical characteristics

Infants have difficulties in feeding, are irritable, have constipation and fail to thrive. Over 60 per cent of children have high serum calcium concentrations. This can be treated with a low-calcium diet and vitamin D restriction. The face is distinctive, with prominent cheeks, a wide mouth and flat nasal bridge often described as 'elfin-like'. Kidney and heart lesions (especially supravalvular aortic stenosis and peripheral pulmonary artery stenosis) are common. Growth is usually retarded. Life expectancy is related to metabolic and heart abnormalities.

(c) Behavioural and psychiatric aspects

Social disinhibition with abnormal friendliness to strangers, overactivity, poor concentration, eating and sleeping abnormalities, abnormal anxiety, poor peer relationships, and abnormally sensitive hearing have been reported.[57,58] About 95 per cent of children with the disorder have a moderate or severe learning disability. Verbal abilities are better developed than visuo-spatial and motor skills. There is an unusual command of language in which expressive language is superficially fluent and articulate but comprehension is far more limited. Further information: www.williams-syndrome.org.

Further information

Information about individual syndromes is available from the source listed after the relevant syndrome in the text above. O'Brien (2002)[2] gives information about behavioural, cognitive, linguistic and psychiatric aspects of several genetic disorders. The Contact a Family Directory of Specific Conditions and Rare Disorders (CaF Directory) is widely used for basic information about characteristics and carer organizations. A new paper edition is published in January each year and it is also available in CD-ROM format on a quarterly subscription basis: www.cafamily.org.uk.

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10.5

Psychiatric and behaviour disorders among mentally retarded people

Contents

10.5.1 Psychiatric and behaviour disorders among children and adolescents with intellectual disability

Bruce J. Tonge

Introduction

Psychopathology is 2–3 times more common in intellectually disabled (ID) children than in the general population.[1,2] Psychiatric disorder is the most common source of additional handicap causing loss of educational, recreational, and social opportunity, burden for carers and cost to the community. Numerically, the size of this problem is approximately equal to schizophrenia, but is less well-recognized due to diagnostic over shadowing in which psychiatric disorder is not differentiated from ID as a separate condition open to diagnosis and treatment.[2] Although there is probably a significant reduction in overall prevalence of psychiatric disorders from approximately 43 per cent of children to 37 per cent of young adults with ID, psychopathology if present in childhood is likely to persist.[2] The profile of disorders varies from childhood into young adult life with the prevalence of attention-deficit hyperactivity symptoms decreasing, the frequency of symptoms of depression increasing and the prevalence of anxiety remaining stable with maturation.[2]

Diagnosis and classification

There are two approaches to the description and classification of psychopathology in young people with ID. First is the application of DSM and ICD diagnostic criteria. The reliability and validity of this approach is not well established when applied to children with ID.[3] Young people with more severe ID and language impairment are unable to report abnormalities of their emotions, thoughts, and perceptions, which are criteria for conditions such as obsessive-compulsive disorder (OCD) and schizophrenia. Some diagnoses, for example attention-deficit hyperactivity disorder (ADHD)[4] require a judgement that symptoms are inconsistent with developmental level, which in a child with ID is delayed relative to chronological age. The DSM-IV TR[4] specifies that either ADHD or separation anxiety disorder should not be made 'exclusively during the course of a pervasive developmental disorder'. These restrictions on comorbid diagnosis should not limit the necessity to describe the range of presenting symptoms and offer appropriate treatment, for example the use of stimulant medication in a child with autism and severe ADHD symptoms. Developmental level and degree of cognitive impairment influence the presentation of symptoms. For example, children with ID are more likely than children in general, to have externalizing symptoms such as disruptive, aggressive, impulsive, or avoidant behaviours; if psychotic to experience hallucinations without delusions; or if depressed to present with irritability and stereotypies. Self-absorbed, autistic, and withdrawn behaviours are more common in children with severe ID whereas anxiety, disruptive, and aggressive behaviours are more likely in children with milder levels of ID.[2] Some patterns of psychopathology recognized by DSM-IV TR[4] are specifically associated with more severe ID such as 'stereotypic movement disorder with or without self-injurious behaviour'. Other emotional and behavioural disturbances seen in people with ID receive non-specific, atypical, or not otherwise specified classifications and await better definition. Recent attempts to produce diagnostic criteria for psychiatric disorders in people with ID (the draft ICD-10 guidelines for the psychiatric assessment

of persons with mental retardation,[5] the Royal College of Psychiatrists diagnostic criteria for psychiatric disorders for use with adults with learning disabilities[6] and the DSM-IV TR for intellectual disability[7] are mainly designed for use with adults and require clinical validation).

The second approach to the definition of psychopathology in young people with ID is the use of informant questionnaires which rate disturbed emotions and behaviour. Factor analysis produces subscales which have clinical utility and refer to dimensions of disturbance such as disruptive/antisocial behaviours, social withdrawal, self-absorbed behaviours, communication disturbance, and anxiety/depression. Two reliable questionnaires validated for use in children and adolescents with ID are the Nisonger Child Behaviour Rating form[8] and the Developmental Behaviour Checklist.[9]

The multiaxial classification system of DSM or ICD, revised for use in people with ID, should form the basis of diagnosis of psychiatric disorder in young people with ID, but are usefully supplemented with standardized information gathered from informant questionnaires.

Contributing factors and context

Assessment of the psychopathology associated with ID requires consideration of the biopsychosocial context.

(a) Cognitive profile

A standardized cognitive assessment provides essential information to inform diagnosis and guide treatment. The level of intellectual and language ability gives an indication of the child's capacity to comprehend and communicate their perceptions, thoughts, and emotions. Subjective experiences such as grief, anxiety, hallucinations, and delusions cannot be assessed if the child is unable to communicate; therefore, psychopathology is more likely to be indirectly expressed by behaviour similar to that seen normally in younger children. For example, depression may be manifest as irritability, anxiety displayed by rocking or aggression and auditory hallucinations inferred from distressed covering of ears or self-injury.[7] Diagnosis is more speculative when the level of ID is more severe because the expression of emotions and behaviour is more atypical hence there is a greater use of unclassified or organic brain syndrome diagnoses. The cognitive subtest profile may also assist diagnosis. For example, children with autism usually perform better on visuo-motor tasks compared to verbal, imitation, and social comprehension tasks and therefore communicate and learn better if information is presented visually. The discovery of inattention and working memory deficits might help to confirm a diagnosis of ADHD.

(b) Temperament

As for the general population, difficult temperamental characteristics such as high levels of emotionality and activity and poor sociability, increase the risk of emotional and behavioural disorders, particularly in boys with mild ID. A difficult temperament might be enduring but improved parental understanding and management skills improve adaptation and reduce disturbed behaviour.

(c) Medical issues

A medical assessment is necessary, both to establish the cause of the ID, if known, and to determine if any medical conditions might be contributing to the emotional and behavioural problem. ID is associated with an increased risk of poor health in general, of brain disorders such as epilepsy (e.g. affecting 20 per cent of children with autism) and of medical complications associated with known causes of ID, such as cardiac and bowel abnormalities in Down syndrome, sensory impairments and deafness in Rubella embryopathy and the neuro-cutaneous brain lesions of tuberous sclerosis which are associated with tic disorder, autistic symptoms, and psychosis.[10] Disturbed behaviour might be the only manifestation of illnesses such as migraine, dental caries, and otitismedia in children with ID who are unable to talk about their pain. Psychoactive drugs are overprescribed in children with ID and their side effects are a well-recognized cause of behavioural and emotional disturbance and paradoxical effects. For example neuroleptic drugs may produce drowsiness, akathisia, and dystonic reactions. Irritability, anxiety, mood disturbance, and tics can be unacceptable side effects of stimulant medication. When prescribing drugs it is essential to systematically record behaviour and monitor side effects to confirm that the drug has a beneficial effect on target symptoms.[9]

(d) Behavioural phenotype

Specific genetic causes of ID often have characteristic patterns of psychopathology of relevance to diagnosis, treatment, and research (see Table 10.5.1.1).[11,12]

(e) Social and family influences

Children with ID are more likely than other children to experience adverse events such as poverty, socio-economic disadvantage, respite care and institutional care, rejection, social exclusion, teasing, school adjustment problems, abuse and neglect.[13] Their limited cognitive ability to comprehend adverse experiences may compromise adaptation. Parental stress, grief, guilt, and mental health problems and poor socio-economic circumstances are factors which are likely to adversely affect attachment and the quality of family care and aggravate child psychopathology.[14] In turn, behaviour problems, communication difficulties and lack of social responsiveness, for example in children with autism, predict maternal stress and mental health problems and placement of the child in out of home care.[15] Cultural responses, expectations, and attitudes may also influence parenting practices and the nature of care provided to children with ID. Observation and assessment of the quality of care, adverse events, parental mental health, family stress, resources, and community support is necessary to understand their contribution to psychopathology and implications for management. These factors are listed in AXIS V of the draft ICD-10 guide for mental retardation.[5]

Specific psychopathological disorders in children with ID

Behavioural disorders

(a) Attention-deficit hyperactivity disorder

Diagnosis of ADHD in children with ID is relatively straightforward because the DSM-IV TR[7] and the ICD[5] criteria are based on observable behaviour such as distractibility and fidgeting. This observed behaviour must also be 'inconsistent' with the child's developmental level. For example, the attention span of a 9-year-old child with a moderate ID would need to be less than that of a typical 3-year-old. Symptoms of inattention and hyperactivity

Table 10.5.1.1 Behaviour phenotypes

Syndrome	Genetics	Behavioural phenotype
Down	Trisomy 21 (1 in 800 live births)	Range of ID but usually moderate to severe Relatively lower rates of psychopathology (20–30%) Childhood: oppositional, attention-deficit problems Young adult: affective disorder, early-onset dementia
Fragile X	Expansion of CGG trinucleotide sequence at Xq27.3	Mild to moderate ID Verbal IQ > Performance IQ Shy, gaze avoidant, anxious inattentive, hyperactive, schizotypal disorder (females) 5–10% have autistic disorder but most responsive to social cues and form attachments Behaviour may settle with age
Prader–Willi	Paternal deletion long arm chromosome 15q 11–13 (70%) or maternal disomy chromosome 15 (25%) or a mutation	Mild/borderline ID Hyperphagia and food obsession Mild obesity, serious psychopathology (50%+) OCD (e.g. questioning, cleanliness) impulsivity, aggression, defiance, skin picking In adolescents anxiety, depression, psychosis (with maternal disomy)
Smith Magenis	Chromosome deletion at 17p 11.2	Moderate ID Severe psychopathology: hyperactive, impulsive, aggressive, insomnia, stereotypic movements (e.g. self-hugging) self-injury (e.g. nail pulling, head banging)
Williams	Micro-deletion on chromosome 7q 11.23 (elastin gene)	Moderate ID Visuo-spatial/motor deficits but recognize facial features, loquacious with stereotypic phrases Children: endearing, 'elfin-face', irrepressible, and affectionate, hyperacusis, phobias, anxiety, inattention, insomnia

must be present in at least two settings such as at home and at school. Symptoms of inattention and hyperactivity might also occur in reaction to stress such as bullying at school, but these adjustment disorders usually respond to psychosocial intervention and do not require medication. Anxiety and oppositional-defiant disorder are common comorbid conditions which need to be considered in a management plan.

Conduct disorder and oppositional-defiant disorder

Disruptive, aggressive, oppositional, and antisocial behaviours are problems in about 30 per cent of young people with mild/borderline ID, particularly males.[2] These children usually have language and learning difficulties and are likely to have experienced inconsistent care and sociocultural deprivation. The diagnosis requires a consideration of both the developmental age and the context. For example a 12-year-old young person with moderate ID might steal from a shop on the demand of a classmate without having a sufficient understanding of social rules and the rights of others. These diagnoses are not applicable in non-verbal children with more severe ID where, for example, aggressive behaviour might be the only means of communicating that an experience is stressful.

(a) Tic disorders (Tourette's disorder) and stereotypic movement disorder

Tics are sudden rapid non-rhythmic recurrent motor movements or vocalizations in response to an irresistible urge that can be delayed.[7] There is a comorbid association with autism or disruptive behaviour. Tics may emerge or deteriorate in a child with ADHD treated with stimulant medication. Relatively small doses of haloperidol or pimozide are often an effective treatment.[16] Stereotypic movements are persistent, driven, non-functional, complex motor behaviours that are differentiated from tics because they appear to be intentional. They occur in 2–3 per cent of children with more severe ID. These behaviours, such as self-biting, can cause serious injury and significantly interfere with daily activities, for example by rocking. They may be self-stimulating, occurring when the child is unoccupied, or might have communicative intent, for example to avoid an activity. The production of endogenous opioids might act to maintain the behaviours.

Emotional disorders

(a) Anxiety

Clinically significant symptoms of anxiety affect 10–12 per cent of both boys and girls with ID, compared to a prevalence in normal children of 2–5 per cent affecting twice as many females as males.[2] Fears are a common symptom and in children with ID are likely to be similar to the simple fears of young children in general, such as fear of the dark, loud noises, insects, and animals. Separation anxiety, typically seen in young children, can persist in older children with ID and is often complicated by fears, for example of school or other children. Children with ID are at high risk of suffering stressful experiences such as being placed in care or suffering physical and sexual maltreatment and neglect and have a limited capacity to understand stressful experiences.[17] Therefore, they are vulnerable to develop post-traumatic stress disorder (PTSD). Symptoms of PTSD are usually manifest as disturbed behaviours seen typically in traumatized young children such as

repetitive play, behaviours which re-enact the trauma, nightmares, withdrawal, increased startle response, and hyper-vigilance.[7] PTSD in children with ID is underdiagnosed and research on its phenomenology is required. The diagnosis of obsessive-compulsive disorder in young people with ID is problematic because they may not have sufficient language to describe persistent thoughts and their attempts to suppress these thoughts. They also do not recognize that their compulsive behaviour is unreasonable. Stereotypic self-injurious behaviours might be regarded as evidence for an OCD, but these behaviours do not usually respond to treatments for anxiety. Some drugs that are an effective treatment of OCD in young people in the general community, such as sertraline and clomipramine, are used for the treatment of anxious compulsive behaviours in young people with ID but their efficacy has not been investigated.

(b) Mood disorder

Depression in young people with ID is more prevalent than in other children.[2] Adolescents with moderate to severe levels of ID, provided they have some language, are able to reliably report sad feelings, but the diagnosis is confirmed by the presence of behavioural symptoms such as irritability, loss of interest in usual activities, loss of appetite and weight loss, sleep disturbance, crying, and withdrawn and regressed behaviours (e.g. rocking).[7] A daily carer completed record of mood and activity may reveal a pattern of cycling bipolar or unipolar mood disorder and is a useful record to document response to treatment.[9]

Pervasive developmental disorders

About 75 per cent of children with autism have ID. There is also an increased association with epilepsy, tuberous sclerosis, congenital rubella syndrome, and phenylketonuria and comorbidity with ADHD, OCD, anxiety, depression, and tic disorder. These comorbid symptoms might be epiphenomena of autism related to fronto-striatal dysfunction, but it is helpful to the child and family to identify any comorbid symptoms and to treat them appropriately.[18] Children with ID also have delayed language and may have stereotypic behaviours and a limited range of interests, but they can be differentiated from those who also have autism because they try to communicate, use gesture and imitation, have reciprocal play and respond with emotion in a manner appropriate to their developmental level.

Principles of management

Effective management begins with a multiaxial diagnostic formulation based on the DSM or ICD which describes the child's psychopathological disorder, cognitive profile, temperamental characteristics, genetic and associated medical conditions, level of adaptive functioning, and the family and sociocultural context. The delivery of effective management usually requires the involvement of a multidisciplinary team with a clear definition of roles and regular communication between professionals, parents, and teachers, for example at a case conference. The involvement of parents as partners in speech, physio, and behaviour therapy improves outcome and facilitates treatment compliance. Parent education and skills training reduces parental stress and improves parental mental health and child behaviour.[19] Family therapy may help reduce family conflict and improve communication and child management, but outcome research is required.

Psychological treatment

Effective behaviour modification techniques based on operant conditioning principles teach positive socially adaptive behaviours and reduce difficult behaviours.[20,21] Antecedent triggers, the functions of the behaviour and any rewarding consequences which reinforce the behaviour are identified. Intervention might focus on changing the antecedent events, for example by removal of an upsetting sound. Replacement of the disruptive behaviour might be achieved by rewarding alternative appropriate behaviour or by teaching a new behaviour which improves communication such as the use of pictures to communicate a need. A further approach which may help extinguish difficult behaviour is a non-aversive modification of the usual response to the behaviour such as moving away from a screaming child instead of paying them attention. Counselling and cognitive behavioural therapy including relaxation exercises, which is modified to take into account the developmental level and language ability of the child with ID might reduce anxiety and depression, but its effectiveness requires further research.

Pharmacotherapy

Drugs, if used to treat specific symptoms, should be part of a psychosocial and educational management plan, which includes informed parent/carer consent and participation. Evidence for the efficacy of psychotropic drugs in children with ID is limited,[16] but children with ID are prescribed or even overprescribed drugs on the basis of evidence for their use in either adults with ID or in normally developing children. These drugs include stimulant medication, atomoxetine, clonidine, neuroleptic drugs, and imipramine for ADHD; clomipramine and selective serotonin reuptake inhibitors (SSRIs) for OCD and stereotypic self-injurious behaviour; SSRIs for depression; neuroleptic drugs for tic disorders. Rigorous empirical studies have failed to demonstrate clear benefit for the use of 'typical' neuroleptic medication such as chlorpromazine in the treatment of disruptive behaviour. There is empirical evidence that low doses of haloperidol or risperidone (0.02–0.06 mg/kg/day) are an effective treatment for disruptive stereotypic behaviour.[22] Problematic side effects, particularly for haloperidol, are drowsiness, akathisia, dystonia, and tardive dyskinesia. Weight gain, prolactinemia, and metabolic disturbances such as diabetes are serious side effects of risperidone. Anticonvulsants used as mood stabilizers (sodium valproate, carbamazapine, and lamotrigine), lithium, β-blockers, and buspirone have been shown, mostly in open trials, to reduce self-injurious and episodic aggressive behaviours. Opiate antagonists (naloxone and naltrexone) may reduce self-injurious behaviour. Difficult sexual behaviour in adolescent males with ID can be treated with testosterone antagonists to reduce libido. In many countries this treatment requires approval and monitoring by an independent committee. The prescription of psychotropic drugs requires regular follow-up to monitor compliance, side effects, and response to treatment using behaviour observations and carer completed symptom checklist such as the DBC[9] or NCBRF.[8]

Early intervention

There is growing evidence that broad-based early intervention for young children with developmental delay, such as those with

autism, promotes adaptive behaviour and skill development. The components of effective early intervention include:

1 Parent education and skills training and the management of parent mental health problems such as depression.[19]

2 Regular medical review and treatment of associated conditions such as epilepsy and any inter-current illness or psychopathological disorder, which might compromise behaviour.[10]

3 A structured behaviour management and social and communication skills programme.[21]

4 Family support, respite care, home help, and holiday programmes.

5 Speech, occupational, and physiotherapy to develop communication, sensory, motor, and play skills.

6 Assisted education and socialization at preschool and school.

Conclusions

Children with ID often suffer the added handicap of emotional and behavioural disorder which seriously compromises their adjustment and causes significant extra burden and cost for their parents and the community. A comprehensive biopsychosocial assessment of the child and family provides the context for understanding psychopathological symptoms and the basis for a best practice management plan incorporating psychological, educational, family, and perhaps pharmacological interventions.

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10.5.2 Psychiatric and behaviour disorders among adult persons with intellectual disability

Anton Došen

Introduction

The behavioural and emotional difficulties that are experienced by adults with intellectual disability (ID) have been regarded for many years as manifestations of their intellectual deficits and maladaptive learning. The awareness that these persons may also suffer from mental illness was a notion that came into being in the mid-nineteenth century.

Nevertheless, the psychiatric problems of individuals with ID were continually ignored in the first half of the twentieth century. In the past three decades, the flourishing normalization philosophy has highlighted the psychiatric problems of this population once again and rekindled the interest of practitioners, scientists, and service providers. Systematic studies have been performed which indicate that the full spectrum of psychiatric disorders as we know them today can be identified among the persons with ID. Moreover, it is probable that they may be prone to a psychopathology that is determined by the specifics of their biological and psychosocial being.

Clinical features

Features affecting presentation

Studies indicate that the types of psychiatric symptoms and syndromes that are observed among persons with borderline and mild ID are similar to those encountered among the population in general. Amongst individuals with moderate and severe ID, however, the presentation of mental illness may be less typical, and the diagnosis more difficult to establish.

Sovner and Hurley[1] have categorized four factors which may influence the presentation of mental illness among the persons with ID: intellectual distortion (impaired ability to conceptualize feelings and to communicate them to others), psychological masking (lack of usual richness of the symptomatology found in general population), cognitive disintegration (inclination to become disorganized and to exhibit regressive behaviour), and baseline exaggeration (increase of pre-existing maladaptive behaviour by emotional stress or mental illness).

Hucker *et al.*[2] pointed to a generally banal symptomatology encountered among these individuals, often accompanied by regression to a child-like state of dependency and hysterical features. Behavioural disturbances were often more important than symptomatic complaints as indicators of psychiatric disorders.

Apparently, the lower the IQ, the more the symptoms of mental illness tend to lose their specificity, or take on a different meaning than is the case with the intellectually normal population. This, undoubtedly, makes it difficult to establish a confident diagnosis of mental illness in people with severe ID.[3]

The following is a concise survey of the striking clinical features of mental illness and behaviour problems as they occur among adult persons with ID.

Mental illness

(a) Psychosis

Among persons with mild ID, classical clinical features are present in psychotic states. The symptoms tend to be florid but banal. In schizophrenia, for example, there is a high incidence of delusions and hallucinations, which reflect the limited experiences, naive and wishful thinking, interests, and social horizon of the patient. Ideas, that have been influenced by radio, television, etc., are found frequently. Catatonic features with odd postures and slowness are common. Impulsive, aggressive, auto-aggressive, and bizarre behaviours may dominate the clinical picture. In the chronic-phase apathy, lack of motivation and social withdrawal are common.[4,5] Increase of 'negative' schizophrenic symptoms and decrease of functional abilities were observed in the group with ID when compared with the group from general population with schizophrenia.[6]

Establishing the diagnosis of schizophrenia in persons with more severe ID can be a difficult task because of verbal communicative difficulties. Reid[7] considers it to be impossible to establish such a diagnosis among persons who only communicate non-verbally. However, the problem of establishing the diagnosis does not mean that psychotic conditions do not occur in these individuals. To the contrary, it is likely that different sorts of psychosis occur in this population more frequently than in general population (see Chapter 4.5.2).

Short-term psychotic states (lasting several days or weeks), usually beginning rather suddenly after a stressful event, are found relatively often among adolescents and young adults with ID. Early Dutch psychiatric literature refers to these states as 'debility psychosis'. The symptoms may be heterogeneous, and remission is usually complete. These persons usually revert fully to the premorbid level of functioning. However, recurrence is frequent.

Establishing the diagnosis of atypical psychosis (contrary to diagnosing schizophrenia) is, for an experienced practitioner, possible even with patients who have no language development and are at a severe level of ID. The primary symptoms are changes in interactional patterns, changes of posture and movement, odd and bizarre behaviour, disturbances of the physiological functions, expression of emotional tension (e.g. anxiety, irritability), aggression, and self-injuring behaviour.

(b) Major depression and bipolar disorders

Depressed mood and vegetative symptoms are the most striking symptoms, even though complaints of depression are not always expressed. A depressive mood often is not verbalized, particularly among individuals at a lower intelligence level, but may well be observable. Similarly, the elevation of mood in mania is usually not expressed verbally either. Atypical features such as regression to child-like dependency, incontinence, loss of social skills, and hysterical symptoms such as pseudo-fits and paralysis may mask classical symptomatology. In persons with a more severe disability, depression should be suspected where there is a change or onset of behaviour problems like stereotypic behaviour, tantrums, aggression, and self-injuring behaviour.[4,8] Catatonic features and visual hallucinations, particularly among persons at lower intelligence levels, have also been reported.[2,9,10] The atypical symptomatology among persons on lower developmental levels may require modification of standard diagnostic criteria.

Aggressive behaviour was observed in 40 per cent of the depressed subjects.[11] Self-injuring behaviour has often been reported as well.

Suicidal behaviour has hardly ever been studied in this population, and suicidality is very rare among the more severely handicapped. This symptom is, however, not rare among depressive patients at a mild level of ID.

Some investigators report the relatively frequent occurrence of rapid-cycling affective disorder,[12] particularly in persons with more severe disability. Episodes of particular mood or of an undifferentiated mood-like dysphoria or irritability have a short duration, and may be expressed in terms of days or weeks.[9] Researchers assume that these disorders, in persons with ID are often related to organic brain disorders, that is, metabolic, neuroendocrine, and other neurological disorders.

In mixed bipolar disorder, there is either the simultaneous presence of manic and depressive features, or these features follow each other rapidly. Schizoaffective psychoses are also described among these individuals.

(c) Dysthymic disorder

Dysthymia is a relatively common disorder among persons with mild and moderate ID.[9,13] Nevertheless, publications on this disorder are rare. The symptomatology includes loss of energy and interest, negative self-image, feelings of helplessness, anxiety, and significant behavioural problems such as irritability, anger, destructibility, and aggression. The disorder is often related to a specific stress, for example, termination of an affective relationship, change in the surroundings, hospitalization, etc. Chronic states, dating back to the childhood or the teens, possibly caused by chronic overdemanding, social deprivation, or repeated abuse, may be interrupted by episodes of major depression, usually elicited by acute stress (so-called 'double depression', see Chapter 4.5.3). Došen and co-workers found this disorder relatively frequent in adolescents and young adults with ID and called it 'developmental depression'.[10, 14] Social interactional problems, poor social skills, and difficulties related to emotional development are considered to be predisposing factors for this disorder.[14]

(d) Anxiety disorders

The most commonly reported anxiety disorders are simple phobia, social phobia, and generalized anxiety disorder.[4] It seems that adults with ID have fears similar to those of children who are at the same mental age: fear of separation, fear of natural events, fear of injury, and fear of animals. The anxieties and fears are probably related to the traumatic events and cumulative failure experiences that these persons have. The presentation may be through behaviour problems, irritability, problems with sleeping, or somatic complaints.[5] In a panic disorder, a sudden onset, blackouts, aggression, sweating, and shaking may be observable. The obsessive–compulsive disorder may be difficult to diagnose in persons with ID because they do not resist against such feelings and the anxiety is often absent. According to some authors,[4] the diagnosis can be established with the emphasis being on the externally observable behavioural components, despite of absence of some internal states like anxiety and resistance. Post-traumatic stress disorder is likely to occur in this population, following relatively less severe stress than among general population. The diagnosis in those who are unable to communicate their experiences should be based on changes in a person's behaviour, mood, and level of functioning following a traumatic event.[4]

(e) Autism spectrum disorder

Autism spectrum disorders have been estimated to be present in 10 per cent of persons with mild ID and 40 per cent of those with severe ID, and account for a large proportion of behaviour disorders. It also appears that mental illness occurs frequently as a secondary disorder among these individuals.

A possible relationship between affective illness and pervasive developmental disorder has been suggested by various investigators[12]; however, this phenomenon has been examined insufficiently and is clearly an area that future research can be directed to. In clinical practice, we have encountered a number of cases of pervasive developmental disorder together with secondary atypical psychosis. Inexperienced practitioners are inclined to diagnose schizophrenia in such cases. However, thorough developmental history will reveal sufficient information to make diagnostic differentiation possible. Other problematic behaviours such as anxious, aggressive, auto-aggressive, or disruptive behaviour are frequently found among persons who have an autism spectrum disorder and ID.[15] In our opinion, these behaviours should be seen as being secondary disorders instead of as part of the autistic disorder.

(f) Dementia

In individuals with dementia, the typical features such as memory impairment, personality change, loss of social skills, and deterioration in habits are always present. Behavioural problems may be the most obvious manifestation. Nocturnal confusion, transient psychotic episodes, and late-onset epilepsy should always alert one to the possibility of a dementing illness in the ageing person with ID. Memory loss is generally difficult to identify in the early stages, but becomes more obvious as the illness progresses. Medical risk factors include a history of hypertension, ischaemic episodes, neurological symptoms, organic brain damage, and a family history of dementia. Dementia Alzheimer type in persons with Down syndrome presents a similar picture and is usually associated with generalized premature ageing.

Behaviour disorders—challenging behaviour

Behaviour disorders including aggression, self-injury, destructiveness, and disruptive, maladaptive, and antisocial behaviour occur commonly among persons with ID. Such behaviour has recently been called challenging behaviour, which emphasizes the need for appropriate care and supervision. These disorders are usually associated with severe ID, but can also occur in individuals who are at a moderate and mild level of ID.

Various attempts have been made to distinguish between behaviour disorders and psychiatric illness in these individuals. Gardner and co-workers[16] have proposed a bio-psycho-social diagnostic approach, which takes account of the multiple factors underlying and maintaining the behaviour disturbances of a particular individual. They point out that behaviour disorders with a neuropsychiatric and organic basis can still acquire a functional component if they are being reinforced by the environment or are of value to the individual. Another approach is from the developmental perspective,[3,17] viewing behaviour disorders as the result of a lack of real understanding of the person's developmental aspects and interactional problems.

(a) Aggressive behaviour

Aggressive behaviour is a common problem among persons with ID. The symptom of aggression is often a feature of the psychosis, depression, or antisocial personality disorder, and is often described in genetic disorders such as the fragile X, Prader–Willi, and

Klinefelter syndromes. Learned aggression through the imitation of aggressive models or as a function of communication is also found relatively frequently among people with ID.

(b) Self-injurious behaviour

Self-injurious behaviour occurs more often among persons with moderate and severe ID (IQ < 50), beginning sometimes in toddler age and most frequently between the ages of 10 and 20. The occurrence of self-injurious behaviour is related to genetic and organic disturbances and adverse environmental and development conditions. Certain psychiatric disorders such as depression and psychosis may also elicit self-injurious behaviour.

(c) Offending behaviour

Owing to their behaviour problems, these individuals may become involved in activities, that bring them into conflict with the law. Insufficient understanding of their problems and needs may result in their not receiving the appropriate support from the social services. The typical offender with ID is, according to Day,[18] a young male functioning in the mild to borderline intellectual range, from a poor urban environment, with a history of psychosocial deprivation, behaviour problems, and personality disorder. The most common offences are acquisitive and technical, but sex offences and arson are considerably overrepresented.

Personality disorders

Various investigators have reported personality disorders among persons with ID.[19,20] The relevance of the concept of personality disorder, in particular with regard to the persons with more severe ID, has been questioned by a number of investigators. Apparently, in these persons, besides the problem of personality disorders, there is a problem of personality development. Zigler and colleagues[21] have explored personality traits thought to be particularly salient in determining the behaviour of individuals with ID. Levitas and Gilson[22] have stressed the importance of a crisis period during the process of personality development and the related psychosocial aspects. Other developmentally oriented authors[14,23] make a link between, on the one hand, the problematic processing of particular phase–specific aspects of emotional development and ego structuring, such as the achievement of secure attachment, an intercompetitive separation-individuation process, and the establishing of ego functions, and, on the other hand, the increased vulnerability of these individuals to particular psychiatric disorders such as depression, social withdrawal, disruptive behaviours, etc. Classification of personality problems within the existing diagnostic categories for personality disorders in general population is questionable and requires modification of particular diagnostic criteria.[4, 5] It is unlikely that personality disorders could be diagnosed in persons with severe/profound ID.

It appears that the main problem at the root of the personality disorders of individuals with ID pertains to an underdeveloped personality structure in relation to a delay in psychosocial development. One is then inclined to speak of an immature rather than a disturbed personality.

Diagnosis and classification

Diagnosis calls for a full and detailed history, careful observation of the patient, knowledge of the natural history of the illness, and the elimination of irrelevant factors (for further information about assessment, see Chapter 10.1).

Assessment

In complex cases, a period of inpatient observation or explorative treatment may be necessary. As full a history as possible of the current illness should be obtained from the patient, together with corroborative histories from the relatives and carers. The standardized format of psychiatric history should be supplemented by a detailed developmental history, a description of current social functioning, environmental circumstances, associated somatic disorders and physical disabilities, and the aetiology of the ID. Enquiries should be focused on behavioural changes such as sleep disturbance, loss of appetite, weight loss, lack of interest, bizarre behaviour, restlessness, anxiety, withdrawal, and any other deviations from customary behaviour. Precipitating factors such as stressful events and possible predispositions to reacting in a particular way in a particular situation should be explored. Full details of previous psychiatric illnesses suffered by the patient and the family history of mental illness should be obtained. Because of the general paucity of subjective complaints by persons with ID, the examiner must rely more on objective data regarding the patient's appearance, manner of communication, facial expression, evidence of hallucinations, posture, etc. If called for, direct observations should be made in as wide a range of settings as is necessary. To these ends, a video recording of the patient in his or her natural surroundings may provide important information about the interactional pattern of the patient and his or her surroundings. The psychiatric examination is usually supplemented by somatic, neurological, neurophysiological, biochemical, and psychological examinations. Assessment of the level of ID is crucial for diagnostic consideration. Currently, in psychological assessment, besides examination of cognitive functioning, the emphasis is on determination of personality development and the level of emotional development.[3]

Diagnosis

IQ assessment, personality development, emotional level, and measuring adaptive behaviour can provide extra background information that may be useful to the diagnosis. Specific tests, for example, of thought disorder in schizophrenia, may be helpful in establishing the diagnosis, but are not yet standardized for this population. Non-invasive neuroimaging techniques promise to be potentionally valuable diagnostic tools, particularly for non-verbal persons with severe ID in the future. Structured interview schedules and rating scales are being used increasingly in an attempt to improve diagnostic accuracy. Instruments developed for use with persons without ID rely heavily on the ability of the patient to describe subjective feelings and are thus of limited value. Diagnostic rating scales that are to be used with the persons with ID should, as far as possible, reflect behavioural rather than subjective components. An early attempt was made by Hucker and colleagues,[2] who published diagnostic criteria for mania, depression, and schizophrenia for use with persons with ID; these have been further refined by Sovner and Hurley[1] and by Menolascino and Weiler.[24] Recently, a number of scales have been developed specifically for use with this population, and different other instruments are in development. These scales were primarily developed for use as research instruments, and whilst they play an invaluable role in epidemiological studies and population screening as well as being useful for monitoring the response to treatment, they are of limited value in clinical practice and rarely, if ever, solve a diagnostic problem.

(a) Integrative psychiatric diagnosis

For clinical purposes, it has been proposed that more elaborate and expanded diagnostic formulations be made.[25] Such diagnosis should incorporate diagnostic categories as well as the onset mechanisms of the psychopathology, biological aspects, psychological functioning, milieu characteristics, life problems, psychosocial needs, and individual strengths. Diagnostics of this sort have been called integrative diagnosis and are an attempt to adapt conventional diagnostic criteria to the complex problems of individuals with ID.[3,14] During the diagnostic process, particular attention is paid to describing the onset mechanism of the psychopathological phenomenon by which the dynamics of different nosological factors come into scope and become more understandable to the direct carers and professional helpers. A better understanding of the processes involved in the mental illness is important to the treatment approach.

Classification

Numerous attempts have been made to apply the traditional psychiatric diagnostic categories of ICD-10 and DSM-IV to the psychopathology of persons with ID. Their applicability to these individuals has, however, been questioned.[1,2,25] Whilst the ICD and DSM criteria may be applied to people functioning in the mild to borderline ID ranges without alteration or with little modification, they become increasingly unreliable as the severity of intellectual disability increases. The limited communication skills of these persons make it very difficult to ascertain the presence of certain symptoms such as delusions and hallucinations. As the role of underlying organic brain damage expands, the phenomenology becomes increasingly more characterized by a range of atypical symptoms. The non-specific nature of behavioural disturbances further confounds diagnostic endeavours. Szymanski,[25] among others, has pointed out that behaviour disturbance is not a psychiatric condition but a symptom, and Reid[26] has drawn attention to the fact that behaviours, that would be deemed abnormal in people functioning in the average intellectual range may be developmentally appropriate to the mental age of a person with severe ID.

Other authors[3,17,27] suggest using the developmental perspective when attempting to understand and diagnose the psychiatric and behavioural disorders of the persons with ID. They point out that there are findings which suggest that there may be a relationship between certain developmental levels and syndromes and specific neuropsychiatric disorders (see below). Some investigators argue that syndromes should be empirically derived, and a 'dimensional' approach would be a more effective way to classify psychopathology than the categorical system.[28,29] Van Praag[30] proposes a 'functional psychopathology model' emphasizing functional problems of the CNS on the background of psychiatric disorders.

Not surprisingly, there have been calls for some modifications of existing DSM and ICD diagnostic criteria with the introduction of behavioural equivalents for some symptom criteria as well as for development of a broader taxonomy, which takes account of the atypical presentation of mental illness in this population.[31]

Epidemiology

Overall prevalence

Psychiatric disorder appears to be more common among the persons with ID than in the general population. Overall prevalence rates range from 20 per cent to 74 per cent,[19,25,32] depending on the diagnostic criteria employed, the type of disorder screened (whether or not behaviour disorders are included, for example), the nature of the sample (community or institution), the type of data collected (case note studies or new data), and the level of ID, ages, and gender of the populations studied. Higher rates of psychiatric disorder have been reported in some studies among the individuals with severe ID in comparison with the person on mild ID level. For further information concerning epidemiology, see Chapter 10.2.

Aetiology

Investigators of aetiology agree that the high prevalence of psychiatric disorders among persons with ID is related to a wide range of neurological, psychological, social, and personality risk factors including impaired genetic factors, delayed cognitive, emotional and social development, organic brain damage, communication problems, environmental problems, and family psychopathology. Alone or in combination, these factors increase the vulnerability of the person with ID to psychiatric and behavioural problems.

Theories

Achenbach and Zigler[33] have pointed out the importance of social incompetence as a factor playing a role in interactional and intrapsychic problems. This theoretical perspective has received support from studies in which discrepancies between self-image and the expectations of others have been shown to be a fertile breeding ground for the onset of psychopathology. Menolascino[34] emphasized the importance of neurophysiological and sociological developmental processes which may have a different timing and take a different course in these individuals, causing deviations from normal development; this is known as the biodevelopmental theory. Matson[35] proposed the biosocial theory, which hypothesizes that due to specific biological factors (neurological, biochemical, genetic, etc.) together with specific social factors (family interactions, culture, and other environmental variables), and specific psychological processes (cognitive development, personality variables), the psychopathology of persons with ID differs in a number of ways from that of persons without ID.

Tanguay,[36] Došen,[3,14,17] and Gaedt and Gärtner[37] have applied the developmental approach to the understanding of the symptoms of psychopathology and to the psychiatric diagnostics in these individuals. These authors based their approach on Piaget's stages of cognitive development as well as on Mahler's and Bowlby's models of psychosocial development. Parallels could be drawn between the symptoms of psychopathology of children without ID at a particular chronological age and individuals with ID at the same developmental age, which indicates that the developmental level may specifically affect the exterior features of mental illness. According to these authors, although developmentally disabled adults who suffer from mental illness resemble other adults in many ways, it is the developmental level and consequent differentiation of psychosocial life (e.g. affect differentiation, personality structuring, moral development), that may be decisive for the symptoms linked to the disorder. For example, because of their underdeveloped psychosocial life, adults with severe ID display little differentiation in their symptomatology. They exhibit agitation, aggression, self-injurious behaviour, or other disruptive behaviours, which in other people may be indicative of various

underlying mental illnesses such as anxiety disorder, depression, or psychosis. Similarly, a toddler may have 'tantrums' when frustrated, anxious, or distressed by somatic pain or separation. Recently, problems of attachment development in these individuals and their vulnerability to stress have been related by some authors to the behaviour problems and psychopathological features.[38,39]

Behavioural phenotypes in relation to mental illness

Recently, the concept of a behavioural phenotype has been introduced as an attempt to assess the interrelationship between specific behaviours and genetic disorders (see Chapter 10.4). In addition to specific behaviours, an increased prevalence of psychiatric syndromes have been reported in association with particular genetic and other syndromes. The possible link between a behavioural phenotype and the particular psychiatric disorder of a person with ID is a highly challenging issue for investigators researching this field. For further information.

Down syndrome has always been associated with specific patterns of language, and cognitive and social development. In most studies, these persons were found to exhibit muted affect and have deficient language development, and yet they showed particular strengths in socialization. The tendency for such individuals to develop Alzheimer dementia has often been described. The occurrence of affective disorder among Down syndrome subjects had attracted the attention of scientists recently.[40,41] Diurnal mood variations, speech reduction, and an increase in aggression are the commonly reported symptoms, which are indicative of depression among persons with Down syndrome. The onset of mania in this syndrome has been disputed. However, cases of mania have been reported later in life among those having Down syndrome.

Persons with fragile X syndrome have often been described as having autistic-like social impairments. However, among these individuals, social anxiety is more characteristic than social indifference.[42] In addition, attention deficit, stereotyped behaviour, self-injury, and hyperactivity are common.

In Prader–Willi syndrome, hyperphagia and obsessive–compulsive and aggressive behaviour are common. The occurrence of brief psychotic episodes with heterogeneous symptoms have been described among these persons.

Different behavioural phenotypes and psychiatric disorders have also been described for other syndromes. For example, anxiety disorder, sleep disorder, and hyperactivity are common in Williams syndrome, depression is often found in Klinefelter's syndrome, and self-injurious behaviour occurs regularly in Cornelia de Lange syndrome.

Genetic and environmental factors and onset mechanisms

It should be stressed, however, that the very same behaviours may be found among individuals with genetic as well as with idiopathic ID, which probably means that these behaviours are not only the product of genetic factors, and that other factors may be the cause as well. Among other things, the process of psychosocial development and interactional patterns with the surroundings can affect the onset of a particular behaviour. Apparently, genetic characteristics can influence the psychosocial development, which then can be decisive for the onset of particular interactional patterns and particular behaviours (so called 'heightened probability', according to Dykens *et al.*[43]). The question is: Do these behaviours play a role in the onset of the aforementioned psychiatric disorders associated with these genetically based syndromes, and if so, how do they do this? Or, does a genetic disorder have a more direct role in the onset of a particular psychiatric disorder?

For a better understanding of how specific psychiatric disorders evolve among these individuals, it is necessary to make a scheme of the onset mechanisms involved in each psychiatric disorder. It is important to take account of the genetic disorder, the developmental processes, and the interaction patterns with the surroundings in which particular behaviours may play an important role. If one takes all these factors into consideration, it may be expected that in certain cases particular psychiatric disorders have a specific aetiology. An example is self-injurious behaviour, which is currently considered to be a 'challenging' behaviour, but, according to various professionals in this field, warrants being seen as a specific psychopathological phenomenon. However, there are also examples in which the connection between genetic and psychiatric disorder appears to be more direct. An example is the velocardiofacial or Shprintzen syndrome.[44,45]

Psychiatric problems in older persons with ID

In elderly persons with ID, besides dementia, other psychiatric disorders, like psychotic conditions, depression, and anxiety disorders, are common.[46] In a mixed community and institutionalized group of persons with ID older than 50 years, Patel and colleagues[47] found a prevalence of psychiatric disorders, mainly depression and anxiety in 11.4 per cent of the population. Other authors[48] ascertained that major psychiatric disorders could be found in 20 per cent of persons with ID aged 65 and above. Behavioural symptoms such as aggression and self-injury frequently accompany psychiatric problems among these individuals. Causes for frequent psychiatric problems in this population group are various; like changes in social milieu and living pattern, loss of beloved ones, social deprivation, and inactivation done to retirement from the work or previous activities and medical, in particular neurological, conditions at this age (see Chapter 9.2.1). Frequently, psychiatric disorders in these individuals can be found as an accompaniment of a dementia process. Differentiation between the beginning stage of dementia and other psychiatric disorders can be a difficult task.[49] In these cases, a total bio-psycho-social picture of the involved person, including a detailed patient history, as well as an insight into the surroundings circumstances and in interactions is necessary for appropriate understanding of the patient's symptoms. Use of some assessment instruments can be helpful (see Chapter 10.1).

Management

In principle, the diagnosis of psychiatric disorders in persons with mild ID and good verbal skills does not differ much from diagnosing these disorders in persons with average cognitive skills.[50] However, in persons on a lower level, the recognition of mental disorders can be difficult, partly because of linguistic limitations and partly because of difficulty in distinguishing between behaviour problems (challenging behaviour) and symptoms of psychiatric illness. Communication problems often ask for management through another individual ('management by proxy'), which means

that carers are relied upon to give description of a person's behaviour. The diagnostician should know that the reliability and validity of such information may be problematical and, besides this information, should search for more objective sources, like information through non-verbal communication with the patient and through observation of the patient in different situations and interactions. Distinguishing between behaviour problems and psychiatric illness is a very important issue for adequate aid. It asks for a multi-disciplinary assessment approach in which experienced (specialized) professionals from different disciplines, participate forming together a multi-disciplinary team. These teams have expertise in both intellectual disability and mental health, and provide direct services to patients and carers. Through the assessment of a specialist team, an insight is being obtained whether presenting behaviours of involved individual are the results of an organic condition, a psychiatric disorder, environmental influences, or a combination of these factors. The clinical symptoms must be viewed in a broad context of a patient's functioning, considering his/her deficits, strengths, and relevant bio-psycho-social factors. For example, a disruptive behaviour can be a symptom of a psychiatric disorder in one person, whilst in another person, and in other environmental circumstances, it may be a means of communication, serving as a vehicle for obtaining a caregiver's attention or avoiding an unwanted task. Delineation of a subtype of an established psychiatric disorder asks for the specialist knowledge of the diagnostician.

The specialist teams should be based locally, providing inpatient care as well as outpatient and community-based interventions (see Chapter 10.9). Involvement of parents and carers as partners in the management plan can have more advantages and is being recommended.

The diagnostic evaluation may require significantly more time than the evaluation of persons of normal cognition. An accurate diagnosis which integrates all assessment results of the multidisciplinary team members should serve as a starting point for the treatment.[51]

Conclusion

Persons with ID are more prone to psychiatric disorders and behavioural problems than the general population. The symptoms of their biological disorders, developmental processes, and interaction patterns may give atypical clinical pictures, particularly in individuals with moderate and severe ID. It is probable that certain disorders are specifically associated with or even unique to the ID. Traditional nosological classifications do not adequately accommodate the phenomenology of psychiatric disorders in this population. A broader taxonomy, that takes account of the atypical presentation of psychopathology in this population is necessary.

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10.5.3 Epilepsy and epilepsy-related behaviour disorders among people with intellectual disability

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Epilepsy is defined as at least one epileptic seizure; this in practice means two or more epileptic seizures unprovoked by any immediate identifiable cause during a relatively short period of time. Epileptic seizure is a clinical manifestation presumed to result from an abnormal and excessive discharge of a set of neurones in the

brain. An epileptic syndrome is a cluster of symptoms and signs including type of seizure, mode of seizure recurrence, neurological findings, and neuroradiological or other findings of special investigations, customarily occurring together. An epileptic syndrome can have more than one cause or the cause may remain unknown; consequently outcomes may be different. Pseudoseizure is used to denote epilepsy-like seizures without concomitant EEG changes.

Epilepsy and intellectual disability are symptoms of brain origin. The former is an unstable condition, where during the seizure or ictally the behaviour of a person with epilepsy is abnormal, but between the seizures or interictally there is no affect of epilepsy on his or her behaviour. Intellectual disability is a more or less stable condition. However, the categories of the degrees of intellectual disability are neither absolute nor static, as some children may move up or down between them.

This chapter deals with the diagnosis, manifestations, behavioural disorders, frequency, aetiology, treatment, effects of antiepileptic drugs on behaviour, and prognosis of epilepsy in people with intellectual disability.

Diagnosis and differential diagnosis of epilepsy

The diagnosis of epilepsy is clinical and requires the collection of historical data, physical and mental examination, EEG, and laboratory tests such as determinations of blood glucose and electrolytes.[1] In babies with epilepsy, attention should be paid to changes of skin colour or cardiac rhythm, sucking and smacking, which all may be epileptic phenomena. In people with intellectual disability it may be difficult or impossible to obtain an accurate clinical history from the patient. The clinician often has to depend on relatives or other professionals involved in the care of the patient. In addition to a description of the seizures, a history of the age at onset of epilepsy and the complete clinical picture of the epileptic syndrome are of value.

General factors that provoke seizures include fever, infection, hypoglycaemia, stress, excessive waking, alcohol withdrawal, hyperventilation, some medications, sudden discontinuation of sedative drugs, and specific activity. Fevers associated with infections such as those of the ears, sinuses, upper respiratory tract, or urinary tract are quite common. If seizures are exacerbated, ensure that any treatable infection is identified. The situation is further confused by the fact that seizures can produce the fever, which, however, resolves within an hour. Withdrawal seizures may be precipitated by a sudden discontinuation of drugs such as benzodiazepines which have an antiepileptic effect, although they may have been prescribed for another reason. Some seizures may be associated with a specific activity, especially if this activity induces excitement or anxiety. Exercise-induced seizures occur regularly in some patients.

Many people with intellectual disability have abnormal behaviour that resembles epileptic seizures but is not epileptic in origin. The diagnostic and other problems caused by non-epileptic seizures or pseudoseizures are well known.[2] Different dyskinesias, psychogenic attacks, and other non-epileptic episodes may be manifested as pseudoseizures at different ages (Table 10.5.3.1). Sudden aggression and other epilepsy-like conditions (Table 10.5.3.2) are in practice the most important reasons for the overdiagnosis of epilepsy, and consequently also for overmedication and subsequent intoxication in patients with intellectual disability. On the other hand, non-convulsive epileptic phenomena and even partial seizures (Table 10.5.3.3) may be difficult to diagnose in people with intellectual disability. The situation is more complicated when patients with intractable epilepsy have both real epileptic seizures and pseudoseizures, for example psychogenic seizures. In such cases the recognition of psychogenic seizures[3] (Table 10.5.3.4) helps to identify appropriate treatment.[4]

Table 10.5.3.1 Salient non-epileptic episodes at different ages

Age	Disorder
0–2 months	Tremor Dyskinesias associated with bronchopulmonar dysplasia Benign myoclonus during sleep Apnoea
2–18 months	Paroxysmal torticollins Ipsoclonus myoclonus syndrome Intestinal obstruction Breath-holding spells Jactatio capitis Masturbation Paroxysmal choreoathetosis Gastro-oesophageal reflux
1.5–5 years	Pavor nocturnus Benign paroxysmal vertigo Nodding puppet syndrome Enuresis nocturnus Confusion with fever Familial dystonic choreoathetosis
5–12 years	Tic Complicated migraine Attention disturbance Sleepwalking Paroxysmalis choreoathetosis
>12 years	Vertebrobasilar migraine Syncope Hyperventilation syndrome Obstructive sleep apnoea Psychogenic attacks Raving fits

Table 10.5.3.2 Conditions often misdiagnosed as epilepsy in subjects with intellectual disability

Sudden aggression
Self-abuse
Bizarre behaviour
Abnormal motor activity
Staring
Eye-blinking
Nystagmus
Exaggerated startle
Intermittent lethargy

Table 10.5.3.3 Underdiagnosis of epilepsy in subjects with intellectual disability

Absence seizures
Non-convulsive status epilepticus
Seizures with periodic headache
Seizures with vertigo
Seizures with paraesthesia
Seizures with visceral and vegetative disturbances
Loss of emotional control
Postictal effects
Simple partial seizures
Complex partial seizures

Using magnetic resonance imaging (**MRI**), it is possible to identify structural brain abnormalities, including neoplasms, dysplasia, heterotopia, or diseases in the brainstem and/or posterior fossa. If MRI is not available, CT is recommended.

Prolonged video-EEG monitoring of the patients is of use in selecting candidates for epilepsy surgery or in distinguishing between epileptic and non-epileptic seizures. Basically, this enables any behaviour to be analysed in relation to the EEG changes. If this investigation is not available, portable cassette recording of the EEG may also be of considerable value. The diagnosis of subclinical seizures, including minimal behavioural or cognitive changes in the absence of any obvious clinical seizures, can be demonstrated as lengthened reaction times during EEG discharges in the Romny test.

The brain function of people with epileptic seizures and syndromes can be examined by interictal and ictal single-photon emission CT, positron emission tomography, functional MRI, magnetic resonance spectroscopy, and magnetoencephalography together with simultaneous EEG. Such investigations can help to define the epileptogenic brain lesion and thereby guide management including decisions about epilepsy surgery.

Epilepsy and epileptic syndromes at different ages

The main categories in the classification of seizures and epilepsy are primary generalized seizures, focal seizures, and secondary generalized seizures[5] (Table 10.5.3.5). The semiologic seizure classification[6] seeks to provide common descriptive terms for typical ictal symptoms and for seizure evolution (Table 10.5.3.6). Epileptic syndromes[7] are quite frequent in people with intellectual disability ranging from early infancy through childhood to adolescence (Table 10.5.3.7). As understanding of the pathophysiologic and anatomic substrates of epileptic seizures, syndromes and disorders increases, these classifications may need to be reappraised. [8–10]

Infancy

Infants with **early infantile epileptic encephalopathy** or Ohtahara syndrome seem initially neurologically normal, but soon develop increasingly frequent seizures with tonic spasms that resemble infantile spasms and are usually resistant to treatment. Severe progressive intellectual disability becomes evident with age. Many die early and most survivals are handicapped. Some may evolve into the West syndrome and some later into the Lennox–Gastaut syndrome (see below). The EEG shows a 'burst suppression' pattern with an almost flat tracing for several seconds, alternating with diffuse, high-amplitude, slow wave-and-spike bursts, poorly modified by sleep–wake stages.[11] The aetiology of Ohtahara syndrome includes usually congenital or acquired malformations of cortical development and diffuse prenatal encephalopathies, the cause of which remains unknown, so far. A report on a case of Ohtahara syndrome included a metabolic defect with cytochrome oxidase deficiency.[12]

Early myoclonic epileptic encephalopathy is another epileptic syndrome occurring during infancy with a grim prognosis.[13] The predominant seizure pattern is erratic, paroxysmal, fragmentary myoclonus, often associated with other seizure types. Brain malformations are not so common as in Ohtahara syndrome.

Infantile spasms occur usually at the ages of 4 to 6 months and in 90 per cent of cases during the first year of life. The events resemble the Moro reflex with sudden, brief flexion of neck and trunk, raising both arms forwards or sideways, sometimes with flexion at the elbows, and flexion of legs at the hips. Less often, the legs extend at the hips. At the early stage flexion of the neck may be the only or main feature; this may be followed by more complex and dramatic attacks later on. A cry is often associated with the attack either as part of the attack or occurring afterwards as an expression of disquiet. The spasms are usually symmetric, but may be asymmetric or even unilateral. The EEG is chaotic with slow waves of high voltage intermixed with diffuse or asynchronous spikes in both hemispheres or in the contralateral hemisphere in unilateral cases. This

Table 10.5.3.4 Differential diagnosis of epileptic and psychogenic seizures

Typical features	Epileptic seizures		Psychogenic seizures
	Generalized tonic-clonic seizures	Complex partial seizures	
Comparison of seizures with known seizure types	Little variation in events	Wide range of events, but the most common are well described	Extremely wide range of events with bizarre or unusual behaviour
Ictal EEG	Abnormal and changed from preictal	Almost always abnormal and changed from preictal	Usually normal and unchanged from preictal
Postictal EEG	Almost always abnormal and changed from preictal	Frequently abnormal and changed from preictal	Usually normal and unchanged from preictal
Effect of antiepileptic medication on seizures	Prominent, especially in severely affected patients	Usually prominent	Usually no effect

Table 10.5.3.5 Classification of epileptic seizures (International League Against Epilepsy, www.ilae.org/Visitors/Centre/ctf/CTF table3.cfm, copyright ILAE)

Partial (focal, local seizures)
Simple partial seizures
With motor signs
With autonomic symptoms and signs
With somatosensory or special sensory symptoms: simple hallucinations (e.g. tingling, light flashes, buzzing), somatosensory, visual, auditory, olfactory, gustatory, vertiginous
With psychic symptoms (disturbances of higher cerebral functions): dysphasic, dysmnesic, cognitive, affective, illusions, structured hallucinations
Complex partial seizures (with impairment of consciousness, may sometimes begin with simple symptomatology)
Partial seizures evolving to secondarily generalized tonic–clonic seizures
Generalized seizures[a]
Absence seizures
Atypical absences
Myoclonic seizures
Clonic seizures
Tonic seizures
Tonic–clonic seizures
Atonic seizures

[a] Combinations of seizures listed here may occur.

Table 10.5.3.6 Semiologic seizure classification[7]

Aura
Somatosensory aura
Visual aura
Auditory aura
Gustatory aura
Olfactory aura
Autonomic aura
Abdominal aura
Psychic aura
Autonomic seizure
Dialeptic seizure
Motor seizure
Simple motor seizure
Myoclonic seizure
Epileptic spasm
Tonic seizure
Clonic seizure
Tonic–clonic seizure
Versive seizure
Complex motor seizure
Hypermotor seizure
Automotor seizure
Gelastic seizure
Special seizure
Atonic seizure
Astatic seizure
Hypomotor seizure
Akinetic seizure
Negative myoclonic seizure
Aphasic seizure

Reproduced from Commission of Classification and Terminology of the International League Against Epilepsy (1989). Proposal for revised clinical and electroencephalographic classification of epilepsies and epileptic syndromes. Epilepsia, **30**, 389–99, copyright 1989, International League Against Epilepsy.

Table 10.5.3.7 Salient epileptic syndromes which may be associated with intellectual disability and salient intellectual disability syndromes which may be associated with epilepsy

Infant
Early myoclonic encephalopathy
Early infantile epileptic encephalopathy with suppression bursts
Infantile spasms
Severe myoclonic epilepsy
Sturge–Weber syndrome
Down syndrome
Fragile X syndrome
Angelman syndrome
Children and adolescents
Epilepsia partialis continua Kojewnikow
Unverricht–Lundborg disease
Lafora disease
Progressive myoclonus epilepsy with intellectual disability
Other neuronal ceroid lipofuscinoses
Sialidosis
Myoclonic epilepsy with ragged red fibres (MERRF)
Rett syndrome
Landau–Kleffner syndrome
Continuous spike-wave discharge during slow-wave sleep

pattern is called hypsarrhythmia. Infants with unilateral spasms need to be examined using a positron emission tomography scan, as contralateral hypometabolism may be due to cortical dysplasia, a condition which may be treatable by resective epilepsy surgery. Aetiology is usually symptomatic including brain abnormalities due to intrauterine infections such as toxoplasmosis, cytomegalic inclusion disease, or rubella. Other aetiologies are brain malformations due to unknown cause. Infants with Down syndrome or tuberous sclerosis may develop infantile spasms. **West syndrome** comprises the triad of infantile spasms, hypsarrhythmia, and intellectual disability.

Progressive degenerative brain diseases and neoplasms are rare causes of infantile spasms. Also neurometabolic disorders such as phenylketonuria, maple syrup urine diseases, non-ketotic or ketotic hyperglycinaemia, and urea cycle defects may lead to infantile spasms.

Severe myoclonic epilepsy in infants includes generalized or unilateral febrile clonic seizures, secondary appearance of myoclonic jerks, and often partial seizures. All the children affected suffer from intellectual disability from the second year of life onwards. Ataxia, signs of upper motor neurone involvement, and interictal myoclonus may appear. [13]

Early childhood

Myoclonic epilepsy of early childhood shares many features with the **Lennox–Gastaut syndrome**.[13] The latter is a group of epileptic disorders of varied aetiology in childhood. West syndrome often evolves into Lennox–Gastaut syndrome characterized by atypical absences, axial, tonic and sudden myoclonic, atonic, partial, and generalized tonic–clonic seizures, diffuse slow interictal spike waves in the waking EEGs and fast rhythmic bursts (10 Hz) during sleep. A progressive decrease in IQ is often found in children with Lennox–Gastaut syndrome. **Myoclonic–astatic epilepsy** or **Doose**

syndrome resembles Lennox–Gastaut syndrome, but is not so severe.

Later childhood and adolescence

Progressive myoclonus epilepsies have the nosological picture of an evolving syndrome of symptoms including massive and segmental myoclonus, myoclonic or tonic–clonic seizures, partial seizures, cerebellar impairment, and higher neurological dysfunctions. [13] Unverricht–Lundborg disease is most common in the Finnish and North African population, but occur also elsewhere. The disease progresses only over a limited period and stabilizes thereafter. [14] The age of onset is around 7 years and the disease starts with myoclonus or nocturnal tonic–clonic seizures. The longest lifespans are more than 60 years. The intelligence level is slightly lowered or even normal. Patients with severe intellectual disability have often had drug intoxication. [15]

Progressive myoclonus epilepsy with intellectual disability (Northern epilepsy) and Lafora disease are more progressive disorders with different gene defects.[16] Sialidosis and mitochondrial encephalopathy with ragged red fibres may also show myoclonic seizures. Epilepsy is quite common in girls with Rett syndrome, affecting about 90 per cent of the patients. They may have several seizure types including partial, generalized tonic–clonic, and myoclonic seizures, atypical absences, short flexion or extensor spasms, and drop attacks or various combinations of such seizures.[17]

Of the progressive **partial epilepsies**, epilepsia partialis Kojewnikow or Rasmussen syndrome type 2 is especially important because the disease is fatal if untreated. The classical model of the association between frequent epileptiform discharges and permanent loss of function is provided by the Landau–Kleffner syndrome or acquired epileptic aphasia. There is increasing evidence that frequent epileptiform discharges, perhaps particularly overnight in the form of continuous spikes and waves during slow sleep, also called electrical status epilepticus, is associated with permanent intellectual impairment if allowed to continue for long periods.[18]

Adulthood and old age

The proportion of cerebrovascular disorders, brain tumours, chronic alcoholism, and sequelae of brain injuries is increasing with advancing age in the aetiology of epilepsy. From about 35 years of age onwards partial epilepsies become more common than generalized epilepsies. Patients with intellectual disability may also develop these disorders.

Behavioural disorders due to epilepsy

Psychic symptoms may be seen as epileptic manifestations of several epileptic seizure types (Table 10.5.3.5). Thus simple partial seizures may manifest themselves with somatosensory or special sensory symptoms including simple hallucinations such as tingling, light flashes, or buzzing or with psychic symptoms such as dysphasic, dysamnesic, cognitive, affective, illusional, or structured hallucinations. Complex partial seizures often include behavioural abnormalities reaching from confusional states to psychotic-like episodes. This is often the case in temporal-lobe and frontal-lobe epilepsies. Among generalized seizures absence status epilepticus resembles psychotic behaviour,[19] which the person in question does not remember afterwards.

In patients with intractable seizures, about one-fifth are non-epileptic in origin. Patients with psychogenic seizures do not generally have seizures when alone or when asleep. Their EEG shows normal activity preictally, ictally, and postictally. The courses of the psychogenic seizures do not show any uniform pattern as is the case with epileptic seizures (see Table 10.5.3.4). Instead their seizures include a variety of behavioural disturbances including conversion, depressive, anxiety, adjustment, somatoform, psychotic, or facitious disorders.[2] The antiepileptic drugs are ineffective against psychogenic seizures but appropriate psychotherapy is helpful in 70 to 80 per cent of cases. Also panic disorder may be difficult to distinguish from complex partial seizures especially in patients with mild intellectual disability. There exists a relationship between brain damage, epilepsy, ictal, and interictal aggressive behaviour, and socio-economic factors. Rarely, ictal aggression occurs in patients with epilepsy, but postictal confusional aggression, and aggression occurring in postictal psychotic states is more common.

Occurrence of epilepsy related to intellectual disability

The prevalences of epilepsy and intellectual disability in the general population are both close to 1 per cent. Epilepsy is more common and more difficult to diagnose and to treat in people with intellectual disability than in those with normal intellect. Population-based studies have revealed that intellectual disability occurs in at least 30 to 40 per cent of individuals with epilepsy,[20] while the prevalence of epilepsy in the population with intellectual disability is about 20 to 25 per cent.[21] It is higher in the more severely disabled (IQ < 50) than in less severely disabled (IQ 50–70)—30 to 50 per cent and 15 to 20 per cent, respectively. Brain damage tends to be more extensive when epilepsy, intellectual disability, or cerebral palsy are complicated by each other or by other conditions of brain origin. It is unlikely that specific causal factors of epilepsy, intellectual disability, or cerebral palsy will ever be positively identified, for these are non-specific clinical features of brain disorder. Epileptic fits themselves, especially if they are persistent, may produce brain damage and play a part in producing a progressive decline in the intellectual functioning of patients. Further apparent deterioration of intellectual functioning may be the result of excessively high doses of anticonvulsant drugs.

Epilepsy occurs in all the main aetiological categories of intellectual disability. In a series of 1000 mentally retarded patients, epilepsy was less frequent in the prenatal category than in the rest of the series (182/515 or 35.3 per cent versus 260/485 or 53.6 per cent). Of the main types of epilepsy, partial epilepsy is more frequent in the prenatal and postnatal aetiological categories and in the category of infections and intoxications.[22]

In people with Down syndrome the frequency of epilepsy is 5 to 10 per cent. There is an age-related bimodal distribution with about 40 per cent of seizures starting before the age of 1 year and another 40 per cent starting after the third decade.[23] Roughly 25 per cent of individuals with fragile X syndrome have epileptic seizures which are usually infrequent, mild, easily controlled, and typically disappear in adolescence, as in benign Rolandic epilepsy. In Angelman syndrome epilepsy is present in more than 90 per cent of the affected individuals.[23] In Rett syndrome epilepsy affects up to 90 per cent of patients[17] Seizures are usually benign

during the early years of life. In patients with aspartylglucosaminuria, epilepsy is found in 28 per cent of adults and in 2 per cent of children.[24] Epilepsy is common (up to 100 per cent) in patients with the various forms of neuronal ceroid lipofuscinoses, especially during the last years of life,[25] and also in other inborn errors of metabolism leading to intellectual disability such as sialidosis type 1, Tay–Sachs disease, type 3 Gaucher disease, mitochondrial encephalopathy with lactic acidosis and strokes, and myoclonic epilepsy with ragged red fibres.

Aetiology and pathogenesis of epilepsy

The presumed aetiology of intellectual disability is also the presumed aetiology of epilepsy in most patients.[21,22] In addition, patients with intellectual disability may develop an ischaemic or haemorrhagic lesion, a neoplasm, or another lesion in the brain which may lead to epilepsy.[21] The presumed aetiology of epilepsy can be found in about three-quarters of the patients. In the aetiological classification based on the time of the presumed cause of epilepsy and intellectual disability, prenatal aetiology is the most common (Table 10.5.3.8). In the aetiological classification based on presumed cause, the categories of unknown prenatal influence, infections and intoxications, trauma and physical agents, and other specified aetiological agents cover most of the cases (Table 10.5.3.9). In patients with intellectual disability and epilepsy it is important to try to find the cause of the intellectual impairment, epilepsy, or epilepsy syndrome. In some cases, the epilepsy syndrome or an underlying inborn error of metabolism may be relevant.

Basic mechanisms leading to epilepsy include disturbances in the balance between excitatory and inhibitory neurotransmitter function within brain cells and their connections to important channels such as voltage-gated sodium channels. For instance, hyperactivity of the excitatory neurotransmitter glutamic acid and/or hypoactivity of inhibitory neurotransmitter γ-aminobutyric acid (**GABA**) may lead to epileptic seizures. The existence of so many genetically determined disorders leading to intellectual disability and epilepsy[26] (Table 10.5.3.10) and the large variation in the prevalence of epilepsy in the specific intellectual disability syndromes and the use of new methods such as an array technology suggest that genetic factors play a more important role in producing epilepsy.

The three following examples, as well as those mentioned above, illustrate this variety of genetic explanations for epilepsy among intellectual disability syndromes.[23] Angelman syndrome is a contiguous gene defect most often caused by a maternally inherited deletion of chromosome 15q11–13. Several of the deleted genes code for GABA receptor subunits. Deficits of inhibitory GABAergic function could directly predispose affected individuals to seizures. This hypothesis is supported by knockouts of analogous chromosome region in mice, which produces an epileptic phenotype.

In Down syndrome or trisomy 21 the bimodal distribution of the frequency of epilepsy between young and older ages is interesting. The fact that more than 75 per cent of adults with Down syndrome develop late-onset epilepsy coincident with the onset of the neuropathological abnormalities in the brain compatible to Alzheimer's disease suggests an aetiological role of these abnormalities. However, as epilepsy is associated with Alzheimer's disease in only 10 per cent of patients without Down syndrome, it is unlikely that Alzheimer's neuropathological abnormalities are solely responsible for the late-onset epilepsy in patients with Down syndrome. As the EEG of most of these patients is characteristic of idiopathic generalized epilepsy and the gene for progressive myoclonus epilepsy is located in the Down syndrome region on chromosome 21, it is quite possible that this gene product predisposes for a senile myoclonus epilepsy in Down syndrome.[23] Abnormal neuronal circuits with fewer GABAergic neurones in certain cortical layers, cerebral dysgenesis particularly of dendritic spines, pathophysiological membrane ion channels, and altered neurotransmitter level are potential mechanisms of epilepsy in Down syndrome.[23]

In the tandem trinucleotide repeat disorder, fragile X syndrome, triplet expansion (CGG) results in shutdown of fragile X intellectual disability 1 gene transcription, which may alter overall neurologic development and lead to seizures.[23]

Thus, there exists a spectrum of epilepsy mechanisms among these three intellectual disability syndromes, ranging from deletion of a gene or genes that directly leads to hyperexcitability (Angelman syndrome), to a chromosomal triplication that alters several aspects of neuronal development and function (Down syndrome), to a

Table 10.5.3.8 Aetiological classification of epilepsy and intellectual disability according to time of presumed cause

	N = 129[a]	*N* = 442[b]
Prenatal (%)	35.1	41.2
Perinatal (%)	10.0	15.4
Postnatal (%)	8.7	18.8
Multiple (%)	14.7	4.3
Unknown (%)	31.4	20.4
Total (%)	100.0	100.0

[a] Data from Forsgren *et al.*[21]

[b] Data from Iivanainen.[22]

Table 10.5.3.9 Aetiological classification of epilepsy and intellectual disability according to presumed cause

	N = 442
Infections and intoxications (%)	18.1
Trauma and physical agents (%)	16.3
Disorders of metabolism (%)	3.2
Gross prenatal influence (%)	24.0
Prematurity (%)	0.5
Major psychiatric disorder (%)	0.5
Psychosocial deprivation (%)	0.0
Multiple causes (%)	7.2
Hereditary (simple) (%)	0.5
Other specified (%)	19.7
Unspecified (%)	0.0
Total (%)	100.0

(Reproduced from M. Iivanainen, Diagnosis of epileptic seizures and syndromes in mentally retarded patients. In *Paediatric epilepsy* (ed. M. Sillanpää *et al.*), pp. 233–41. Copyright 1990, Wrightson Biomedical, Petersfield)

Table 10.5.3.10 Genetic diseases with epilepsy and intellectual disability

Disease	Mode of inheritance	Gene location
Progressive epilepsy with intellectual disability (Northern epilepsy)	AR	8p
Unverricht–Lundborg disease (Baltic, Mediterranean)	AR	21q22
Infantile neuronal ceroid lipofuscinosis	AR	1p32
Late infantile neuronal ceroid lipofuscinosis	AR	11p15
Variant late infantile neuronal ceroid lipofuscinosis	AR	15q21-23
Juvenile neuronal ceroid lipofuscinosis	AR	16p12
Finnish variant neuronal ceroid lipofuscinosis	AR	13q22
Lafora disease	AR	6q23-25
Mitochondrial encephalomopathy, lactic acidosis (UUR) and stroke-like episodes (MELAS)	Maternal	tRNA-Leu (UUR)
Myoclonic epilepsy with ragged red fibres (MERRF)	Maternal	tRNA-Lys
Epilepsy and mental retardation limited to females	X-linked	Xq22
Tuberous sclerosis[a]	AD	9q34 16p13
Angelman syndrome[a]	AD	15q13
Neurofibromatosis type[a] 1	AD	17q11
Fragile X[a]	X-linked	Xq27
Rett syndrome[a]	X-linked	Xq28-McCP2

AR, autosomal recessive; AD, autosomal dominant.

[a] Epilepsy and/or intellectual disability may be manifested as part of the phenotype.

specific tandem repeat which alters neuronal function in a non-specific and probably benign manner (fragile X syndrome). It remains to be seen how much dissection of genetic mechanisms underlying other intellectual disability syndromes will provide additional insight into epilepsy mechanisms.

Treatment of epilepsy

The diagnosis of epilepsy and its underlying disorder needs to be made without delay. The identification and avoidance of provoking factors likely to precipitate seizures in each individual is an essential aspect of the overall management. If this is insufficient, antiepileptic drug treatment is needed. If this is still insufficient, epilepsy surgery should be considered. Important points to be considered include not only the nature and severity of an underlying disease, but also the degree and location of brain lesion, the age of the patient at onset of epilepsy, and possible pseudodisability (pseudoretardation) caused by epileptic seizures or by inappropriate medication. It is emphasized that treating frequent epileptiform discharges may not only reverse the intellectual disability which in such cases is pseudodisability or state-dependent intellectual disability,[18] but may also in some cases prevent permanent intellectual disability.

Antiepileptic drug therapy

Drug interactions

Antiepileptic drugs interact with each other by three principal mechanisms: enzyme induction, enzyme inhibition, and through altered protein binding. Phenytoin and phenobarbital induce a wide range of enzyme activity. Carbamazepine induces its own enzymatic metabolism, and may induce the metabolism of valproate and phenytoin, resulting in lower concentrations of these drugs. Valproate inhibits the metabolism of phenobarbital and the epoxide of carbamazepine, resulting in high concentrations of each of these. Lamotrigine is metabolized in the liver. Valproate inhibits the metabolism of lamotrigine, resulting in a longer half-life and higher blood levels of lamotrigine while the blood level of valproate may be decreased. Lamotrigine does not affect the blood level of carbamazepine. Gabapentin is not metabolized at all and is excreted in the urine unchanged. If levetiracetam is administered with enzyme-inducing drugs, its clearance may increase by 22 per cent, although the drug concentrations in serum do not change when using other antiepileptic drugs simultaneously. The free or unbound fraction of antiepileptic drugs is in equilibrium with the brain concentration and is considered to be more relevant than the total blood level. When two drugs with a high degree of protein binding are used together, for example phenytoin and valproate, there may be some displacement of each drug from protein binding, increasing the unbound fraction. This may result in clinical neurotoxicity even when the total (bound plus unbound) blood level is within the reference range. The antiepileptic drugs also interact with many other drugs and may affect their blood levels and action, and vice versa. For instance, chloramphenicol, cimetidine, anticoagulants, ibuprofen, imipramine, propranol, and some psychotropic drugs inhibit the metabolism of phenytoin and lead to an increase of phenytoin blood level and possibly to phenytoin intoxication unless the dose of phenytoin is reduced.

Choice of drug

Once the diagnosis of epilepsy has been made, the decision must be made as to whether antiepileptic medication is needed or not. If it is, the most appropriate antiepileptic drug is to be selected. The choice of antiepileptic medication depends primarily on an accurate classification of the seizure type and/or epilepsy syndrome.

Most treatment decisions have to be based on the results of studies of people who do not have intellectual disability. The exceptions are mainly studies of adults with the Lennox–Gastaut syndrome, where lamotrigine is recommended.[27] Valproate, carbamazepine, oxcarbazepine, and levetiracetam[28] are other antiepileptic drugs recommended in generalized and partial seizures based on uncontrolled studies or consensus. Valproate is the first choice for generalized epilepsies/seizures, while oxcarbazepine/carbamazepine is the choice for the focal epilepsies/seizures of people with intellectual disability.[29] Dosage and recommended drug levels in blood are presented in Table 10.5.3.11. If newer drugs are not available, phenobarbitone, primidone, and phenytoin may be used with caution, if special attention is paid not only to control of seizures, but also to behavioural, cognitive, and cerebellar functions which may

Table 10.5.3.11 Pharmacokinetic properties of antiepileptic drugs

Drug	Dose (mg/kg/day)	Doses per day	Therapeutic range	
			(μg/ml)	(μmol/ml)
Phenobarbitone	1–3	1	10–30	40–130
Primodone	10–15	2 or 3	6–12	25–50
Phenytoin	4–6	1 or 2	10–20	40–80
Carbamazepine	15–20	2 or 3	4–12	15–50
Oxcarbazepine	15–40	2 or 3	15–23	30–120[a]
Valproate	15–30	2 or 3	50–100	300–700
Ethosuximide	15–30	1 or 2	40–100	280–700
Levetiracetam	20–40	2	5–65	30–370
Vigabatrin	40–100	1 or 2	NA	NA
Lamotrigine	2–4(10)[b]	1 or 2	NA	NA
Gabapentin	20–40	3	NA	NA
Topiramate	400	3	NA	NA
Tiagabine	32–56	3	NA	NA
Clonazepam	0.01–0.2	2 or 3	20–75	60–240

NA = not available.

[a]For monohydroxy derivative.

[b]With enzyme-inducing drugs.

be affected adversely, and sometimes insidiously, by these drugs. However, it is stressed that because of the lack of well-designed properly conducted randomized controlled trials for patients with newly diagnosed generalized of focal untreated seizures/epilepsies and for children in general, it is impossible at present to develop evidence-based guidelines aimed at identifying the overall optimal recommended monotherapy antiepileptic drug.[30]

Most antiepileptic drugs may aggravate certain epilepsies. This is the case especially with phenytoin and carbamazepine in idiopathic generalized absence and myoclonic epilepsies. Although valproate has low risk of seizure aggravation,[31] it may cause weight gain, polycystic ovaries, and hepatitis. Oxcarbazepine may lead to hyponatraemia and vigabatrin to visual field defects.[32]

Withdrawal of treatment

Withdrawal of antiepileptic medication in patients with intellectual disability needs to be considered, provided their epilepsy is well-controlled and there are no specific contraindications for the withdrawal. Depending on the type of seizure or epilepsy syndrome and the individual history, it might be worth considering slow reducing the medication after a 2-year seizure-free period. However, if the individual is in a particularly poor prognostic category or if there is a history of severe, prolonged status epilepticus, it is worth waiting for longer before attempting medication reductions. Despite these reservations, attempts to discontinue antiepileptic medication in people with intellectual disability can be successful. It would appear that a better likelihood of a successful outcome may be suggested by later onset of epilepsy, i.e. after 2 to 2.5 years of age, a shorter duration, lower antiepileptic drug levels, and normal EEGs, together with complete control of the seizures.[33] The risk of recurrence of the seizures also depends on the type of epilepsy.[34] In complex partial seizures, where exogenic factors are more significant than genetic ones, the prognosis is good after 2 to 4 seizure-free years. The prognosis is worse in simple partial seizures, or in absence seizures with tonic–clonic seizures and grand mal tonic–clonic seizures, and at least four seizure-free years are recommended before ceasing medication. Patients with juvenile myoclonic epilepsy, or absence seizures with clonic–tonic–clonic seizures or grand mal clonic–tonic–clonic seizures, may need to take long-term, even lifetime medication. The relapse rate is likely to be high, even if these patients have been free of seizures for several years.

Status epilepticus

First-line treatment of status epilepticus is usually with intravenous benzodiazepines, either diazepam or lorazepam. If this is not effective, then intravenous phenytoin or intravenous or intramuscular fosphenytoin is recommended. Rectal paraldehyde may be of value in children. Rectal diazepam has been the pre-hospital treatment of first choice because it can be administered by non-medical personnel. A history of status epilepticus is liable to influence decisions about withdrawing regular antiepileptic medication. It would be wise to proceed with caution, ensuring that any recurrence of status epilepticus can be treated readily, if withdrawal of regular medication is to be undertaken.

Epilepsy surgery

Frequent severe epileptic seizures despite treatment with adequate antiepileptic medication for about 2 years means that epilepsy surgery needs to be considered. In addition there are certain disorders such as Sturge–Weber syndrome or unilateral infantile spasms where epilepsy surgery may be of benefit during infancy. The treatment of choice in children with Rasmussen syndrome type 2 currently is hemispherectomy, which needs to be done as early as the diagnosis is clear. Difficulties in cooperation and minimal psychosocial gains due to low IQ as well as progressive underlying disease may be contraindications for epilepsy surgery. The preoperative consideration includes extensive examinations such as video-EEG monitoring, high-resolution MRI, positron emission tomography, and neuropsychological and psychiatric evaluation according to the generally accepted principles.[35] The goal is to select those candidates who will benefit from epilepsy surgery. Surgical outcome varies according to the different pathologies of epileptogenic lesions. Thus, the results of surgery are better among patients with mesial temporal sclerosis, chronic encephalitides, infantile hemiplegia, focal cortical dysplasia, tuberous sclerosis, Sturge–Weber syndrome, or post-traumatic cicatrix than among patients with extratemporal focal sclerosis, polymicrogyria with or without heterotopia or hemimegalencephaly, anoxic brain damage, gliosis of obscure aetiology, or no structural pathology.[36] All these findings must be taken into account when selecting patients with epilepsy and intellectual disability for epilepsy surgery. As onset of intractable epilepsy within the first 24 months of life is a significant risk factor for intellectual disability, early intervention for epilepsy surgery is emphasized.[37]

Behavioural disorders caused by antiepileptic drugs

Most antiepileptic drugs may also cause behavioural disturbance and cognitive dysfunction.[38] For example, the diplopia caused by carbamazepine may result in considerable distress and consequent behavioural disturbance. Communication difficulties, which are

common in intellectually disabled patients, may add to the distress and make behavioural disturbance more likely.

Intellectually disabled people with epilepsy are especially vulnerable to harmful neurotoxic effects—sedation caused by phenobarbital or benzodiazepines or cognitive and cerebellar dysfunction caused by phenytoin alone or often together with other antiepileptic drugs. If these alarming effects are not taken into account, inappropriate medication may even jeopardize the rehabilitation of the patients.

Uncertainty about the long-term effects of antiepileptic drugs on brain function and development is largely due to conflicting results of often biased human observations. The problem can only be resolved in controlled experiments which by necessity must be done in animal models. Behavioural and structural consequences of epileptic activity and their modification by antiepileptic drugs are important points to be evaluated in experimental studies. It was reported[39] that enhancement of GABAergic inhibition by administration of vigabatrin prevented both pyramidal cell damage in CA1 and CA3 areas of hippocampus and the disappearance of somatostatin immunoreactive neurones from the dentate gyrus after perforant path stimulation in rats. Furthermore, the preservation of hippocampal structure was accompanied by prevention of the spatial memory deficits seen in control animals after such stimulations. Another study using a kainic acid status epilepticus model in adolescent rats[40] showed that animals that received kainic acid followed by valproic acid resembled control animals who had never received kainic acid with respect to their behavioural and memory performance and had fewer histological lesions. Animals that received kainic acid followed by saline or phenobarbital had impaired learning and behaviour, and more extensive lesions in the hippocampus. Thus, in this experiment valproic acid suppressed seizures and subsequent epilepsy while phenobarbital was only partly effective in suppressing seizures and did not prevent epilepsy. It is likely that seizures themselves, as opposed to the drugs, produced negative behavioural consequences in these rats. It is noteworthy that valproate at very high doses was protective against neuronal damage and prevented epileptogenesis in the kainic acid model.

Chronic phenytoin intoxication, especially in multiple drug therapy, may lead to ataxia, balance impairment and in the worst case finally to persistent loss of locomotion.[41] Another example of an insidious and dangerous effect of phenytoin was documented by the changed course of Unverricht–Lundborg disease. Its rather benign course worsened during phenytoin treatment, so that the patients became bedridden and pseudoretarded and their lifespan shortened from 50 to 60 years to under 30 years. When these patients were treated with valproate instead of phenytoin, their lifespan increased to the prephenytoin level.[15] In some of these patients, the loss of locomotion was reversible after valproate replaced phenytoin.

Prognosis

The long-term outcome of patients depends primarily on the underlying disorder; prognosis is better in idiopathic than in symptomatic cases. If it is not a question of a progressive brain disorder, epilepsy is quite easily treatable in patients with intellectual disability. Thus, about 70 per cent may obtain good seizure control with appropriate antiepileptic drug therapy or epilepsy surgery. The outcome of patients with specific disorders may vary considerably. For instance, the outcome of patients with Doose syndrome is variable but basically better than that of patients with Lennox–Gastaut syndrome. If hemispherectomy is not undertaken in time in Rasmussen syndrome type 2, the course of the disease, including neurological deficits, other types of seizures, and mental impairment is progressive.

Epileptic seizures themselves, and frequent epileptiform discharges, may produce brain damage and play a part in producing progressive decline in the intellectual functioning of patients[18] probably through at least two mechanisms: excitatory glutamate storm within cerebral neurones[42] and opening of the blood–brain barrier[43] during and after epileptic seizures.

Sleep disorders are often associated, in people with intellectual disability, with difficult-to-treat epilepsy and behavioral problems.[44,45] When sleep disorders are diagnosed and treated, antiepileptic and also psychotropic medication can be reduced successfully.

Conclusions

The quality of life in this population benefits from early diagnosis and differential diagnosis of epilepsy, including epilepsy-related behavioural disorder in patients with intellectual disability, identification of its aetiology, and appropriate antiepileptic drug treatment using firstly one drug therapy and, if needed later, rational multiple drug therapy. Currently, valproate is the first choice in generalized seizures while oxcarbazepine or carbamazepine are used for partial seizures with or without secondary generalization. Broad-spectrum drugs such as levetiracetam, lamotrigine, topiramate, or zonisamide are promising. The usefulness of epilepsy surgery should be considered in intractable cases no later than within 2 years, if adequate antiepileptic drug therapy does not help. To minimize sedative and other behavioural and other side-effects caused by antiepileptic drugs the fewest possible drugs should be administered at the lowest effective dose. This means that there should be careful clinical observation of the patients together with determination of drug concentrations in blood and other appropriate laboratory tests. Psychological aspects and sleep behaviour of the patients need to be taken into consideration in the treatment of epilepsy in patients with intellectual disability. Doctors and other personnel working in this field need special education.

Future prospects in the treatment of intractable epileptic seizures might involve the development of gene therapy, neuroprotective drugs, and drugs targeting epileptogenesis. Such treatment possibilities may bring new hope for people with epilepsies that are difficult to treat, including the population with a high proportion of refractory cases, namely that with intellectual disability.

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10.6

Methods of treatment

T. P. Berney

Introduction

The presence of intellectual disability (mental retardation) affects the character of treatment in a number of ways.

1 Limited communication will hamper diagnosis so that much more has to be inferred from observable behaviour and greater weight given to the interpretation of carers.

2 Diagnosis is provisional, a therapeutic hypothesis that provides the basis for a programme of treatment that is, essentially, a therapeutic trial. There is a wide variation in individual characteristics, with intellectual disability (mental retardation) extending over an enormous range of ability, associated disabilities, aetiology, and psychopathology. Multiple pathology means that the response can be unexpected and ambiguous. For example, diminished aggression with carbamazepine may simply reflect its psychotropic effect but may also be the result of better control of unrecognized epilepsy. A well-designed behavioural milieu may produce a rapid improvement in disturbed behaviour. However, the improvement might simply reflect the effect on someone with autism of moving to a more settled, structured, and predictable environment. It may also reflect an improvement in organic disorder (such as epilepsy or gastritis) in response to a reduction in stress. The therapist has to tailor the treatment to the individual, to try to be specific as to what aspect of the disorder is being targeted and be very selective as to whom they treat.

3 Ill-defined treatment objectives often leave it unclear whether a treatment is aimed at a disorder (e.g. autism), a symptom (panic), or an associated disorder (depression or epilepsy).

4 Normal developmental change may be misattributed to a coincident treatment programme. For example, autism and epilepsy often show spontaneous improvement at about 4 years age and again in late adolescence, both times when the person is likely to be moving into new programmes. This propensity for maturational improvement, coupled with a tradition of care, has led developmental psychiatrists to have a greater therapeutic optimism for many problems, such as personality disorders, that are often considered intractable in those of normal ability.

5 Natural cycles of change can give a misleading sense of success. For example, a behavioural programme may be credited with the remission of a self-limiting episode of disorder such as depression. The true diagnosis may become clear only when it recurs and fails to respond to booster programmes.

6 There is limited evidence for the effectiveness of most treatments. Except for the behavioural therapies, most treatment is based on small series, open trials, the theoretical, and the ideal.

7 A large component of the therapeutic relationship is indirect, being with the family, carers, or professionals rather than directly with the patient. Many programmes utilize the power of the placebo effect, a dynamic that confounds scientific trials but one that should be used to its full in everyday clinical practice. Limited communication and greater dependency lead to work with the systems around the patient; many of the approaches, such as family therapy, deriving from child psychiatry.

8 The ability to consent to treatment is often underestimated. Circumstances often make it difficult for people with mental retardation to choose or refuse a particular therapy, particularly behavioural programmes and drug treatments. Their capacity to give or withhold consent should be assessed automatically and their care should fall within a legal framework that safeguards their rights and protects them from abuse.

Treatment services for people with intellectual disability (mental retardation) have two components. There is the routine support that should be available to all with intellectual disability (mental retardation). Its aim is to help people to grow up as normally as possible, offsetting the effects of their disability, and to establish the therapeutic environment. Second is the provision of treatment for individuals with disturbance; aimed at specific symptoms or disorders based on a multiaxial diagnosis[1] that includes the following:

- Axis I: The nature and degree of intellectual disability (mental retardation) for, in addition to the overall developmental delay other, specific, disabilities, and abilities are often present. For example, a discrepancy between receptive and expressive language may result in someone understanding little of what is said to him while sounding falsely fluent.
- Axis II: The aetiology of the retardation—there is increasing recognition of the contribution of a behavioural phenotype. Of particular note are autism and its imitators, drawing on the ubiquities of social impairment, obsessionality and communication problems, which are being teased apart.

- Axis III:
  - Level A: Other developmental disabilities that are associated with intellectual disability (mental retardation) such as autism, attention deficit disorder, and epilepsy,
  - Level B: Psychiatric disorder—the way this is defined will define the mode of treatment. A functional analysis with antecedents, triggers, and consequences leads into a behavioural programme. A more biological label (such as psychosis) opens the door to drug treatment. They are not mutually exclusive,
  - Level C: The patient's personality—this is often unusual and it may be difficult to distinguish from a pervasive developmental disorder (see Chapter 9.2.2) which runs through Intellectual disability (mental retardation) and which, once recognized, often explains the inexplicable,
  - Level D: Other disorders such as habit disorders and sexual preference disorders,
- Relevant comorbid, physical conditions such as hay fever, asthma, hypothyroidism, or gastro-oesophageal disorders. Particularly important are epilepsy and the antiepileptic agents that are dealt with in detail elsewhere (see Chapters 6.2.6 and 10.5.3, respectively).
- The patient's environment which includes not just their physical surroundings but also the people and their relationships with them.
- Contributory factors from the patient's past, notably the various forms of abuse.

The therapeutic environment

Support may be provided in different ways:

- Level A: General, the network of care provided for people with a intellectual disability (mental retardation) and their carers. This will include community teams, special schools, and the specialized residential placements that might be resorted to either as a short break or as a long-term home.
- Level B: Specific to a particular disorder, parental support groups exist for autism, epilepsy, and specific forms of intellectual disability (mental retardation) such as Prader–Willi, Fragile X, and Cornelia de Lange syndromes.

A primary aim is to integrate people into their community as far as possible. The concept of normalization implies that services should avoid the demarcation that leads to adverse discrimination. Conversely, those with disabilities too severe or too complex for their families or standard teaching or occupational placements may fare better in specialist settings. Examples of these are as follows:

- Some of those with autism, who are so distracted by the complexity of everyday life and the unpredictability of people that they need specialist environments which are well structured, predictable, and under their control.
- Those with severe or intractable seizures.
- Those with aggressive or disinhibited behaviour so disruptive as to block the progress of their peers.

The specialist setting encourages mutual support for people and families with similar problems; the staff gain experience; and it allows a concentration of expertise. However, it also encourages stigmatization and there is the risk that, in a group, disturbed people may copy or amplify each other's behaviour.

The family and other carers

Disturbance arises in the setting of a system of care which includes not only the family but also the staff of other placements, whether day or residential, educational or occupational. Its management will depend on the way these people perceive mental retardation and its care, their attitudes deriving from both past experience and present relationships (Fig. 10.6.1). A great deal depends on the extent to which they feel supported and assured in their roles, as much by each other as by the available system of care. For example, a mother or a teacher who is told frequently that, whatever it was that went wrong, it was her fault, is unlikely to cope confidently.

Disability and disturbance both hinder normal developmental experience; the child's atypical response spoiling the carers' efforts to learn parenting skills and leaving them demoralized or deskilled. They may need formal teaching in such skills as how to engage socially with the child, to play with them, to give clear and understandable instructions, and how to divert rather than confront. Disturbance may reflect boredom and be reduced simply by increasing the amount and variety of activities. Any approach must take a broad view, for carers have to work together comfortably enough to be consistent over time. A treatment programme may have to address the relationship between the carers as well as their needs. This may be sufficient in itself, improvement in the patient following an overall improvement in functioning in the family, school, or residential placement. There has been a growth in the conscious application of a systemic approach to work in this field.[2]

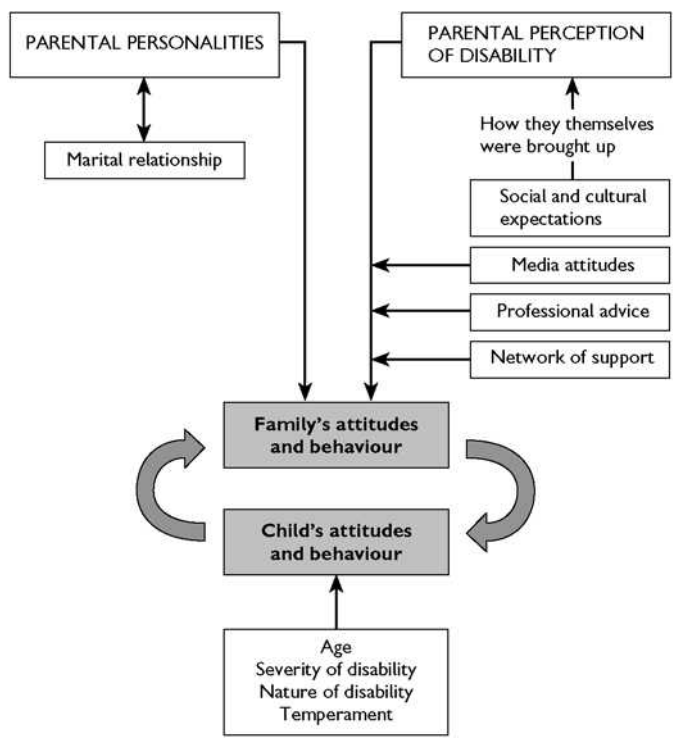

Fig. 10.6.1 The start of disturbance: factors influencing behaviour.

Education

This forms the core of any treatment programme, the individual getting formal instruction in the skills which others acquire in passing. Teaching and training are lifelong processes, invoking teacher–student relationships, and take place within a structured framework, essential for those students who have difficulty in understanding their environments. The difficulty is to gauge the degree of structure required: too much and the relationship risks becoming a battle about control, easily turning into trench warfare; too little and the student is distracted and learns little. It is a balance that shifts over time, promoting the student's self-control and autonomy.

(a) Independence and self-help skills

These include the basic skills of everyday life, such as feeding, dressing, or managing stairs or, at a higher level, the use of public transport, how to care for clothes, shop, and budget. Acquiring these, gives a sense of achievement and confidence as well as of increased independence.

(b) Communication skills

The frustration of living in an uncomprehending world frequently contributes to disturbance as the person falls back on various forms of attention seeking or violent behaviour to get their message across. Easier and more effective means of communication may range from simple gestures (e.g. pulling at the trousers to show the need for toileting), through a system of pointing to symbols or pictures, to complex signing which can convey abstract concepts such as emotional states. Language may be verbal or non-verbal and both modalities are taught simultaneously, reinforcing each other so that a course in signing can improve speech. Whatever the system used, it depends on, and will be limited by, the extent to which those around can understand it.

(c) Social and sexual relationships

People are vulnerable to exploitation until they learn the distinction between an acquaintance and a friend. Relationships become complicated by sexual behaviour, hedged around with cultural rules which vary between families making it an especially difficult area to teach and therefore tempting to ignore. Occasionally sexual arousal drives disturbance in someone for whom masturbation is inefficient, physically damaging, unlearned, or forbidden. Wider discussion can bring out strongly held beliefs and family conflicts which had been unsuspected or denied until then. This may lead on to other areas requiring resolution, such as whether a person with intellectual disability (mental retardation) should have a sexual relationship, marry, or have children.

Out-of-home placement

As described earlier, a move out of the home can be seen either as part of the wider programme of support and social d evelopment or else as part of the treatment of a specific disorder: the distinction is often blurred.

(a) Support

Short breaks allow individuals and their families some relief from the uninterrupted intimacy of care. They also widen social networks and pave the way towards the eventual departure from home, something that is ideally planned as part of an increasing, adult autonomy. Frequently however, this is left until there is a crisis, for example when the person's behaviour or dependency has outstripped their family's resources, too often the result of parental infirmity or death. At this point the unfortunate individual may find themselves in a series of short break (or even treatment) placements until something long-term can be arranged.

Placement for educational needs (e.g. in a residential school or college) becomes necessary where the person's disabilities (e.g. autism or intractable epilepsy) require specialist skills and settings. It can also be a compromise with a parent who, unable to care for their child themselves, will not accept more standard forms of social care.

(b) Treatment

The threshold for admission for assessment and treatment will depend on the extent of the supportive service available in the community and is often:

1 For the treatment of more complex disorders such as epilepsy or psychosis.

2 For the assessment of disturbance where it is difficult to disentangle the relative contributions of innate from environmental factors. For example, a patient's behaviour may be amplified by an exasperated or exhausted family, particularly where disturbed nights have left them short of sleep. This sets off a secondary, self-perpetuating cycle of disturbance involving the whole house which makes it impossible to discern the underlying, primary disturbance. The cycle may only be interrupted by changing the patient's circumstances, either by moving staff into the home or by moving the patient out.

3 For the management of behavioural disturbance where:

(a) The carers are unable to cope—the more frequent reasons include:

- marital disharmony
- the carer's inability to manage others simultaneously, for example single parents who have several children or a home with several disturbed children
- the loss of resilience in a demoralized carer
- an adverse or hostile neighbourhood where the carer has to give way in case the patient becomes so noisy that the neighbours complain, or where the patient is bullied or led astray.

(b) There has been a failure of earlier therapeutic trials with a family locked in their pattern of behaviour.

(c) The patient is at risk of harm.

(d) The patient presents a substantial risk to others, for example, of violence or sexual offending.

(e) There is the need for effective control of the patient with clear limits to disturbed behaviour.

Treatment methods

Discomfort is a potent and frequent cause of disturbance. Because the person may have difficulty in identifying and localizing pain, let alone communicating symptoms, it is frequently missed. It is essential that it is a matter of routine to seek and treat common ailments such as hay fever, toothache, earache, dyspepsia, and gastric

reflux. The last of these is particularly associated both with severe retardation and with certain disorders such as Cornelia de Lange syndrome as well as an increased possibility of oesophageal carcinoma.[3] The more severe the degree of disability, the greater the need for an active programme of health care.[4]

Behavioural treatments

Intellectual disability's communication barrier has led to an emphasis on observable behaviour rather than on reports of subjective emotions or perceptions. Although the function of a behaviour is often misinterpreted, the first, unthinking response is often a behavioural programme. At the same time, increasingly sophisticated and more solidly research-based than other forms of psychiatric treatment, such a programme can produce profound and rapid change.[5] Programmes are divided broadly into two areas:

1 Teaching appropriate habits and skills which can range from basic skills such as dressing, continence, communication, and sleep, through to the more sophisticated training in social skills, dating skills, and assertiveness.

2 The unlearning of other, maladaptive forms of behaviour.

These two areas are complementary—it is more effective to replace an undesirable behaviour than simply to remove it. Operant, incentive programmes dominate much institutional and offender work. While punishment techniques can be effective and in certain, very unusual situations may be justified, there must be concern about their effect on the trainer as much as on the patient and they need close, ethical control.[6]

Cognitive behavioural therapy

Behavioural principles can be used to target thought as well as behaviour. With disability comes a tendency to a polarized perception of the world—people seeing themselves either as acceptable, competent, and successful or else as worthless failures; similarly, others are seen as all good or all bad. Cognitive therapy is usually used with people in the mild or borderline range of retardation, and has a particular importance in the treatment of sex offenders, although there are some examples of work with non-verbal people or those with severe intellectual disability. Indeed, where problem-solving skills are formally taught, the improvement may be greater in people with moderate than with mild intellectual disability, suggesting some form of ceiling effect to their acquisition. Furthermore, performance may have more to do with the type of problem than with formal measures of ability.[7]

Rational emotive therapy, developed in an educational setting, seeks to change the perceptual set and thereby the impact and influence of events on the person. Its background in education resulted in a didactic format, giving the therapist a directive role. The orientation is behavioural, focusing on the development of skills, and it lends itself to being taught to groups. A number of open studies of people with moderate and mild intellectual disability have shown it to decrease irrationality and anxiety, and to increase internal control and self-esteem.[8]

Anger management was developed with people of average ability but now has a well-established place in intellectual disability.[9] Therapy has to cope with a number of obstacles including:

1 The inherent nature of anger which means that the problem is an excessive response rather than a deviant one. In this population organic factors are frequent, particularly brain damage, epilepsy, and medication.

2 Its usefulness where there is limited communication or more severe disability which, combined with personality factors, can make therapeutic engagement difficult.

3 The degree to which habitual use has made anger an entrenched response.

4 The emotion of anger can be difficult to distinguish and label, particularly where autism is a component. Here an aggressive response may be the result of excessive anxiety, often amounting to panic.

A programme of anger management may take place at several levels.

1 General clinical care—strategies to reduce anger which include ensuring that the person feels well, that their physical and social environment is suitable, and that there are suitable occupational and recreational programmes.[10]

2 Anger management—information is given to help the person recognize anger, its nature, the signs, and consequences, as well as ideas and information about changing their behaviour. This uses a more didactic group instruction which is general and involves less disclosure and engagement by the individual.

3 Anger treatment—an individually tailored programme which targets change in cognitive perception, autonomic arousal, and behaviour. Individual engagement is essential and transference and countertransference are important and likely to evoke distressing emotions.

Another target is anxiety reduction through treatments that range, depending on the degree of disability, from formal relaxation training through to physical activity. Some do not measure up to their promoter's promise and may even have a detrimental effect,[11] endorsing the principle of treatment as an individual therapeutic trial.

Psychodynamic therapies

Although we have moved away from the early belief that intellectual disability itself was the result of emotional abuse and psychological disturbance, we are beginning to recognize the extent to which these factors can reduce adaptive functioning and amplify a cognitive disability. Psychotherapy can complement educational programmes to produce someone who, although no more intellectually able, is more mature emotionally and better able to cope with the tasks of everyday life. However, the field is poorly researched so that its efficacy is uncertain.[12]

Both the family and the individual have to adjust to disability; a process akin to a series of grief reactions through which people come to terms with the loss of normality.[13] The process of adjustment occurs as a series of crises triggered by events such as the point of initial diagnosis, the failure of initial treatments, educational assessment and specialized placements, puberty, and leaving home. Each stage brings home afresh the degree and significance of the child's disability.

The therapist may require specific training and supervision in adapting standard approaches to cope with a number of potential elements peculiar to intellectual disability:

1 The therapy is likely to have to deal with the core themes of loss and disability.

2 Communication will be limited in various ways and there may be unexpected, conceptual barriers. For example, the patient may not understand or even notice gestures, facial expressions, and different tones of voice. They may be unable to identify many emotions, label them, or form abstract concepts. Communication has to be at the patient's level, being concrete and using simple words, short sentences, and their colloquial or slang terms as well as allowing sufficient time for the patient to process the thought. The therapist may circumvent some of these barriers by using alternatives modalities, such as music, art, play, and drama.

3 The patient may have a limited and distorted understanding of the roles and relationships of the people around. Unable to appreciate either that the therapist's knowledge is restricted or that they occupy a different world, the patient may assume that they know all about the patient's setting and routines. This means that, in order to make sense of what is said, the therapist must learn something of the patient's background.

4 A combination of memory problems, obsessionality, or poor executive function means that there may be repetition of information, ideas, and conclusions, often given as if they have never been mentioned before.

5 Many people have had lives marked by short and changing relationships and a nomadic change of accommodation. Engagement in therapy can be difficult with a disconcerting readiness to disengage.

6 Confidentiality may be difficult to establish for someone where dependency and total care has discouraged privacy. Carers frequently expect to be told what happens in therapy and the relationship may be complicated by a disclosure that can range from poor care to frank abuse.

Psychopharmacological treatments

Drugs are widely used in intellectual disability. Besides being used for the medical disorders which are more frequent in this population, such as epilepsy, Tourette syndrome, and attention deficit disorder, drugs play a part in the management of a wide range of symptomatology which includes symptoms as varied as aggression, self-injury, outbursts of distress, compulsive routines, and social withdrawal. A recurrent criticism is that a drug may be used for a purpose other than that suggested by its classificatory label (e.g. that antipsychotics and antiepileptics are used for conditions other than psychosis or epilepsy, respectively) or that they are prescribed outside their manufacturer's licence. While this often is simply semantic, there must be concern at the level of prescribing of psychotropic drugs.[14] The prevalence and number of drugs being prescribed is associated with, the degree of retardation, and the presence of autism; it is no less in the community than in inpatient institutions;[15] and is only reduced by a determined programme of rationalization.

The presence of intellectual disability colours prescribing:

1 Non-compliance usually results from an unpalatable formulation or a carer's prejudice rather than forgetfulness.

2 Coexistent disorders, as wide-ranging as epilepsy, constipation, and cerebral palsy, can make a patient more vulnerable to adverse effects.

3 Cerebral dysfunction may cause more frequent atypical responses, sensitivity being either increased or decreased to various aspects of the drug's effect. Most of these are dose-specific, paradoxical effects for therapeutic windows are frequent and, as frequently, forgotten. Prescribing details are subject to revision and should be checked with a current formulary but treatment should be started at a lower dosage and increased more gradually than is generally recommended. The unexpected should be expected so that, besides the routine warning of adverse effects, carers should be able to contact someone if they are in any doubt about the drug's effects.

4 Evidence of efficacy is largely anecdotal, with trials being mostly small, open, and uncontrolled, but providing some justification for almost any neuropharmacological adventure. The information that needs to be weighed up is more complex and is therefore less likely to be within the patient's capacity to decide whether to take it. This leaves the prescriber with a greater responsibility to put the patient's interests first.

(a) Neuroleptics

These are used frequently and for a variety of symptoms despite a shortage of consistent, demonstrable, and specific effects. For example, although a series of studies have shown haloperidol to be effective in autism, the response can be in any of a variety of areas including improved discrimination learning, a reduction in overactivity, anger, and in the frequency and intensity of outbursts.[16] A number of reports suggest violence, whether to others or self-directed, might be more responsive to fluphenazine or clozapine (although the use of the latter is severely limited by its potential for marrow toxicity).

The recent proliferation of neuroleptics has been driven by a search for greater effectiveness together with a reduced risk of adverse effects and has been steered by the theoretical clinical attributes of various neuroreceptor systems. The atypical neuroleptics are reputed to bring less risk of adverse effects such as the dyskinesias, but, as they become better known, are being linked with sedation, weight gain, and elevated prolactin level and there is growing concern about their potential to produce a metabolic syndrome.[17] The most established of these, risperidone, has shown itself effective in several random controlled trials in reducing behavioural disturbance in children and adults across a number of symptoms such as aggression, social withdrawal, inattentiveness, and overactivity.[18]

(b) Antidepressants

Depression, once noticed and identified, is as treatable as in the normal population. Obsessive–compulsive symptomatology is frequent and responsive to the drugs augmenting serotoninergic transmission although there is increasing anxiety about potential adverse effects, particularly suicide and dependence although there is, as yet, no evidence for this in the population with intellectual disability. More frequent is a paradoxical increase in anxiety that may be a partial serotonin syndrome. The use of the selective serotonin reuptake inhibitors and lithium is being extended beyond the management of apparently compulsive violence and self-injury to include bouts of non-specific distress. Clomipramine, fluvoxamine, and fluoxetine have both shown success in random controlled trials but the evidence for the effectiveness of other drugs largely consists of open label and retrospective case-series in which it can be unclear whether the underlying disorder is autism, intellectual disability, or both.[19, 20]

The serotoninergic system appears to be central to a wide number of vegetative functions suggesting a potential for these drugs in appetite disorders such as the compulsive search for food and lack of satiety of Prader–Willi syndrome.

(c) β-blockers

Propranolol and nadolol limit the autonomic response to anxiety and the propensity to panic. They have a particular place where acute anxiety underlies aggression as, for example, in someone with autism who lashes out or flees in panic when feeling crowded. They are non-sedative but can cause lethargy and even depression. Open-label series suggest propranolol may take some weeks to take effect with a dosage range of 50–960 mg/day; characteristics that may explain the lack of random controlled trials.

(d) Stimulants

There has been a widespread re-evaluation of the place of attention deficit-hyperactivity disorder (ADHD), its prevalence, and management. Well recognized in children in the normal range of ability, it is now being identified more frequently in adulthood, coexistent with autism spectrum disorder, and with intellectual disability. There is a general, unsubstantiated belief that ADHD is more intractable, the greater the degree of intellectual disability. Methylphenidate and amphetamine remain the standard treatments but amantadine might be more effective and less toxic.[21] Clonidine can be of use although sedative and short-lived and atomoxetine is finding its place. When effective, stimulants can produce a global improvement in behaviour that includes appetite, sleep, and mood, even though anorexia, insomnia, and depression head the list of potential adverse effects.

(e) Mood stabilizers

Antiepileptic drugs are being more widely used to reduce emotional lability, particularly outbursts of rage which have an organic, possibly epileptic, basis; the episodic dyscontrol syndrome. Supportive evidence is slight and occasionally the drug may make matters worse.[22] Aggression has a large variety of causes and, after the exclusion of physical discomfort, the primary approach is psychological. However, lithium has shown to be effective in reducing outbursts of aggression, particularly where there is irritability and explosiveness.[16]

(f) Opioid antagonists

The hypothesis is that opioid excess might underlie both autism and its frequent associate, an indifference to pain that encourages excessive self-stimulation. Naltrexone has been used to treat both autistic disturbance and repetitive self-injury. Where autism shows any response it is to a very low dosage (5–20 mg/day), a window in a therapeutic U-curve while, if self-injury should respond, it is likely to be to a higher dosage (100–200 mg/day). Unfortunately, many of the random controlled trials have fallen between the two dosages.

(g) Antilibidinal drugs

For a few, a strong sexual drive overrides the teaching, training, and psychological therapies which, as in other areas of psychiatry, are the main approach to sexual offending. An antilibidinal agent[23] can supplement the other approaches, reducing the drive to a level where it is under the patient's control. The sensitivity of this area, let alone potential adverse effects, makes it especially important that the patient, the family, and the carers are all part of any decision to use medication.

Conclusion

There are a large number and variety of treatments for any disorder in intellectual disability: a good indication of the complexity of disturbance and the inconsistency of the effectiveness of any one treatment. Therapists have an unusual responsibility not to exploit the limited ability of their patients to withhold consent as well as to try to see the world through their eyes. They have also to combine simultaneously the enthusiasm of the charismatic healer with the objectivity and scepticism of the scientist. As in any experiment, changes in treatment should be introduced singly to ensure that it is clear which manoeuvre produces which result. However, treatments complement each other and should not be used in isolation. This is the area above all in which the patient depends on teamwork and cooperation between therapists, disciplines, and agencies.

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10.7

Special needs of adolescents and elderly people with intellectual disability

Jane Hubert and Sheila Hollins

Introduction

Social health and mental health needs change throughout life, and this chapter highlights those particularly relevant for adolescents and elderly people. As a general rule, people with intellectual disabilities have the same needs as other members of the community, but they may also have additional needs for which they are entitled to extra support.[1]

Adolescents

Administrative prevalence of intellectual disability in adulthood vs. childhood

The UK Government White Paper 'Valuing people' estimates that there are about 2 10,000 people with severe and profound intellectual disabilities in the UK: around 65,000 children and young people, 1 20,000 adults of working age, and 25,000 older people. They estimate that there are some 1.2 million people in the UK with mild or moderate intellectual disabilities. Worldwide, it is estimated that there are some 20 million people with intellectual disabilities.[2]

Although there is now a trend in the UK towards mainstreaming children with special needs, and providing extra support, separate special schools for children with moderate and for severe learning difficulties are still provided in many places. At school leaving age, many young people with mild or moderate learning difficulties (roughly equating to IQ > 50) will not receive special services; only people who have severe intellectual disabilities, and those with additional disabilities, including epilepsy, autism, mental illness and/or behavioural problems will be referred on to adult specialist services. The administrative prevalence of adults with intellectual disabilities is thus much lower in adulthood as it is a measure of those in contact with services. The administrative prevalence rates should not be confused with true prevalence rates, which are far more difficult to assess.

Transition to adulthood

Among young people in the general population, there are certain important life events which are usually considered necessary for a successful transition to adulthood. These include getting a job or going to college, having economic and social independence from parents, and leaving home.[3] Although the transition to adulthood can be a difficult and painful process for anyone, for most people it is also a time at which choices and opportunities open up. For people with intellectual disabilities, the transition to adulthood does not usually follow the same pattern as it does for others. For many, the transition is marked simply by an abrupt move from the protective and relatively well-defined children's services to adult services, and by leaving school. These imposed transitions into adulthood are often abrupt and traumatic for the young people and for their parents.[4]

Overall, the criteria for 'successful' transition to adulthood are less likely to be fulfilled the greater the severity of the intellectual disability and other factors such as physical or mental health problems, communication difficulties and/or challenging behaviour. Those who have mental health problems, or aggressive challenging behaviour, are particularly unlikely to receive the necessary support and services to enable them to live independent 'adult' lives.[5] In 2001, a new service, Connexions, was established to improve the management of the transition to adulthood, by providing young people from 13–19 years of age with access to advice, guidance and support.[2]

In the UK, the majority of young people with intellectual disabilities attend Day Centres. Those who are more able may enter sheltered employment, workshops for disabled people or supported open employment. Relatively few people with intellectual disabilities are in paid employment, and although employment schemes are now being developed in many places, there are substantial barriers that are faced by people with intellectual disabilities in getting and maintaining employment in the open job market.[2]

In all parts of the world, the majority of young people with intellectual disabilities continue to live at home with one or both parents, and often have little or no social or economic independence, or participation in major, or even minor, life decisions. Although adolescence and leaving school imply a transition to adulthood, in many cases young people become more dependent

on their parents at this stage than they were before. For those who have severe or multiple disabilities, and/or challenging behaviour, there may be few practical alternatives and choices open to them.[6]

In different countries, various approaches have been developed to recognize the needs of young adults. One widely adopted approach, the development of small group homes in the community, has meant that more young people with intellectual disabilities are able to move away from home, even some who have severe intellectual disabilities and challenging behaviour. In many countries however, the responsibility remains firmly with carers, with institutional provision being the only backup when family care breaks down. An international carers' advocacy organization, Inclusion International, researched the views of carers in 80 or more countries in both developed and developing nations, and their report makes a number of recommendations about how communities and governments can provide better support to individuals and families.[7, 8]

Health needs

There is a high prevalence of epilepsy, psychiatric disorder, hearing and visual impairments and autism among people with intellectual disabilities. Children with intellectual disabilities are the responsibility of a paediatrician, and parents can discuss and monitor their children's needs and progress through one agency. When a child is transferred to adult services this situation changes, and there are many different agencies and individuals who become responsible for different aspects of the overall service to adolescents.

For the families concerned, the world of adult services can be bewildering. The situation is particularly problematic in relation to adolescents with severe intellectual disabilities, especially if there are also behaviour problems, during this transitional phase from child to adult services. Impairments in adaptive behaviour associated with intellectual disability lead to problems in developing normal social functioning, communication, and the ability to use community facilities. In addition, the relationship between parental and professional roles and responsibilities is often unclear. Multidisciplinary assessment is advisable, and parents should remain involved, but all too often are told that their opinion is no longer valid now their child is an adult.[2] The Royal College of Psychiatrists publishes leaflets for family carers to help them manage these changing professional relationships.[9]

It is often not apparent who, among the professionals, is directly responsible for someone in the context of services, and there may be inconsistencies between Health, Education, and Social Services in terms of policies and practice. Also, health professionals, including general practitioners, may be relatively inexperienced in dealing with people who have intellectual disabilities.

A coherent strategy for developing comprehensive health care services for young people with intellectual disabilities requires collaboration between service providers, to ensure that the health care needs of all people with intellectual disabilities, including those with autism, are properly identified, and that access to mainstream primary and secondary health care is supported. One initiative developed for the white paper 'Valuing people' was the introduction of Health Action Plans to try to address some of these information and knowledge gaps.[10]

Mental health needs

Diagnostic overshadowing of mental illness in people with intellectual disabilities was common in the past, but there is now increasing awareness and assessment of psychiatric disorders, and acceptance of dual diagnosis among people with intellectual disabilities. Although mental health needs can in some cases be met by general mental health services, some specialized mental health provision is still necessary to meet the needs of people with dual diagnosis, including those who also have challenging behaviour.[11]

Until recently, people with intellectual disabilities were seldom thought to suffer from depression, but recent research shows that adolescents with intellectual disabilities report more depression and other symptoms of psychopathology than others without intellectual disabilities.[12]

There is increasing awareness, and continuing evidence,[13,14] of the high prevalence of abuse of people with intellectual disabilities, of all ages, including emotional, physical, and sexual abuse, resulting in Post Traumatic Stress Disorder,[15] severe behavioural disorders[16] and damaging long term effects on the family as a whole.[17] Challenging behaviour in people with intellectual disabilities may be indicative of psychiatric disorders, such as psychosis, depression, and anxiety disorders.

A recent report concludes that people with intellectual disabilities who present behavioural challenges are often marginalized, stigmatized, disempowered, and excluded from mainstream society,[18] indicating the need for changes in policy and practice.

Sexual relationships, marriage, and parenthood

Long-term sexual relationships and parenting children are generally considered to be an integral part of being an adult. In adolescence, emotional and sexual interest and needs develop, and it is at this stage that most young people start to have sexual relationships. However, people with intellectual disabilities are seldom encouraged to develop sexual relationships. Parents tend to actively discourage it, and service managers and care staff, though they may not necessarily actively discourage it, often provide little opportunity, or privacy, to enable it to happen. Many people in the general population find it difficult to accept that men and women with intellectual disabilities have ordinary sexual feelings and desires, let alone that they should be allowed to act on them.[19] The argument against allowing people with intellectual disabilities to have sexual partners often involves judgments about whether someone is deemed fit to be a parent. People with intellectual disabilities are discouraged from parenthood, and the experiences of childbearing and child rearing are still usually denied to women with intellectual disabilities. In Norway, 40 per cent of a study cohort of 126 children born to parents with intellectual disabilities were found to have suffered from 'failures of care'.[20] In England, however, research has demonstrated that some people with intellectual disabilities can become successful parents, provided they are given appropriate and effective support.[21]

Cultural differences

People with intellectual disabilities from black and ethnic minorities are less likely to have their needs met by service organizations as compared with the rest of the population.[22] This is not only the result of difficulties in accessing services, and lack of appropriate

information, but also because too little attention is paid to the different social norms, beliefs and preferences of people from different cultural backgrounds.

It is vital that service planners and providers know in what ways and to what extent the belief systems of the people they provide services for coincide and/or conflict with their own. They must also be aware of the implications of these differences for the acceptability, expectations and outcomes of the services offered to people from different cultural groups.

Elderly people with intellectual disability

Life expectancy

People with intellectual disabilities have an increased risk of death compared with the general population. Whereas the majority of deaths (83 per cent) in the whole population in the UK occur in people aged 65 years and over, less than 50 per cent of deaths among people with intellectual disabilities are in this age group.[23] In a study of young people in one state in the US, the mortality rate was almost three times higher than average,[24] and in Denmark 'preventable' mortality was four times higher than average.[25] However, life span is increasing among people with intellectual disabilities,[26] especially among people with Down's Syndrome.

As a result of this increasing longevity, causes of death common in a normal ageing population are becoming more prevalent among people with intellectual disabilities, such as stroke, heart disease and cancer. The most common cause of death for people with intellectual disabilities is still respiratory disease, which occurs far more frequently than in the whole population, suggesting lack of effective care.[27] This cause of death is linked to pneumonia, swallowing and feeding problems, and gastro-oesophageal reflux disorder.

People with intellectual disabilities frequently suffer from epilepsy, and it is suggested that the mortality rate for people with epilepsy and intellectual disabilities 'may be as high as five times that of the general population'.[28] This high mortality rate is related to seizure type and frequency, rather than directly to seizures.

Health needs

Cooper[29] reviews the effects of age on the physical health of people with intellectual disabilities, and stresses the existence of significant health needs among this population. These needs arise not only from the normal ageing process but also from the specific social health and mental health needs of people with intellectual disabilities, including dementia.

There are serious problems relating to access to health care,[30] which is further complicated by their failure, and the failure of their carers, to recognize the signs and symptoms of illness. Overall, uptake of services by elderly people with intellectual disabilities is poor. There is an increased risk for a number of medical conditions in people who have Down's Syndrome,[31] including sensory impairments, thyroid disease, leukaemia and atlanto-axial instability. The later consequences of congenital heart disease include pulmonary hypertension and congestive heart failure.

Carers may not recognize that changes in behaviour are due to physical or mental illness, instead attributing changes to the learning disability itself. It is important to determine the aetiology of any learning disability, even late in life, because of the possible health implications.

People with intellectual disabilities currently access health screening less than others in the general population.[32] Pictorial health education materials are available to help health care professionals provide information about illness, medical procedures and treatment to people with limited verbal communication (for example, see www.rcpsych.ac.uk/bbw).

Signs of poor physical care among elderly people with intellectual disabilities, e.g. eye infections or tooth decay, may indicate a deterioration in functioning, but may also reflect the fact that carers are not coping effectively. This emphasizes the need to ensure access to primary care.

Recent reports from the Disability Rights Commission[33] and Mencap[34] identify the existing health inequalities faced by people with intellectual disabilities, and cite evidence suggesting the discrimination they face at every level of the health service.

Recent studies of the prevalence of mental ill-health problems among people with intellectual disabilities have shown that the prevalence rate of mental ill-health among adults with intellectual disabilities is higher than those recorded in the general population, with anxiety states and depression increasing with age.[35] People with intellectual disabilities are prone to the same risk factors as other people, but there are additional ones, such as living in more deprived areas, not having any daytime occupation, single marital status and epilepsy, all factors associated with mental ill-health. These factors need to be addressed if the existing inequality gap is not to be widened further.

People with Down's syndrome have an increased risk of developing Alzheimer's disease in middle age. Although the neuropathological changes of Alzheimer's dementia are widespread, development of clinical dementia is not inevitable.[36] Dementia occurs more frequently among elderly people with intellectual disabilities in general than among the rest of the population, and with the increasing longevity of people with intellectual disabilities the number with dementia is rising.[37]

Complexity of care needs of elderly carers and the people they care for at home

In households where an older person with intellectual disability is living with an elderly carer, there will be a complex set of individual and joint needs. Both are likely to be vulnerable at this stage in their lives, and their needs may not always be compatible.

Parents may reach a point where physical mobility and capabilities are declining, and in some cases dementia (in either), may complicate the situation. Input from services becomes essential, even if families have managed with few or no services until now. Many families will have relied on informal sources of support such as family members, friends and neighbours, but in later life these networks tend to break down, and households such as these become increasingly isolated. This isolation in the community tends to coincide with the increasing frailty of ageing carers. [38, 39] Parents of children with severe disabilities, and/or challenging behaviour, may well become isolated from kin and friends at a much earlier stage as a result of their dedicated caring role, increasing the likelihood of social isolation in later years.

Some elderly parents who come to light at this late stage will have made a decision not to accept help many years ago. This decision will have been made on the basis of information about services which were available up to 40 or 50 years ago, and elderly carers may not have received information about the range and nature

of current services. There are also some parents who originally concealed their child's disabilities in order to avoid institutionalization, and these parents may still be unaware of less institutionalized alternatives to care at home.

Systems of mutual caring often develop in households such as these, and such families may continue to be independent, in spite of serious long-term problems, and only come to the attention of the services when crisis intervention becomes necessary.

Families such as these present a double challenge to the services, requiring co-ordination between all relevant service providers. Co-operation between services for the elderly and those for people with intellectual disability is often inadequate, and some older people with intellectual disability fall into a limbo between them, with no one taking overall responsibility for assessing and meeting their needs. However, part of the brief of local Partnership Boards in the UK is to ensure that there is co-ordination between the services, so that individuals can obtain the range of services that they need.[2]

Planning for the future

Ageing parents often wish to continue to look after their adult child until they can no longer cope. If a son or daughter has mild intellectual disability this may not cause problems for the parents, and may represent a welcome continuation of family life, especially when other children leave home. Also, as parents grow older they may become increasingly dependent on their adult child with intellectual disability. This may outweigh their wish to see their son or daughter settled in a new home before they themselves die, or become too frail to care for them.

Although keeping an adult with severe disabilities at home may cause considerable hardship, many parents are unwilling to let their child go into residential care, because they believe that only they can provide the quality of care that he or she is used to.

Although separation and/or bereavement are likely to occur in the relatively near future, professionals working with families find that many elderly carers are reluctant to plan for their child's future care, and attempts to develop care plans are often fraught with anxiety.[40]

Bereavement

The experience of bereavement is often ignored by people involved with people with intellectual disabilities, although the effects of bereavement among this population are particularly severe and long-lasting, and there may be a significant increase in aberrant behaviours, and an increase in psychopathology. The experience is often made more difficult by their exclusion from the rituals and processes associated with dying and death. The onset of grief may also be delayed and thus there is a greater chance that it will not be recognized as grief, but attributed to something else or labelled as challenging behaviour.[41]

The experience of bereavement of a parent is particularly hard because, in addition to trying to come to terms with this loss, there are often other major life changes, such as moving into a strange home, living with unfamiliar people, and being cared for by a number of new carers.

Life changes in old age

Any major life changes in old age can have serious emotional and physical consequences. Life events which can trigger physical or mental deterioration include institutionalization, when caring parents die or become too frail to continue caring. This often sudden upheaval following unexpected separation or loss of a parent can be extremely traumatic.[42] Conversely, deinstitutionalization, i.e. moving into the community after a lifetime in a long-stay institution, requires complex and sympathetic planning and monitoring.[43]

Cultural differences

The implications of the lack of specific policies for older people with intellectual disability are particularly relevant to those from black and ethnic minority groups. Their needs may not be appropriately met by existing day services, but the alternative, i.e. living in residential homes in which staff and residents do not share the same background or language, may result in increased isolation in old age. Appropriate community services, which respond to different cultural preferences and expectations, have been developed in some areas of Britain. In general, however, service providers tend to focus on the issues of age and intellectual disability, rather than the cultural background of those individuals who have different expectations and preferences in the context of age and approaching death.[44]

Further information

St George's, University of London www.intellectualdisability.info Down's Syndrome Association UK http://www.downs-syndrome.org.uk

British Institute of Learning Disability www.bild.org.uk

The Department of Health – Learning Disabilities www.doh.gov.uk/learningdisabilities

Down's Syndrome Medical Interest Group www.dsmig.org.uk

The Foundation for People with Learning Disabilities www.learningdisabilities.org.uk

International Association for the Scientific Study of Intellectual Disabilities www.iassid.org

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10.8

Families with a member with intellectual disability and their needs

Ann Gath and Jane McCarthy

Introduction

It is now more than 30 years since children with intellectual disability were among the first patients to emerge from long-stay hospitals, where the mothers had been persuaded to part with the disabled children. The argument given was that not only would that child have the best possible chance of a happy life, but so also would the other children. Fears that there would be adverse effects on parents and on brothers and sisters prompted much of the early research.[1]

Assessment of effects on the family

Recent sophisticated methodology has been used to explore a variety of factors, including the family as a whole as well as the parents, and what impinges on the family such as wider social, economic, and cultural influences.[2,3] Positive adaptation and coping strategies within families were identified and are highly relevant in providing a basis for intervention. Other life events and protective or compensating influences are not ignored. Families with a disabled member are exposed to the same risk of adverse factors, such as poverty, divorce, unemployment, or mental illness as any other and, in most cases, will have the same strengths, such as humour, good friends, or staunch relatives as their neighbours. Previously, all the complaints were added together as a measurement of 'stress', a concept too amorphous to be the basis of helpful intervention.

The early impact on a family of a disabled child

Diagnosis of an abnormality now frequently happens in pregnancy from screening tests or from ultrasound scans, all of which are routinely offered. Termination of pregnancy is offered when results are positive. Although negative tests by no means guarantee normality, they are often interpreted by the parents as meaning that major disability is ruled out. Hence their disappointment when a child is born with a defect is even more intense, and it often follows a prolonged highly anxious period in which the baby is in special care. Initial hope is followed by temporary relief and then by the reality of gross developmental delay. Others believe they have a normal child until they become aware of the slowness of development or the onset of seizures occurs in the second half of the first year. Parental reaction to these tragedies is often anger mixed with grief.

A major change in recent years has been the rapid expansion of intra-vitro fertilization. Although accurate figures are not available, it is estimated that at least 10 000 babies are born in the United Kingdom. Foetal abnormalities are more common than following normal conception as evidenced from clinical reports, but there is no data available about the effect on the parents who already have had much anxiety.

The mixed feelings at the time of the initial impact

The feelings that parents experience have been likened to those of grief occurring with a sudden loss. It is a useful comparison as there is a loss. Every expectant parent daydreams about the child and the arrival of a sick or damaged baby destroys many of those dreams. Commonly the first stage is shock or a numb disbelief. The next phase is often denial, 'This cannot be happening to me', followed by anger, which may be directed against the other parent, the doctor, or God. The last two phases are constructive active adaptation, which might involve learning about the condition or joining a parents association, leading on to resolution. Unfortunately, not everyone goes through all these stages and certainly not at the same rate. The mother might still be feeling as if she is shell-shocked while the father is making contact with a particular society or support group on the Internet.

The effect of diagnosis

Down syndrome is the most common disorder recognizable at birth and known to be likely associated with intellectual disability. Despite screening in early pregnancy, the condition is still common but is now often in the child of younger parents. Genetic diagnosis of unusual children is more rapid than hitherto. Parents are almost without exception relieved by a clear diagnosis, providing an explanation of why the condition has arisen as well as an estimate of future risk. Most families are also greatly helped by meeting others with similar problems. Parent support groups for each specific diagnosis are now worldwide and recruits are quickly introduced

to information via the Internet. Some diagnoses have implications for other members of the family, as with fragile X syndrome or tuberose sclerosis, as some relatives may have a minor form or be carriers, with a risk of further children in the family being affected. Genetic counselling is essential and may be requested very early on, in pregnancy or even before conception. To be preventative, decisions have to be made fast and at times when young couples are at their most vulnerable.[4]

The effect on the parents of a child with intellectual disability

Informal support from the family or neighbours is much more effective than more formal, professionally led support. Frequent outpatient appointments where little happens are not cost-effective for the family; often the father loses money as he must take time off work and the cost of travel with a difficult baby or toddler is high. Not all can benefit from discussion groups of parents, and other mothers find a succession of home visits from a variety of professionals very disruptive. Families with active participation in religion can strengthen family ties, especially among immigrant groups, such as Hispanic people in the United States, and Indian families, where Hinduism is central to family life and where children have specific roles to play, such as that of sons in funeral rites. A child with an abnormality makes many parents look again at their fundamental beliefs, but few make lasting changes. Other families find membership of other groups (social, cultural, even sporting) supportive, provided that the family feels that they and their child are unconditionally accepted.

Family functioning with a child with intellectual disability at school

The finding concerning the greater efficacy of informal support holds true for families of children across the whole age range, from school entry to adult life. The children are enrolled into school or special preschool groups earlier than normal brothers and sisters, thus widening the informal network of friends and confidants. Conversely, policies about choice of school, and the frequent necessity for children to be sent to schools at a greater distance away from home than other children, can lead to ostracism often felt more by the mother than by the child himself. An advantage of a special school is the relatively small size, allowing personal teacher–parent association and an open-door policy to parents, who are agreeably surprised to find themselves enjoying the school years of their disabled children.

Transition to adult life

For many years, families have learnt to work in partnership with schools, and enjoyed frequent contact with teachers, face to face or thorough the progress book that goes to and from school everyday. The last few years at school are much concerned with the choice of type of further education, sometimes residential, and with encouraging independence. The process of finding a suitable and acceptable place is often described as a lottery or a battle, and is very stressful for the parents. Many have struggled through the school years, hoping that a permanent placement will be found when they come to an end. Others fear the loss of their close contact with their child, particularly if given adult rights to make choices, with which the parents do not agree. The possible outcome is one of three: independence, semi-independence, and dependence.

For those who remain at home, the other children in the family leave home as expected, leaving their disabled brother or sister in what one mother described as 'a ghetto of the middle-aged'. However, in many families, the provision of good further education programmes or day centres plus club or leisure activities can lead to liberation and more happiness for all members of the family. It is those with severe behaviour difficulties who are not accepted by further education establishments and who become increasingly frustrated and difficult to manage at home.

Other members of the family

In many countries, the family has changed markedly in the last 30 years. There are many more divorces so that the 'parents' involved in the care of a child or young person are frequently one natural parent and one step-parent. There are also many divorced mothers living alone with the young adult after all the siblings have gone. Although some have adapted very positively, others feel very lonely, particularly if there are no members of the wider family to share the care and, often more pressing, to share the worries. Grandparents are as important in families with a member with intellectual disability as they are in ordinary families, although initially grandparents can become severely affected by the grief, take sides in attributing blame, or offer unsought advice. One mother described her mother as an enormous help because 'she was always behind me in every decision I took'. When no helpful grandparent is available, an older neighbour, another member of a parents group, or a teacher at the school or day centre could provide the sort of informal support that has proved to be so valuable.

Ageing parents and ageing 'children'

The physical work of looking after a still dependent and sometimes very heavy adult takes its toll on parents. With a severely physically handicapped adult 'child', some help can be provided with people coming in to help with bathing, but few houses can be adapted to minimize lifting which may be needed many times during the day and night. Sooner or later the work gets too much, particularly for a sole parent. Parental frailty makes aggressive behaviour much more frightening and potentially dangerous. Where there are few opportunities for outside contact, the adult, with intellectual disability complicated by severe behaviour disorder, can become possessive and may show jealousy, sometimes making visits even from grandchildren impossible. Despite the evident difficulties, a study in Wisconsin,[5] found that many parents, reaching the end of their lives after many years of looking after a disabled child, were fitter than others of the same age and had a much greater sense of having achieved something in life. Other studies of ageing people with intellectual disability have also shown the role reversal that occurs when the adult child for whom they have cared so long tenderly looks after a very old parent. Because of the increasing longevity of disabled adults as well as of older people in the general population, there are increasing numbers of very old frail parents left with a disabled offspring for whom they feel responsible and for whom they often feel anxiety about the future, commonly saying 'I always thought he would go before us'.

Brothers and sisters

The well-being of the family members continues to be an area of interest with an emphasis on siblings.[3, 6] The initial decision had

been made not just for the sake of the child with intellectual disability, but also in the belief that it was also in the interest of the other brothers and sisters, but it was possible that they who would pay the price. There is now a considerable body of literature confirming the early findings that siblings are by no means invariably damaged. Thirty years ago, there was evidence that the older girls in the families did suffer, or were difficult or distressed at school while having more than usual amounts of responsibility at home. As services improved, these findings were no longer replicated except in those countries with few facilities and many social problems. In general, the other children in the family have identified themselves with their parents' decisions and take some part in the caring. This 'assistant' parent role comes easily to older siblings, but younger siblings who grow up fast, first catch up with the disabled sibling and then overtake in development terms and in the privileges earned by greater maturity. Parents describe this period of catching up and gradual overtaking as one of the most difficult in bringing up their children because of rivalry or jealousy. However, subsequent interviews with parents show that they are as sensitive to the needs of their 'normal' children as to those of the disabled child, and the balance between the siblings is readjusted. There is little evidence of long-term damage, but on the contrary, a consistent finding that the brothers and sisters are drawn to the caring professions, particularly medicine, nursing, or special needs teaching. The majority of families with children with intellectual disability are ordinary families with 'one feature in common'.[7]

Mental illness

In the early months following the birth of a child recognized as having a major developmental disorder, such as Down syndrome, there was clearly much distress and disappointment, but little evidence that the mothers had a higher incidence of postpartum psychiatric disorder. Later in the childhood of the affected child, particularly in families with many other problems, depression was more common in the mothers of children with Down syndrome than in mothers of normal children. But when the mothers of children with a variety of disorders all producing intellectual deficit were compared with mothers of children with Down syndrome, there were less reported health problems in the Down group. Children with brain damage and severe hyperkinesis and those with autism were rated as the most stressful. Hyperkinesis and autism both occur in Down syndrome and their families report a similar degree of stress, as recorded by the questionnaire. For all families with a disabled child, many of the same factors appeared to be protective, for instance a good relationship with a partner and, in addition, support and affection from female relatives like the woman's mother or sisters. There were professionally led groups, assigned social workers, and parent–teacher associations at the school, but the informal sources of support were consistently more effective than formal, with studies in the United States showing very similar results to those in the United Kingdom.

The mental heath, composition, social background, and functioning of the family can increase the risk of psychopathology in the child with intellectual disability as can these risk factors for all young people.[8] There is no evidence that severe psychiatric illness is more common among the families of people with intellectual disability than in anyone else. However, the combination of a severe mental illness, such as bipolar disorder coexisting in the same family with intellectual disability is overwhelming for any family, particularly if one person has the dual diagnosis.

People with intellectual disability who become parents themselves

Although sexuality and pregnancy is a fear of many parents of severely intellectually disabled adolescents, their fertility appears to be very low and there are very few pregnancies in people who are totally dependent. The majority of people identified as having mild retardation during the period of education disappear from services when they leave school and so it is not possible to estimate how many women with mild or borderline retardation become mothers. However, a certain number do come before the family courts or are already known to services for other reasons. However, problems arise with planning ahead and the constant protection from danger that young babies require. There are now techniques to help teach these skills. The secret of success in such teaching is a positive attitude of enhancing skills and not one of undermining the mother. With a partner who is both stable and more able, many quite limited young women cope. As the children grow older, the problems increase as the balance between protection and encouraging new skills becomes more difficult. However, intellectual limitation in itself is not an absolute bar to parenthood. Sadly, many young women with difficulties in intellectual and emotional immaturity are likely to find partners with even more problems and have, for example, a high risk of being hurt by a violent man and of failing to protect children from similar abuse. It is problems such as these rather than the intellectual deficit that make the courts question the safety of the children.

International perspective

The very many worldwide studies that exist show a remarkable consistency in their findings. For all families of whatever ethnic origin, economic status, or religious persuasion, there is grief at the birth of a child, who is in any way defective, and anxiety and sadness about a child who later is seen to fail. In some cultures, an affected boy is harder to bear than an affected girl, as boys have special roles, for instance taking part in the funeral of the parents. Obviously, a high infant mortality will mean an even higher rate in children with any sort of disability. There are a few studies that have come from countries in an early state of development. The authors of these papers are anxious not to repeat what they understandably see as the mistakes of Europe and America. There are for example excellent community services in Asian countries based on the strengths and the beliefs of the local people,[9] whereas others model their services on those in the West, and thus have similar problems but cannot reach a significant proportion of the population. Other countries in Europe are struggling to establish new services for children at the same time as they deal with the very many older people who have been poorly treated for many years. The changes are difficult for the families of these older ones, yet even after many years, families have cooperated with rehabilitation and, in some cases, taken the adult 'child' home.

Needs and priorities

Today in the United Kingdom the social and economic needs of these families are often unmet with a significant number of families with a disabled child living in poverty.[10] Most families will agree that the needs of the disabled member should be given priority—provided that other members of the family are in good

agreement. The needs concerning the best possible communication and training or appropriate treatment for behaviour problems are very much in the interests of other members of the family. Hence the needs of the others would include:

- education that supports the development of the child and the needs of the family
- as accurate a diagnosis as possible
- genetic advice to other members of the family likely to produce children
- to be treated as informed partners by therapists and teachers
- available and interested primary health care
- a key worker to coordinate access to the different agencies and further financial support
- informed specialist care within reasonable distance
- advice and help to 'get through' to the child should communication be a problem, with the chance to learn sign language, symbols, or computer aids
- specialist and domiciliary help with behaviour problems
- respite care, arranged in partnership with the family
- when things go wrong, support and if necessary psychiatric care that treats the other members of the family as whole people with many other facets and not stereotyped family members of a disabled child.

Conclusion

Having a child with intellectual disability is a major and usually totally unexpected blow to any family. However, most families show great resourcefulness and adapt to give their normal child as well as themselves a happy, rewarding life. Parents strongly resent being treated as potential psychiatric patients and have vigorously thrown out the concept of 'the handicapped family'. They do suffer understandable grief. From the point of discharge, the encouragement of informal support is more useful than providing hospital-based services. Children with all sorts of disability go to school early and the provision of unobtrusive familiar services is helpful. Unfortunately, there is often a gap in services between children's services and those for older adolescents and adults. The gap occurs at the worst time for parents who of all times require a familiar knowledgeable person who can offer a service throughout the transition period. The services required by the parents are practical help, such as appropriate equipment, respite care, advice about behaviour, and the ability to find emergency or specialized help at short notice. Parents also require some notice to be taken of their increasing age and/or infirmity, the financial difficulties arising out of the disability, and their anxiety that a humane plan can be made for their son or daughter when they die.

Further information

www.downs-syndrome.org.uk
www.fragilex.org.uk

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10.9

The planning and provision of psychiatric services for adults with intellectual disability

Nick Bouras and Geraldine Holt

Introduction

The functioning of people with intellectual disability (ID) is affected by many factors. As well as their ID, their ability to communicate with others, their social competency, personality, life experiences and circumstances, and their health (including mental health) also influence their behaviour and adjustment.

This chapter focuses on the development and provision of services for adults with ID who have additional psychiatric and behavioural disorders. Developments have taken place in various parts of the world in recent years and a wide range of services has emerged.

History and concepts

In the mid-nineteenth century the conceptualizations of the needs of people with ID and of those with mental illness, and of how to meet these needs were separated. Intellectual disability was not then included in psychiatric training curricula and generations of psychiatrists did not see people with ID, apart from those involved with administrative functions or the prescription of psychotropic medications in institutions. The mental health needs of those with ID at this time were largely unrecognized and so ignored.

Ideologies, sociological theories, civil rights issues, and the normalization philosophy[1,2] together with families' organizations inspired current care practices and directed the way ID services developed.

Policy initiatives originating in the United States during the 1960s and 1970s produced profound and far-reaching changes offering the integration of people with ID into mainstream community life. Similar policies were adopted gradually around the world, particularly in North America, Europe, and Australasia, and in several countries the number of people with ID remaining in institutions has been drastically decreased. Deinstitutionalization of people with ID has been probably the largest social policy experiment of our time. Vivid accounts have been published recently offering enlightening narratives from individuals who were resettled in community living.[3] Overall people with ID and their families have benefited, having a better quality of life. Nevertheless, there are significant variations in the quality of community-based services and of the experiences of people who use them.[4]

Psychiatric disorders and ID (dual diagnosis)

Many service planners and providers assumed that psychiatric disorders in this population would substantially diminish when community care programmes had been put in place. With the implementation, however, of the deinstitutionalization process the need for services for people with ID and psychiatric disorders emerged as a major issue.

This is because a significant number of people with ID, 5 to 12 per cent of children[5] and 15.7 to 40.9 per cent of adults with ID[6] have psychiatric disorders and despite progress in care delivery systems, require appropriate input to manage their mental health needs, sometimes over considerable time. Behavioural or psychiatric disorders can impair people's quality of life, cause regression of adaptive and intellectual functioning, and create unnecessary escalation of family stresses.

The presence of severe behaviour or psychiatric disorders in people with ID is one of the main reasons for the breakdown of community placements and of retention in residential environments that are more restrictive than otherwise required. Such people are at risk of being placed in out of area facilities[7] if local resources are not adequate to meet their assessment and treatment needs or ongoing support needs. These placements are often expensive and divert resources from developing local initiatives. The care provided may be inadequate and difficult to monitor. People may lose contact with families, friends, and those people and structures that previously supported them.

It has become clear that people with ID and mental health problems need services from both the ID network and the mental health system. The overall position of governmental policy has been that people with ID should have access to generic (i.e. for anyone with or without ID) health services, but with additional specialist (specifically for people with ID) support when needed.[8,9]

The argument for the provision of mental health care for people with ID from generic services appears sound and is supported widely.[10] Some argue that specialized services lead to stigmatization,

labelling, and negative professional attitudes. Others argue that special expertise is required for the diagnosis and treatment of psychiatric disorders in this population, because although it is theoretically possible to train staff in generic settings, the relatively small number of cases gives little opportunity for staff to gain or maintain the necessary skills.[11]

Problems arise particularly when admissions to adult acute inpatient units occur, as people with ID often require longer admissions, and may be vulnerable without additional support on the ward. Furthermore, people with ID represent a very heterogeneous group with a varied range of highly complex mental health needs which generic staff may feel ill equipped to meet.[10]

Menolascino[12] recommended that services be provided according to need and be delivered in the context of both ID and psychiatric disorders coexisting allowing for more appropriate treatment, support, service planning, and development. The result is to create a partnership between the mental health and ID service structures to ensure responsive supports and treatments to previously underserved individuals.

Models of services for people with psychiatric disorders and ID

There has been a growing interest internationally as to how to address this issue. Davidson and O'Hara[13] offer a comprehensive review of service developments for this population. Long-term resolution of behavioural or psychiatric disorders in persons with ID requires community-based activities. Hence since the year of publication of the first edition of the *New Oxford Textbook of Psychiatry* new developments in most countries of the world are community-based. The pace and form of change depends on each country's unique historical perspective and national philosophies about care for people with ID.[14] However, resolution of an acute crisis may require, in addition to community-based psychiatric or behavioural resources, inpatient acute psychiatric assessment and treatment services, specialized outreach, emergency respite, or emergency behaviour stabilization services.

The most common models of services for adults with ID and psychiatric disorders that have emerged in recent years in the United Kingdom can be described as: (a) generic ID community-based multidisciplinary (interdisciplinary) teams, (b) specialist community-based mental service for people with ID.

Generic ID community-based multidisciplinary (interdisciplinary) teams

A multidisciplinary (interdisciplinary) team offers assessment and specialist services to people with ID. Initially, most of these teams were involved with deinstitutionalization, carrying out tasks such as identifying appropriately adapted and staffed houses, matching clients to live together, assessing health and social needs, and so on. Most of them have input from clinical psychologists and usually some input from a psychiatrist specializing in people with ID. Some teams have developed innovative ways of working with people with challenging behaviour often with severe ID. Members specializing in functional analysis and/or behavioural treatments strengthen such teams.

One considerable problem with this model has been the lack of links with mainstream mental health services. Despite the psychiatric input, such services may experience difficulties in meeting the mental health needs of people particularly those with mild ID and mental illness. The problems are extended to people with ID who may have additional forensic mental health problems, autistic spectrum disorders including Asperger's syndrome and co-morbid conditions as well as those with borderline intellectual functioning.

Specialist mental health service for people with ID

Since 1982, the Community Mental Health in ID Service in South East London[10,15] has operated using this model. It has secondary and tertiary care functions. This Service includes outpatient clinics, outreach work, inpatient assessment and treatment, and consultation with community agencies. The clinical team comprises of psychiatrists, community psychiatric nurses, and administrative staff, and has a regular interface with clinical psychologists and behaviour support specialists. The clinical team also receives regular input from occupational therapists, speech therapists, and social workers. The composition and functions of the Service have evolved over a number of years. An integrated part of the Service is the provision of training to direct support care staff and others to promote and sustain the development of a competent workforce at every level, from direct care staff to managers and organizations.

There are three phases in providing clinical services: assessment, intervention, and follow-up.

The clinical team carries out a structured clinical assessment on all referrals with the additional application of standardized instruments, e.g. Aberrant Behaviour Checklist[16] and CANDID.[17]

Therapeutic interventions are based on multidisciplinary work and include medication and environmental manipulation, as well as psychological treatments such as anxiety management and cognitive behaviour therapy. Regular weekly clinical team meetings are held to review progress. Crisis prevention plans are developed to help families and service providers identify early signs of breakdown and to take appropriate action. Training is offered to improve the capacity of families and service providers, to better understand and respond to the mental health needs of people with ID. This includes seminars, books and videos as well as modelling and role-playing exercises. Ongoing support and consultation is also provided while other specific therapeutic interventions are implemented.

Follow-up is provided for as long as it is required. Once a client seems stable and the agreed upon strategy appears to be effective the team maintains quarterly or half yearly contacts.

If an inpatient stay is warranted for acute psychiatric crises, admission is into generic mental health facilities with consultative advice and support from the community-based team. Patients can also access a six-bed specialist unit at a tertiary level. The function of this unit is to provide comprehensive assessment of the mental health problems when this cannot be achieved in a community setting or within generic mental health services, to make recommendations and implement therapeutic interventions and to ensure the appropriate care plans are transferred to the community setting on discharge. Care is delivered and coordinated via a person centred, Care Programme Approach (CPA),[18,19] to help ensure effective links with the full range of psychiatric health and social care services.

This Service is compatible with the development of other specialist services in the United Kingdom over the last few years to address specific needs for example of children and adolescents, older adults, those with forensic problems, mothers and babies, those with

eating disorders, home treatment teams, assertive community treatment services, eating disorders teams, early intervention teams for psychosis, etc.[20]

Outcomes

Evaluation and measuring of outcomes in mental health care for people with ID is very complex. This is because most health care service developments and reforms are politically and socially driven rather than evidence led and researchers cannot embargo change until they have defined systems. Accumulating evidence from chronological studies will still require judgement and interpretation.[20]

Moss *et al.*[21] considered a variation of the Matrix Model, first described for non-disabled people with mental health problems by Thornicroft and Tansella,[22] for the evaluation of mental health services for people with ID. This consists of two dimensions, one determined by the level within the service system (i.e. national, local, or individual), and the other by the point in the temporal sequence of service provision (i.e. inputs to the service, the process of providing the service, and the resulting outcome). Bouras *et al.*[15] adopted the Matrix Model partially (inputs and processes) to evaluate their model of service and found that over 18 years statistically significant changes in referrals trends in ethnicity, type of residence, level of ID, the number of admissions to inpatient units and psychiatric diagnoses. In addition they also found that patients admitted to the specialist unit—in contrast to those admitted to a generic inpatient unit—showed a significant decrease in psychiatric symptoms, an increase in overall level of functioning, a reduction in severity of their mental health problems, and an improvement in behavioural function on discharge, at 6 and 12 months following discharge.[23]

In an attempt to compare the effectiveness of assertive and standard community treatment in people with psychotic spectrum disorders and ID, with a randomized controlled study, no significant differences were found between the two treatments.[24]

Clinical effectiveness studies in mental health care for people with ID still have to overcome important methodological limitations. At present the Matrix Model[21] seems to offer the most advantageous way of evaluation, providing a framework to conceptualize the factors that influence service developments in the field.

Residential programmes for people with psychiatric and ID

Successful community-living opportunities for people with ID require a comprehensive and collaborative service structure, including appropriate residential and vocational facilities. However, whilst these have been developed for many people, services to meet the needs of those with psychiatric disorders have lagged behind.

Housing for people with mental health problems must be compatible with all the main principles of 'ordinary housing'. It should be located in an acceptable community setting that offers opportunities for community integration, be designed to provide services and supports to meet the needs and desires of the person residing there, and be affordable, safe, and comfortable. This requires that staff have the necessary skills and service structures to meet client needs.

As institutions have been closed residential facilities have been developed in their stead. The trend across North America, Europe, and Australasia has been for larger residential homes (sometimes on the sites of the old institutions) to be replaced by smaller group homes for 3–8 people supported by staff. More recently, 'supported living schemes' have become more common, where people rent or own the property, and receive support from agencies that do not control the accommodation.[4] The pace of change varies between and within countries.

The aim is to empower individuals in smaller settings, organized to respond to a wide range of needs, creating environments that promote physical and mental health. However, no one model will necessarily meet the needs of all individuals with mental health problems and ID. Some people may become isolated and lonely in one or two-person settings, or have difficulties that cannot be managed in housing where additional staff or clinical support is not readily available. Some people may simply prefer to live in a supervised group living situation rather than supported living and should be given the opportunity to live in a place they prefer.

Residential services should include a full range of alternatives to enhance an individual's capacity for community living. The individual receiving residential services should be allowed to have as much comfort, ownership, and autonomy as possible. Housing can offer a wide range of options, and maximize opportunities for community integration and personal independence. Specialist mental health services for people with ID should work in collaboration with residential providers, to provide clinical support and a safety net when difficulties arise. Delivery of services in this manner represents one of the most important organizational challenges for services for people with mental health problems and ID.

Vocational programmes

Vocational services should also offer work in integrated settings in a person's community, opportunities, and supports that are manageable and productive for the worker and the workplace with adequate salary compensation.

There have been significant changes in employment and vocational services for people with disabilities and several have moved from traditional workshop settings to integrated supported employment. The majority of placements have been in the service sector consistent with shifts towards entry and low-skill jobs in the national employment market. Individual placement has had the greatest positive effect on wages. Supported employment enhances the quality of life of people with ID. Although there is an acceptance in society that people with mild levels of disabilities can be meaningfully employed, traditional views of the capabilities of people with severe disabilities continue to be major obstacles to their access to the most progressive contemporary, educational, and rehabilitation practices. People with mental health problems and ID may be under-represented in both the sheltered and supported employment workforces.

Staffing issues and training

The availability of specialist training varies markedly between countries, and not surprisingly bears a close relationship to the level of service development.

In the United Kingdom the need for specialist mental health services for people with ID and psychiatric disorders was recognized in the early 1970s. Specialist training programmes for psychiatrists, nurses, and other health care professionals including family doctors,

community nurses, and direct care staff have been developed. However, whilst such training is available its uptake is dependent on the interests of individuals or of their employees. Only for some is such training mandatory, e.g. psychiatrists.[25] Attention has focused in recent years on the training needs of first-level care workers in community day and residential facilities. They often receive little or no training in the psychiatric aspects of ID with the consequence that psychiatric illness amongst their clients frequently goes unrecognized and untreated.

Benefits of training

Staff finds working with people with ID and mental health problems stressful. Giving them skills in this area so that they can manage, with support, people with mental health problems enables them to find this work more rewarding. The most basic and vital role of support staff in this context is the awareness that a person with ID may suffer a mental illness, as we all may. They need to be aware of the range of therapeutic options that might be helpful, including environmental changes, behavioural strategies, psychotherapeutic techniques, medication, and so on. A fuller knowledge and consideration of this topic will help to dispel myths and prejudices, for example that medication is to be avoided at all costs, or that its use signifies that staff has in some way failed the client. Specific knowledge about some disorders will provide insights into why and how interventions must be tailored around someone's strengths and needs, for example someone with autistic spectrum disorder may hit himself when his routine is changed. The intervention chosen may be to provide a timetable, which the staff and client follow. This may need to be in pictorial form to meet the client's communication needs, and small and durable enough for him to carry at all times.

Training materials

Flexible training materials (e.g. The Training Package in the Mental Health of Learning Disabilities),[26] which can be used by staff groups in their own settings, are now available. It is often useful to design training around particular clients. Training should be a part of the culture of an organization. Including managers in training activities is helpful. It allows them to share a knowledge base with their staff, and to set-up processes, which facilitate the continued development of issues identified by the training. For instance, each client's mental health might be considered in his or her individual planning meeting. Actions agreed can then be regularly discussed in individual staff supervision, at staff groups, and at meetings with the mental health and multidisciplinary teams. Raising awareness on mental health issues for people with ID is also important for carers and families as they are a pivotal source of support.[27]

Commissioning services

The deinstitutionalization of people with ID in the United States, United Kingdom, and other parts of the world is well-advanced. A variety of service models are provided. Comprehensive local services systems for those with additional mental health needs are emerging. The old institutions represented a complete system of care, inasmuch as they provided accommodation, health care, social care, and occupation in a single setting. Current provision, by contrast, involves a range of agencies and settings. This requires that care is integrated and organized around an individual. This is not an easy task for those with complex needs.

Those commissioning services need to determine what services are needed locally and decide how they should be provided, monitored, and reviewed. This chapter provides an overview of the social and policy context and some models of services for adults with psychiatric disorders and ID. Local demographics and resources will of necessity shape services. To ensure that the commissioning of services is well-informed all planning partners should be involved. Using the Matrix Model[21] described earlier the commissioners might consider:

1 Joint commissioning. Various policies and legislation (**national inputs**) have proposed joint commissioning by health and social care services so that a joint strategy drives the joint commitment of resources (**local inputs**).
2 Client and carer participation (**individual inputs**). This is essential to ensure that appropriate priorities are set and that services are satisfactorily delivered (**individual and local processes and outcomes**).
3 Involvement of statutory and voluntary agencies (**local inputs**). This will enable the commissioners to make informed decisions.
4 A baseline needs assessment of the population to be served (**local inputs**) e.g. from census statistics, epidemiological research, local register data.
5 Local and national policies (**local and national input**) to enable them develop a vision, e.g. community-based services, mixed economy of provision.
6 Desired **outcomes at local level** (e.g. increased use of generic provision, reduction in number of people placed out of area) and translate these into
7 Service specifications
8 Purchase services, which have the necessary skills (**local and possibly national inputs,** e.g. for people with very particular needs) to deliver processes (**local and possibly national processes**) that will provide these outcomes.
9 Set in place monitoring systems, which may include individual and local outcomes e.g., complaints and incidents monitoring, scrutiny of statistics derived from CPA documentation (**individual and local processes and outcomes**).
10 Commissioning is a cyclical process and the monitoring and review of services (**local inputs**) will ideally enable more effective future commissioning.

Components of an effective psychiatric service for people with ID should include:

- Organizing services around clients' wishes and needs.
- Good interagency communication among health, social, and voluntary sectors.
- Good interagency communication between services for children and adults.
- High level of awareness of mental health issues by direct support staff in residential and day care services.
- High level of awareness of mental health issues by primary care staff.

- Multidisciplinary composition including psychiatrists, mental health nurses, clinical psychologists, behaviour support specialists, therapists, and social workers.
- Ability to provide consultation, assessment, and treatment.
- Provision of community-based interventions.
- Access to local specialist and generic community and inpatient assessment, treatment, forensic, and rehabilitation facilities.
- Resources to meet residential, recreational, and vocational needs of those with enduring needs.
- Clear coordination of inputs by a named person.
- Staff training.
- Measuring outcomes.

Conclusion

There has been considerable debate as to whether specialized mental health service for people with ID services should be established or generic mental health service providers should serve this population. Whatever strategy is undertaken it should be based on high professional standards. Standardized diagnostic and assessment tools should be used. Appropriate, individually tailored treatments should be given in the least restrictive environments. Staff must have the necessary expertise, training, and support.

Mixed results have been obtained for the evaluation of existing services.[28] No direct studies of comparative treatment effectiveness exist, and studies on single specialist services contain some methodological weaknesses. It is essential that the quality of services is monitored, to maintain and improve standards of care. Increasing fragmentation of provision makes this a complex but essential task.

Often mental health services for people with ID are provided in a crisis. This highlights and amplifies the existing deficiencies in care. In order to become more effective and accessible these services will have to address both individual needs and service systems complexities for children, adolescents, and adults with ID and mental health problems. Several years into the post-institutional period and the era of community care, meeting the mental health needs of people with ID remains a challenge. Though the initial philosophical concern about the coexistence of mental health problems in people with ID has been partially eroded with the emerged evidence-based research, nevertheless the clinically effective responses remain scanty. In times of hard fiscal constraints for mental health services in general, it is hoped that people with ID will not be further overlooked and marginalized but they will receive the required professional attention matched by adequate distribution of resources.

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SECTION 11

Forensic Psychiatry

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11.1

General principles of law relating to people with mental disorder

Michael Gunn and Kay Wheat

Introduction

This chapter provides a scheme for assisting in the analysis of two areas of law that provide some of the general principles that operate in relation to mentally disordered offenders. These two areas are (a) the law concerning decision-making and other action-taking to which the concept of competence is crucial, and (b) the law of responsibility in relation to liability for criminal offences and the tort of negligence. Whilst the focus of the chapter is on the law of England and Wales, it is clear that there are similarities in other common-law jurisdictions, and in other jurisdictions that have borrowed ideas from common-law jurisdiction, such as Japan, in relation to the concept of informed consent.

Decision-making and action-taking law and competence

Generally, the law in relation to decision-making and action-taking might take one of three approaches to mentally abnormal offenders.

- The law might adopt the same approach for mentally abnormal offenders as for anyone else.
- The law might adopt an approach dependent upon the competence of the individual that might be affected by the mental state of the mentally abnormal offender.
- The law might adopt an approach recognizing the impact of being a mentally abnormal offender that may be based upon the effects or mere status of the mental state.

  There is no reason to examine further the law that is not different for the mentally disordered.

Autonomy and Competence

The most appropriate approach that introduces different law is by reliance upon competence or capacity. Internationally, there is increasing acceptance that, where someone is incompetent to make their own decisions, there must be a route to making such decisions on their behalf. For example, in Japan, the approach of informed consent has been adopted and whilst there is not yet a fully developed concept of competence, it is accepted that that is the next necessary development. Where there is significant variation is in the approach to adopt if someone is not competent. Traditionally, the approach has been to adopt guardianship whereby someone is either under guardianship or not and if so that all decisions are taken by the guardian. More recently, a more varied approach has become preferred whereby the decisions taken by others are only those that the individual cannot take and the basis for taking those decisions is the decision that the incapacitous would have made if competent and otherwise the decision that is in their best interests. It must be accepted however that this may be viewed as an approach grounded in a particular approach to law and ethics, i.e. that grounded in Western societies. Even there, there is a tradition for making decisions on a paternalistic basis, that is largely now discredited, though there are also concerns about the focus on autonomy. However, in other societies much greater emphasis is placed on the importance of the family as decision-makers or the basis of a decision on the presumption that the individual is a part of a particular society that has a genuine and proper interests in decision to be made on their behalf. Having recognized that as an approach, this chapter will largely focus upon the Western legal systems' basis, using England and Wales as an illustrative jurisdiction.

Increasingly, there is recognition internationally that action should only be taken with regard to a person if either they are incapable of deciding or acting for themselves or if they present a harm to others (or self) and that harm is linked with a mental health problem. The extent to which different jurisdictions have a developed view of respect for the principle of autonomy and its legal application through a test for capacity or competency unsurprisingly varies in practice. But the major issue is how decisions are to be made if someone is incapable.

International statements of principle

Acceptance of respect for the principle of autonomy can be seen through at least two international instruments. Nascently, it can be identified in the provisions of the *United Nations* Declaration on the Rights of Mentally Retarded Persons (1971) which includes the following commitments.

1 The mentally retarded person has, to the maximum degree of feasibility, the same rights as other human beings.

2 The mentally retarded person has a right to proper medical care and physical therapy and to such education, training, rehabilitation, and guidance as will enable him to develop his ability and maximum potential. . . .

5 The mentally retarded person has a right to a qualified guardian when this is required to protect his personal well-being and interests.

6 The mentally retarded person has a right to protection from exploitation, abuse, and degrading treatment. . . .

7 Whenever mentally retarded persons are unable, because of the severity of their handicap, to exercise all their rights in a meaningful way or it should become necessary to restrict or deny some or all of these rights, the procedure used for that restriction or denial of rights must contain proper legal safeguards against every form of abuse. This procedure must be based on an evaluation of the social capability of the mentally retarded person by qualified experts and must be subject to periodic review and to the right of appeal to higher authorities.

More recently, the *Council of Europe* has agreed a set of recommendations that should be implemented across Europe and are attracting significant international attention, e.g. by the South African Law Commission. The *Principles Concerning the Legal Protection of Incapable Adults* (Council of Europe, 1999). As Jansen demonstrates, the Recommendation confirms the functional approach to capacity and seeks to provide the incapable adult, where necessary, with representation, assistance, measures of protection, and other arrangements. The Recommendation opens with a statement that underpins the general approach adopted in this chapter:

> 'I.1. The following principles apply to the protection of adults who, by reason of an impairment or insufficiency of their personal faculties, are incapable of making, in an autonomous way, decisions concerning any or all of their personal or economic affairs, or understanding, expressing or acting upon such decisions, and who consequently cannot protect their interests.'

This is followed up by an important statement that captures an underlying theme for most jurisdictions endeavouring to provide suitable approaches.

> 'II.1 In relation to the protection of incapable adults the fundamental principle, underlying all the other principles, is respect for the dignity of each person as a human being. The laws, procedures, and practices relating to the protection of incapable adults shall be based on respect for their human rights and fundamental freedoms, taking into account any qualifications on those rights contained in the relevant international legal instruments.'

The principles adopted are then:

- securing the maximum preservation of capacity that demands a functional approach to capacity and so not accepting that someone is either capable or not capable for all decisions, but may be able to make some decisions and not others (II.3.1) and that no step should be taken unless it is necessary (II.5.1).
- where steps are taken they must be proportional to the degree of capacity retained and they should be tailored to the needs and circumstances of the incapacitous person (II.6.1).
- there just be fair and efficient procedures for the taking of steps which must protect human rights and prevent possible abuses (II.7. 1 & 2). These requirements are expanded upon in Part III.
- the interests and welfare of the incapacitous person are the paramount consideration, thus ruling out a paternalistic basis for the taking of steps (II.8.1).
- the past and present wishes and feelings of the incapacitous person should be ascertained as far as possible, and should be taken into account and given due respect, and of most importance are the choices made by the incapacitous person themselves (II.9. 1 & 2).
- there is a preference for action taken without the intervention of a judicial or administrative authority but that such powers must be limited and their exercise controlled (IV.18.1)
- in the health field, no action should be taken if someone is capable of making the decision (V.22.1). The intervention may then be carried out if it is for the incapacitous person's direct benefit and authorization has been given by their representative or by an authority or person or body provided for by law (V.22.2). As not all jurisdictions are ready for this approach even in Europe, an alternative is provided so that where a person is under protective steps, the incapacitous person's consent should be sought even though there is someone with the power to make the decision (V.23.1). Where the incapacitous person cannot provide consent, the intervention is permissible where it is for their direct benefit and authorization has been given by their representative or by an authority or person or body provided for by law (V.23.2).

For Jansen, the key principles are, first, those in Principle 5, that is '*Necessity* and *Subsidiarity*' as they 'imply, first of all, that no measure of protection should be established unless it is necessary, taking into account the circumstances of the particular case. Secondly, in deciding whether a measure is necessary, account should be taken of any less formal arrangements which might be provided in particular by family members, or by public authorities or other means. The latter is the principle known as 'subsidiarity' . . ..' The second key principle is, that in Principle 3, that is 'that of maximum preservation of capacity . . .. In particular a measure of protection should therefore not result in an automatic complete removal of legal capacity.' The third key principle is that in Principle 6, that is 'Proportionality: where a measure of protection is necessary it should be proportional to the degree of capacity of the person concerned and tailored to the individual circumstances of the case. The measure should restrict the legal capacity, rights and freedoms of the adult by the minimum which is consistent with achieving the purpose of the intervention.'

The international picture is completed by the Convention on the Rights of Persons with Disabilities that was signed in 2006 but is not yet in force. This is a Convention of the United Nations and has 129 States as signatories to it. The Convention is a broad Convention and covers many areas not directly relevant to this Chapter. It takes a similar approach since, its General Principles (art. 3) are (a) respect for inherent dignity, individual autonomy including the freedom to make one's own choices, and independence of persons; (b) non-discrimination; (c) full and effective participation and inclusion in society; (d) respect for difference and acceptance of persons with disabilities as part of human diversity

and humanity; (e) equality of opportunity; (f) accessibility; (g) equality between men and women; and (h) respect for the evolving capacities of children with disabilities and respect for the right of children with disabilities to preserve their identities. In, for example, outlawing discrimination on the basis of disability (art. 4) and providing for freedom from exploitation, violence and abuse (art. 16), the Convention identifies the balance to be drawn between recognising the importance of decision-making with that of protecting those not capable of making their own decisions. It affirms the importance of the capacity to make decisions as a key requirement in the law. Article 12 states that State Parties reaffirm that persons with disabilities have the right to recognition everywhere as persons before the law and that State parties shall recognise that persons with disabilities enjoy legal capacity on an equal basis with others in all aspects of life.

Tests of capacity and competence

Thus, it can be seen that key to respect for the principle of autonomy is to have a workable concept of capacity or competence. The functional approach requires that the test of competence be related to the particular decision to be made, at the particular time that it must be made. There is a range of abilities that competence might involve. Much of the work on competence has been undertaken in the context of health-care law and in relation to consent to treatment. Much of this work has been undertaken in the United States. Two leading thinkers, Grisso and Appelbaum, have, with colleagues, identified four abilities that can be involved in competency:

- evidencing a choice
- understanding
- appreciation
- reasoning or rationality.

Any given jurisdiction will adopt one or more of these abilities[19] in what it looks for in relation to competency assessments. There is no consistency, currently, as to which one or more of the abilities must be satisfied, except to say that almost all jurisdictions require understanding to some degree. This lack of consistency reflects the developing international understanding of the concept of competence. If we take English health-care law as an example, it can be seen that, in the early stages, understanding was the prime ability that had to be established, though the patient also had to evidence a choice. But, more recently, it seems that the courts are being attracted to an approach that may ultimately see competence only being satisfied where all four abilities are satisfied. Requiring rather more of an individual to satisfy the requirement of competence may be regarded as a better means of satisfying the crucial bioethical principle of self-determination or respect for the principle of autonomy, since if someone is not truly able to exercise self-determination, there is no respect for autonomy if, nevertheless, that person's 'decisions' are legally binding. This means that rather more 'decisions' are open to the challenge on the basis that they are not made by someone competent to do so. A stringent approach to competence may be hard to accept. It must then be assessed (as a general matter) whether it would be better to reduce the standard and so enable more people to be assessed as competent or whether lowering the standard is illusory as being for the benefit of people whose competence may be open to question. Therefore it is hardly surprising that there continues to be debate as to the abilities that any individual must possess (and the level of functioning of that ability) in order to determine whether he or she is competent to make a particular decision. Wong *et al.*[19] make the point, drawing on the work of others, that the functional approach is not without problems. They point out that it is time consuming, legal standards vary between jurisdictions, and there is uncertainty about the threshold to be satisfied in determining competence.

The English Mental Capacity Act 2005, sections 2 and 3 creates a definition of capacity consistent with those abilities. The central elements of that definition are to be found in sections 2(1) and 3(1).

> 2(1) For the purposes of this Act, a person lacks capacity in relation to a matter if at the material time he is unable to make a decision for himself in relation to the matter because of an impairment of, or a disturbance in the functioning of, the mind or brain.
>
> 3(1) For the purposes of section 2, a person is unable to make a decision for himself if he is unable –
>
> (a) to understand the information relevant to the decision,
> (b) to retain that information,
> (c) to use or weight that information as part of the process of making the decision, or
> (d) to communicate his decision (whether by talking, using sign language or any other means).

Three approaches

What is key is that the approach is a functional one that is it is related to the abilities of the individual at the time a decision is required and is not dependent upon either status or outcome of the decision, though these clearly have formed part of either definitions or approach to capacity in the past and are relevant factors in identifying the possibility that someone may not be capable of decision-making and in exercising judgment about that capacity. To state that competence if to be interpreted functionally.[19] means that the status of the decision-maker is not determinative of the question of her or his competence. *A status approach* makes assumptions about an individual's decision-making competence on the basis of a particular characteristic, and there is no empirical evidence to support the validity of such an approach.[19,21] The mental state of the decision-maker may be the reason why competence is put into question, but mental state in itself is rarely, if ever, sufficient to determine the matter. Mental state may have relevance to decision-making in that certain states will impact on the ability to understand and process information. Furthermore, the outcome of a decision is also not in itself sufficient to determine the matter. The fact that any given decision is not reasonable does not mean that the decision-maker is not competent to have made that decision. For example, the simple fact that a patient disagrees with the doctor does not mean that the decision is that of an incompetent decision-maker, though lack of congruence with the proposals of a doctor may cause questions to be asked about decision-making competence. *The outcome approach* has been rejected in a number of jurisdictions.[19] It is internationally recognized that anyone can make what might be termed objectively silly decisions without necessarily giving rise to doubts about competence. However, the regularity with which silly decisions are made may raise doubts about competence as also will the inter-relation between mental state and quality of decisions. In the United Kingdom, these points are further reflected in the fact that there is a legal presumption

that a person is competent to make her or his own decision once adult state is reached.

The functional approach requires that the test of competence be related to the particular decision to be made, at the particular time that it must be made.[19] There is a range of abilities that competence might involve. Much of the work on competence has been undertaken in the context of health-care law and in relation to consent to treatment. Much of this work has been undertaken in the United States. Two leading thinkers, Grisso and Appelbaum,[20] have, with colleagues, identified four abilities that can

The functional approach to decision-making is not limited, in its application, to health-care decisions, even though that is where most of the debate has taken place. In principle, it may be applied to any type of decision. For example, the making of wills and the entering into of contracts are obvious examples where a functional approach applies, but it does not follow that the same abilities will be required for these decisions as for treatment decisions. Under the law prior to the Mental Capacity Act 2005, this is demonstrated by an old case which was, nevertheless, the leading case in relation to the making of wills. *Banks* v. *Goodfellow*, requires that a person:

> ought to be capable of making his will with an understanding of the nature of the business in which he is engaged, a recollection of the property he means to dispose of, of the persons who are the objects of his bounty, and the manner in which it is to be distributed between them.

This test demanded not just understanding, but also the appreciation and the reasoning ability noted above. Whilst the level at which the will writer must operate is not that of a lawyer, nevertheless he or she must be aware of the context in which the will is being made and must think through the competing potential demands on his or her estate. Of course, will writers can make silly dispositions, even going so far as to exclude financially dependent relatives. However, it must be recalled that an outcome that is questionable or unreasonable is not the same as the will or decision being made on the basis of an unacceptable reasoning process. An eccentric person might well, for example, not wish to leave anything to his or her relatives. Thus, in any jurisdiction, care must be taken to consider a particular test in deciding which of the four abilities are to be identified, and the answer to that question may demand very careful analysis.

Persons not competent to make decisions or take action

If a person is not competent to make a decision for themselves, there is more variability as to the approach to be taken. Some of this difference is related to the commitment to do the best for a vulnerable person and leads to a desire to act paternalistically, so making the decision that objectively is in the best interests of the individual A more frequent approach is to make the decision that that individual would have made for themselves. This is most recently reflected in the English Mental Capacity Act.

If a person is not competent to make a decision or to take certain action, the law increasingly provides mechanisms whereby these decisions can be made. In England and Wales, until the abolition of the sign manual by the Mental Health Act 1959, the Crown had the power and the process to make decisions in the best interests of a person not competent to decide or to take action. This power is the basis of many substitute decision-making procedures in common-law jurisdictions (including the United States). The specific adaptation is jurisdictionally specific. In some jurisdictions, it has been used to follow from generic decisions as to competence and to create overarching substitute decision-making procedures (e.g. those jurisdictions that adopt a full guardianship of the person approach). In England and Wales, the first approach, after the abolition of the sign manual procedure, was to allow for people to be received into guardianship (a process that was not, interestingly, dependent upon a finding of incompetence), but this proved not to be an acceptable procedure. Current English guardianship is very much only a community mental health power that does not enable anyone else to make decisions on behalf of the person received into guardianship.[33]

In England, the courts had to invent a procedure for making decisions on behalf of someone who was not competent. The House of Lords, the senior English court, provided a mechanism in relation to treatment decisions whereby, if a person was found to be incompetent, treatment could lawfully be provided if it was necessary, that is if it was for the life, health, or welfare of the patient and was in her or his best interests. Despite substantial improvements that made the approach identify what was the one, best approach that took into account the full range of interests (see below), this judicial approach has been replaced by a statutory format. The approach introduced by the Mental Capacity Act 2005, does not take a full guardianship or guardianship of the person approach and is not necessarily triggered by a judicial or administrative authority. Rather, a person may act on behalf of an incapacitous adult (as defined by sections 2 and 3, see above) provided she or he acts in that person's best interests (as defined by section 4, see below), but such actions are subject to significant procedural protections, since some areas must be referred to other procedures (e.g. in relation to research, where sections 30–34 allow intrusive, non-therapeutic treatment consistently with the European Convention on Biomedicine), some decisions cannot be made as they are too personal (so such matters as consenting to marry or to entering a civil partnership and consenting to sex do not fall within the Act, see section 27), some decisions necessitate, where carer's views are not available, the views of an independent mental capacity advocate to be taken into account (sections 35–41), some decisions may be made by a court or by a court appointed person where the court determines that is appropriate (through the new Court of Protection, with preference given to decisions made by the court rather than the appointment of a deputy), and all decisions are challengeable in court. This latter element is vital. Whilst this approach is clearly consistent with the provisions of Recommendation 99(4), it is challengeability which lies at the heart of its compliance with human rights obligations. What the Act does not do is require an initial judicial or administrative decision. It is not triggered by having to go to court or through some other governmental or quasi-governmental body. This will no doubt be challenging to many whose commitment to due process and procedural justice would rely upon judicial instigation of a procedure. However, this is not the only necessary approach, as is evidence in Recommendation 99(4). What is provided instead is a straightforward ability to challenge decisions by taking a matter about competence or about decisions made on behalf of someone who is or may be incompetent to the Court of Protection. Some people can launch a case as of right, some need the permission of the Court (section 50). This is clearly sufficient to meet

the demands of, for example, Article 6 of the European Convention on Human Rights, provided it operates in practice. If it fails to take cases that should get to the Court, then that might be a base of challenging the provision. The advantage of this approach, which are consistent with that of Recommendation 99(4), is that it more closely reflects the process applicable in relation to someone who is capable, it places emphasis on the fact that most decisions are taken on behalf of an incapable person by carers and those decisions are proper and appropriate and it provides a system that can work (that is the workload should be manageable and not be prohibitively expensive).

The focus for making decisions on behalf of another in England and Wales as in most jurisdictions, is individualized, function specific and based on the best interests of the individual. This *best interests approach* is, realistically, the only one available to the courts where there is no evidence of that person's preferences. Substituted judgement is, however, an appropriate approach where there is sufficient evidence of the decision that the person now incompetent would have made.[30] So, for example, decisions made in advance, advance health-care statements, are legally valid[22] (see also Kennedy and Grubb,[30] referring to such an approach in Florida, Ontario, Manitoba, and Victoria). Indeed, the old thinking that substituted judgement was an alternative to best interests should be re-thought so that what an individual wants is what is in her best interests, but if that is not known, nest interests is the only available approach. Where the person is not competent, the best interests approach, in England, was achieved by deciding whether what the doctor proposes in the given case is a treatment regime of which a responsible medical opinion would approve. This approach to 'best interests' was rightly severely criticized for it failed to address the issue by concentrating upon the interests of incompetent persons but professionalized it through one (medical) profession when it is possible to take a broader view of the issues in question when deciding upon what treatment to agree upon. Some of these criticisms were ameliorated by judicial developments (see, e.g. *Re S (Adult Patient: Sterilization)* and *Re A (Male Sterilization)*) that ensured two key changes. First, it was decided that, where there were options of a number of possible, acceptable approaches, only one of those options could be in the best interests of the individual. Secondly, in deciding what was in someone's best interests that should not be limited to scientific or medical matters, but should take in the whole range of social, welfare, and emotional factors. Despite such substantial judicial development, new law is in place through the Mental Capacity Act 2005. Section 4 provides a definition of best interests.

> 4(1) In determining for the purposes of this Act what is in a person's best interests, the person making the determination must not make it merely on the basis of –
>
> (a) the person's age or appearance, or
>
> (b) a condition of his, or an aspect of his behaviour, which might lead others to make unjustified assumptions about what might be in his best interests.
>
> (2) The person making the determination must consider all the relevant circumstances and, in particular, take the following steps.
>
> (3) He must consider –
>
> (a) whether it is likely that the person will at some time have capacity in relation to the matter in question, and
>
> (b) if it appears likely that he will, when that is likely to be.
>
> (4) He must, so far as reasonably practicable, permit, and encourage the person to participate, or to improve his ability to participate, as fully as possible in any act done for him and any decision affecting him.
>
> (5) Where the determination relates to life-sustaining treatment he must not, in considering whether the treatment is in the best interests of the person concerned, be motivated by a desire to bring about his death.
>
> (6) He must consider, so far as is reasonably ascertainable –
>
> (a) the person's past and present wishes and feelings (and, in particular, any relevant written statement made by him when he had capacity),
>
> (b) the beliefs and values that would be likely to influence his decision if he had capacity, and
>
> (c) the other factors that he would be likely to consider if he were able to do so.
>
> (7) he must take into account, if it is practicable and appropriate to consult them, the views of –
>
> (a) anyone named by the person as someone to be consulted on the matter in question or on matters of that kind,
>
> (b) anyone engaged in caring for the person or interested in his welfare,
>
> (c) any donee of a lasting power of attorney granted by the person, and
>
> (d) any deputy appointed for the person by the court,
>
> as to what would be in the person's best interests and, in particular, as to the matters mentioned in subsection (6).

The law relying on status

Whilst it has so far been asserted that approaches not facilitating decision-making by a competent person are the norm and that capacity should not be questioned on the basis of status, it is the case that status may play a role. Indeed, for example, in England and Wales status has, in the past, been the basis for effectively determining whether someone is capable, but increasingly these are being removed, as is the case across the globe. It is also a move demanded by international instruments, such as the Council of Europe Recommendation.

In England and Wales, it used to be the case that children under 16 were not able to consent, but the House of Lords changed this in 1985 when, in *Gillick* v *Wisbech AHA* it was recognized that, at least for some treatments, a person under the age of 16 could be capable of deciding upon treatment provided they had sufficient maturity to do so. The removal of the automatic barrier was an important step.

Further, the Sexual Offences Act was another example of law in a private area that was dependent upon status. For most people, it always has been a matter that is dependent upon their own consent. So, for example, it is rape for a man to have anal or vaginal sexual intercourse with a woman or a man who does not consent. The critical question is whether the victim is competent to make the decision. For example, does she or he understand what sexual intercourse is so that her or his apparent assent is indeed consent? However, under the 1956 Act this was not the case in all instances. First, there was an age of consent. Below the relevant age, the consent of the victim was irrelevant, however competent she or he may be. Second, a person who was a 'defective' in the terms of the Sexual Offences Act 1956 or had a 'severe mental handicap' could not consent. These terms were defined in the same way and referred to a person who had a state of arrested or incomplete development of mind that is associated with severe impairment of intelligence

and social functioning. If a person fell into this category, she or he could not, in law, consent to sexual intercourse or other sexual activity, however competent he or she might have been. This had real impact on some people with mental retardation (intellectual disability) and their carers. Interestingly, if a man married a woman with a severe intellectual disability (who was, therefore, a 'defective'), he did not commit the offence contrary to Section 7 of the Sexual Offences Act 1956 (whereby it was an offence for a man to have unlawful sexual intercourse with a woman who was a defective). Marriage was and is not dependent upon status, but is dependent upon an assessment of the competence of the particular individuals at the time of the marriage ceremony, and so falls into the second category of laws.[33] The law meant that a person could not prevent an indecent assault in any circumstances by consent. This could have unfortunate consequences for sex education for some people with severe mental retardation. It may be that no other form of sex education is possible than hands-on education to provide skills to enable appropriate behaviour by the person in question. Whatever the level of necessity, the individual could not consent in law. The only possible defence was to argue that the activity, contrary to appearances, was not indecent because of the purpose for which it was being undertaken as evidenced by the context, that is a carefully tailored and developed personal relationships programme. The difficulty here was that, when perceived from this stance, the law fails to allow sexual expression without good individual reason. Therefore it could be contrary to Article 8 (privacy) or Article 12 (founding a family) of the European Convention on Human Rights. The law presumably assumed that sexual experiences would, by definition, exploit or abuse such people. Law to prevent exploitation and abuse is very important, but it must not improperly limit a person's human rights. Subsequently, there has been an attempt to redress the balance somewhat and provide for a set of laws that is more acceptable. The Sexual Offences Act 2003 creates three types of offences that have an impact where the victim is a person with a mental disorder. The first consists of offences involving sexual activity with a person with a mental disorder and apply where that person cannot consent. The second consists of offences where the person's agreement is achieved through an inducement, threat or deception. The third comprises offences where the defendant is in some form of care relationship with the victim and these offences are committed regardless of consent and clearly deal with a significant form of exploitation and abuse. It is worth focusing, briefly, on an exemplar from the first type of offences. Section 30 of the 2003 Act makes it an offence for someone to engage in sexual activity with a person with a mental disorder impeding choice. As the offence relies upon touching, it involves sexual intercourse and other sexual activity short of intercourse but that involves touching. What it does not include is activity that would not involve touching, but would have been regarded as an assault under the old law. That touching must be sexual. It remains to be clarified whether sex education of a direct manner described above would be caught, but there must at least be an argument that it would not, provided that there is a non-sexual purpose, established by the education programme within which it falls and that a multi-disciplinary group identifies the need for the touching and the means of doing it. The offence applies potentially to anyone with a mental disorder, and is not so status driven as was the old law reliant upon being a defective or a person with a severe mental handicap. There is, though, still the status requirement of a mental disorder, which might be justified upon the basis of necessity and proportionality, as there must be an inability to refuse on the basis of that mental disorder, so the offence does not apply simply because of the presence of the disorder. This requirement is the equivalent of a capacity requirement tied to the specific requirements of the matter in hand. Finally, the defendant must know or could reasonably have known of the other's mental disorder and that he or she is thereby unable to refuse. These offences are likely to produce a much better balance of the need to protect the rights of vulnerable persons to understand and exercise their sexuality with their right to be protected by the law from sexual abuse and exploitation.

The law of responsibility

Criminal liability

Mental disorder is relevant to criminal liability in a variety of different ways, and this is true, at least, in all common-law jurisdictions. First, the presence of a mental disorder may be a reason to convince the decider of the fact that, contrary to external appearances, the defendant did not have the mental element for the crime with which he or she has been charged. Lack of the mental element is, of course, a complete defence. Whilst there is not a mental element requirement for all crimes (since there are some crimes for which conviction is based on strict liability), most serious crimes require a mental element that takes the form of intention, recklessness, knowledge, or belief and demand a consideration of what was the individual's purpose, awareness, foresight, or realization at the time that the crime was committed.

Second, the presence of a mental disorder may give rise to a defence. This will arise either by it being raised by the defence (even if the defence does not have the formal burden to prove it, but will have the burden of raising the matter for consideration) or by the prosecution challenging a point made by the defence (so, for example, if the defence raises mental disorder as an explanation for lack of criminal intent, the prosecution may respond by arguing that the defence has, in fact, raised one of the defences concerned with mental disorder). The most obvious defence is that of insanity. Whilst this takes various forms in many jurisdictions, there is usually a relationship in common-law jurisdictions with the English defence that was established by judicial answers to questions posed in McNaghten's case in 1843. This defence demands that the following matters be established by the defence (this is one of those rare instances in which the burden of proof lies, on a balance of probabilities, on the defence).

The defendant must have a disease of the mind. There are at least two possible ways of approaching this concept. First, it may be regarded as a simple concept in that it is present if the defendant has a condition (loosely termed) that has an internal cause, whereas there is no disease of the mind if there is an external cause. This simplistic distinction means that a person who has a brain tumour has a disease of the mind as does a person who has arteriosclerosis, schizophrenia (or other mental illness), epilepsy diabetes (provided the defendant had not taken her or his insulin and caused the offence in a hypoglycaemic state, or is a sleepwalker). The person with diabetes who causes the offence after taking insulin (an external agent) but falls into a hypoglycaemic state does not have a disease of the mind because the cause (insulin) is external and not internal. Immediately it can be seen that there is no congruence

between the legal construct of disease of the mind and any medical approach. Furthermore, this definition of disease of the mind produces outcomes that are clearly unacceptable (no one would argue that many of the conditions identified above are identifiable with mental disorders). An alternative definition, which may reduce some of the impact of the concept is to follow Lord Denning: 'it seems to me that any mental disorder which has manifested itself in violence and is prone to recur is a disease of the mind. At any rate it is the sort of disease for which a person should be detained in hospital rather than be given an unqualified acquittal'.

One difficult area of applying the external/internal causes distinction is in relation to 'whether a 'dissociative state' resulting from a 'psychological blow' amounts to insane automatism'. This was recognized by the Supreme Court of Canada so that the psychological blow can be recognized as an external cause giving rise not to insanity but to automatism as a defence.

The disease of the mind must cause a defect of reason. Defect of reason means that 'the powers of reasoning must be impaired and that a mere failure to use powers of reasoning which one has is not within the [McNaghten] Rules'.(47)

The consequences of actions A and B must be that the defendant either does not know what he is doing or does not know that what he is doing is legally wrong. The latter is contentious, because the Rules simply state that the defendant must know that what he was doing was wrong. In *R* v. *Windle* it was the Court of Appeal that established that the requirement was that the matter is concerned with legal wrong. The High Court of Australia has refused to follow this approach. In *Stapleton* v. *R*, 'their view was that if D believed his act to be right according to the ordinary standard of reasonable men he was entitled to be acquitted even if he knew it to be legally wrong'.(45)

The outcome of a finding of insanity is that the defendant is found not guilty by reason of insanity and, since the amendments introduced by the Criminal Procedure (Insanity and Fitness to Plead) Act 1991, the disposal of the defendant is not limited to being sent to a mental hospital under the equivalent of a restriction direction, but extends to less draconian forms of disposal, including discharge.

Third, there are limited or partial defences. In English law there is a defence of diminished responsibility that is a defence only to murder and produces, if successfully raised by the defendant, a conviction for manslaughter. There are similar defences, often of more general application in most, if not all, common-law countries. The defence of diminished responsibility was created by the Homicide Act 1957, Section 2, which provides:

> Where a person kills or is a party to the killing of another, he shall not be convicted of murder if he was suffering from such abnormality of mind (whether arising from a condition of arrested or retarded development of mind or any inherent causes or induced by disease or injury) as substantially impaired his mental responsibility for his acts and omissions in doing or being a party to the killing.

The question of impairment of responsibility is one for the decider of fact to make. Interestingly, expert evidence usually contains an assessment of the degree of impairment, though this would appear not to be a matter upon which the expert has the relevant training or expertise. Were the expert witness not to proffer a view on the matter, the practical reality is that the court would find it very difficult to know how to react to a claimed defence. The original rationale for this partial defence was to avoid the rigour of capital punishment. Its current rationale is wide ranging. Amongst other reasons why this defence is important is that it allows the defendant to argue that he or she was incapable of resisting an impulse produced by mental disorder, an argument that is not permissible in the insanity defence as is made clear by the McNaghten Rules themselves.

Fourth, there are other defences which are or may be related to mental disorder. One obvious defence is that of intoxication. If a person is intoxicated by drink or drugs such that he or she does not have the criminal intent for an offence, he or she is not guilty of that offence provided the offence is one of specific intent (such as murder), whereas he or she will be guilty if the crime is one of basic intent (such as manslaughter). Although a range of theories have been propounded to establish when a crime is one of specific or basic intent, the only approach that actually works is to take previous decisions as precedents for future approaches, and so develop a list of crimes of specific and of basic intent.

Tortious liability

Liability in tort encompasses a wide variety of non-contractual civil wrongs, including such diverse matters as negligence, nuisance, defamation, and trespass to land. It is worth noting that trespass to the person (assault and battery) is a civil wrong as well as a criminal offence and concurrent liability will lie. A battery occurs when there is a non-consensual touching, which includes the administration of medical treatment and care so that regardless of any benevolent motivation, if this is given without a valid consent an action will lie. However, this section concentrates on the law of negligence which is the main area where liability problems might occur. Again, the authors must emphasize that we concentrate mainly upon English law, but within the area of tort the law in other English-speaking jurisdictions is very similar e.g. in the USA, Australia, New Zealand, and Canada, and even in codified jurisdictions, such as France, Spain and Germany, there are similarities of approach.

In this section we look first at the general principles of the law of negligence. Second, any special considerations when examining the liability of the mentally ill defendant in an action in negligence are discussed. Third, the liability of third parties for the acts of the mentally ill are examined, and finally, the liability of third parties towards mentally ill patients, particularly in the context of statutory duties, and the difficult and controversial issue of the liability towards patients who harm or threaten harm to themselves are discussed.

(a) Negligence

The law of negligence is heavily circumscribed by a conceptual framework which is designed to restrict the ambit of claims. Much of this need not concern us here, suffice to say that the most relevant parts of it relate to the difference between certain types of loss and the way in which the courts regard these differences, and the role of public policy in some of the principal judgments.

The basic requirements of the tort of negligence have a certain simplicity; it is in the application of these requirements that complexity and confusion result. The claimant must show (a) that the defendant owed him or her a duty of care; (b) that the defendant breached that duty by failing to meet the requisite standard of care, and (c) that the breach of duty caused the resulting damage. It is important to note the latter. Negligence is not actionable *per se*;

there must be some tangible damage caused by the breach. There is insufficient space here to consider duty of care in all its contexts, but it is important to note the test put forward in *Caparo Industries PLC* v. *Dickman*. In that case it was said that in order for a duty of care to arise, the damage suffered must be foreseeable; there must be sufficient proximity between the claimant and the defendant; and it must be 'just and reasonable' to impose a duty of care. The first two aspects are well illustrated by examining the difference between physical injury and other forms of damage; the third is an important consideration in the context of statutory duties. As far as the standard of care is concerned, the standard is objective and based upon reasonableness. Causation must always be proved as there will be no liability for falling below the requisite standard of care if a causal link to the damage cannot be proved. The standard of proof throughout is that of the balance of probabilities.

(b) Liability for clinical negligence

Duty of care is not usually an issue in the doctor/patient relationship. It should be noted that the standard of care relating to professionals such as doctors is set by the 'accepted practices' test, well known as the 'Bolam test' as framed in the case of *Bolam* v. *Friern Hospital Management Committee*, where diagnosis and treatment will not normally be negligent if supported by a responsible body of medical opinion. The only exception to this is if the doctor's actions do not withstand logical analysis; in those circumstances the fact that the practice is 'accepted' will not be sufficient to avoid a finding of negligence. The test is slightly different if the allegation of negligence relates to the provision of inadequate information about risks, side effects and alternative treatments. In such cases it seems that the courts are moving towards a more stringent standard of informed consent. Proving that the damage was caused by the breach can raise many problems in medical negligence claims, largely for two reasons. First, in almost all cases the patient will be suffering from some medical condition at the outset, and it might be arguable that the resulting condition would have materialized anyway. Second, if the resulting condition is the realization of some form of risk (e.g. side-effects of medication), even if the patient was not warned of the risk it is open to the defendant to argue that regardless of warnings, the patient would have gone ahead and consented to the taking of the medication in any event.

(c) Liability for different types of damage—psychiatric injury

The law distinguishes between different types of damage. This appears to stem from judicial fear that certain losses might result in an unacceptably large number of potential claimants i.e. the fear of opening the 'floodgates'. Thus, what is known as 'pure economic loss', that is financial loss, which is not consequential upon physical damage, will only be recoverable in certain circumstances. For similar reasons the law makes another significant distinction: the difference between physical damage and psychiatric damage.

There are a number of reasons for the disparity between the treatment of the two types of injury. The first stems from the misunderstanding in earlier cases of the nature of psychiatric injury. In the authors' view, it is no coincidence that some of the early cases involved pregnant women who had miscarried or given birth to damaged children (e.g. *Dulieu* v. *White & Sons*). In these cases the courts could see a tangible manifestation of the 'shock' suffered by the claimants. Psychiatry was in its infancy, and the myriad of subtle manifestations of mental disorder was unknown. A further illustration of this is the phenomenon of 'railway spine'. In 1875, J.E. Erichsen, Professor of Surgery at University College Hospital, London, published a number of findings as a result of studying spinal injury cases following railway accidents, where there was no obvious physical cause of the symptoms manifested. His conclusion was that trauma caused 'concussion of the spine'. Later medical opinion such as that of surgeon Herbert Page denied that the spine as such was affected by the trauma, but that the resulting condition was 'nervous shock'. It is therefore understandable that courts may have taken a somewhat crude approach to shock-induced injury, which crudity is still reflected in the law, which is commonly referred to as 'the law of nervous shock'. However, the second reason is, perhaps, less acceptable: the fear of the floodgates opening and admitting unacceptably large numbers of claims. It is worth pointing out that the floodgates argument, being only a spectre (but a highly influential one at that) and not overtly referred to by judges, has never been supported by hard empirical evidence. Nevertheless, the Law Commission, in its report on psychiatric injury claims supported the floodgates argument, largely on the basis of the lack of clear demarcation lines between general, uncompensatable, mental disturbance, and specific psychiatric illnesses. A third reason for the distinction between physical and psychiatric injury is the argument that a too-liberal attitude might result in fraudulent claims. However, again there is no clear evidence to show that it is easy to fake psychiatric injury, and furthermore it is an odd system of justice which takes the approach that, because there is a possibility of fraud, genuine claims should fail.

It is important to note that in cases where the claimant has suffered physical injury, any claim for damages for associated psychiatric injury will not be controversial, subject to the claimant establishing causation. However, where the claim is for psychiatric injury alone special considerations apply and, it is worth summarizing the essential elements of such a claim. The distinction is often drawn between primary and secondary victims. A primary victim is either someone who is physically injured or is within the range of foreseeable physical injury (see *Page* v. *Smith*).

A secondary victim is someone who has some sort of proximity to one or more primary victims. (There are similarities in other European jurisdictions; for example in France this is *dommage par ricochet*, and the rules are more generous than in English-speaking jurisdictions.) In English law this has two aspects: first, there must be close physical proximity to the trauma which injures or threatens the primary victim; second, the secondary victim must either have a close tie of love and affection with one or more of the primary victims, or he or she must be a rescuer of a primary victim and be within the range of foreseeable physical injury (see *Alcock* v. *Chief Constable of South Yorkshire Police*) and *White* v. *Chief Constable of South Yorkshire Police*). There is one other condition which a secondary victim must meet: the trauma must have the necessary quality of 'shockingness' about it so as to make a sudden impact on the senses to such an extent that the person of normal fortitude must be shocked by it (the 'impact rule'). Finally, in the cases of both primary and secondary victims, the negligence must result in a recognized psychiatric injury; emotional conditions such as grief, fear, and distress will not suffice. Of course, if the claimant has suffered distress as a result of physical injury, not amounting to a recognized psychiatric condition, then this should be reflected in the damages for pain, suffering, and loss of amenity. Although ordinary 'shock' is not compensatable, both DSM-IV and ICD-10 refer to 'acute stress reaction' which, although only a short-term

reaction to stress, should be compensatable as a recognized psychiatric condition, albeit that any compensation would be modest. (See *Phelps* v. *London Borough of Hillingdon*, where it was held that the failure to mitigate the adverse consequences of a congenital defect such as dyslexia was capable of constituting an 'injury'.)

The impact rule states that the claimant must be present at the traumatic event or its immediate aftermath, and it must be an event such as to make an impact upon the unaided senses of the claimant, and to be shocking to a person of normal fortitude. The case of *Sion* v. *Hampstead Health Authority* is illustrative of this point. The claimant was the father of a young man injured in a road accident. The defendant was the health authority because the father alleged that his son was treated negligently. The claimant watched his son deteriorate and die over a period of 14 days. The resultant psychiatric illness suffered by the claimant was not compensatable as the process of death was slow, predictable, and not 'shocking'. This can be contrasted with *Tredget and Tredget* v. *Bexley Health Authority*, which concerned the negligent delivery of a child, which took place in an atmosphere of 'chaos' and 'pandemonium', and resulted in the child being born in a distressed state requiring immediate resuscitation. The father, who was present at the birth, recovered damages for his subsequent psychiatric condition, as the event was sudden and impacted in a shocking way upon his senses. In *Walters* v. *North Glamorgan NHS Trust*) damages were awarded to a woman who spent 36 hours with her baby son from seeing him choking on blood and vomit to the termination of life support. This period was found to be a shocking series of events. However, in other jurisdictions the need to find something sufficiently shocking upon which to hang liability has been rejected. In the US case of *Ochoa* v. *Superior Court (Santa Clara County)*, a woman recovered damages when, despite her son begging her to stay, was forced to leave his hospital bedside on the basis that he only had flu, and never saw him alive again. In Singapore, it was said that in the context of medical negligence as the secondary victim would rarely witness the act of negligence, it was wrong to impose the 'shocking event' requirement. Thus, in *Pang Koi Fa* v. *Lim Djoe Phing*) damages were awarded when a negligent medical procedure took place in June 1985 and the claimant watched her daughter die from then until the following September.

These sorts of distinctions are important in the context of so-called 'creeping trauma' in which there is no sudden impact to the senses. This refers to cases where victims have been exposed to some form of risk, for example contamination or the administration of a drug which has caused no harmful effects yet, but which might do so. The absence of 'impact' might be thought to be fatal to such claims. However, in an Australian case (*AQP* v. *Commonwealth Serum Laboratories Ltd*) the Victoria Supreme Court held that such cases should be decided on the basis of foreseeability and proximity alone. Subsequently this was confirmed in an English decision, where a group of claimants had received the human growth hormone at a time when the Department of Health and the Medical Research Council was aware of the possibility of Creutzfeldt–Jakob disease contamination. They subsequently developed psychiatric illnesses through fear of contracting Creutzfeldt–Jakob disease, and recovered damages. At first sight it may be thought that the impact rule would not apply because the claimants were primary victims. Although this is the common-sense view inasmuch as they had all been directly administered a drug, the judge was not willing to concede this. This was on the basis that it would widen unacceptably the number of potential primary victims in a much more widespread case of possible contamination and would inhibit manufacturers and providers of drugs and other goods from warning the public even when the risk was very small. Therefore the claimants were held to be secondary victims, but to whom a duty was owed simply on the basis of foreseeability. A duty of care was found to exist because of the nature of the relationship between the defendants and the claimants, the small and readily identifiable size of the group of claimants, the ways in which the claimants might become aware of the risk of Creutzfeldt–Jakob disease, and the nature of the suffering in terminal Creutzfeldt–Jakob disease.

(d) The liability of the mentally ill in negligence

It might be thought that if the law of negligence is about fault and blame (as it is in the English-speaking jurisdictions, and some European jurisdictions as well), then those who have less appreciation of risk and the consequences of their own actions might be regarded as being less culpable than those with a reasonable degree of appreciation. However, this is not the case. It is important to understand that, although the historical development of the law of tort suggests a number of different principles behind tort compensation and that in rare circumstances punitive damages can be recovered, generally tort is concerned with compensating victims rather than punishing wrongdoers. In the light of this it is not difficult to see why the same standard of care is applied. The second reason for this is that courts do not want to apply a different standard of care depending upon factors such as intelligence, emotional reactions, and experience. Therefore the standard is objective, being that of the reasonable person who is reasonably competent at whatever task is being undertaken. (Note that there is an exception in the case of children who are judged in accordance with the reasonable foreseeability of the child of the relevant age.) The general principle is illustrated by the classic example of the learner driver who is expected to meet the standard of the reasonably competent driver (see the case of *Nettleship* v. *Weston*). In *Wilsher* v. *Essex Area Health Authority*, in the Court of Appeal, Mustill LJ stated: 'this notion of a duty tailored to the actor rather than to the act which he elects to perform, has no place in the law of tort' (the case subsequently went to the House of Lords but this aspect of the judgment was undisturbed). However, there have been some exceptions to this (albeit not in England) which have interesting implications for the liability of the mentally ill. In the case of *Cook* v. *Cook*, the High Court of Australia held that, in the case of an inexperienced driver, liability may be different depending on the identity of the defendant. For example, a car driver who injures a pedestrian will be subject to the same standard of care as the reasonably competent driver, but when a driving instructor is injured, the fact that he knew of the claimant's lack of experience might mean that there is no liability for his injuries. By analogy with *Nettleship* v. *Weston*,[68] a mentally disordered person who negligently injures a third party will be subject to the objective standard of care. These latter considerations, however, might have some relevance to the position of those who care for the mentally ill in that they might be expected to be aware of the increased likelihood of unstable behaviour. In other words, can it be argued, that if a patient negligently injures a doctor, nurse, or social worker, in the course of, say receiving treatment, then he might not be liable? It should be noted, however, that we are concerned with negligence, not deliberate acts, so this would not be a common scenario. Furthermore, the patient would very likely have

few resources and no insurance so may not be worth suing. Nevertheless, if an action were financially feasible, it may be that, for policy reasons, the courts would be unlikely to render someone in the position of a carer, without compensation. However, in *Mansfield* v. *Weetabix Ltd*, the Court of Appeal stated that in a case where the defendant could not reasonably have known about his condition, or the effect of it, there would be no liability.

(e) Liability to third parties for the acts of the mentally ill

The next question that arises is the extent, if any, of the liability of third parties for acts of the mentally ill, whether these are criminal or civil acts. The third party might be an individual who is caring for the defendant, but is much more likely to be a local authority or a health authority. This area of negligence has given rise to some emotive and controversial litigation. In *Hill* v. *Chief Constable of West Yorkshire Police* the House of Lords held that no liability attached to a police force that failed to arrest the serial killer Peter Sutcliffe before he killed his last victim. The decision did not turn upon whether there had been negligent conduct, but upon public policy. The interests of the public as a whole are best served, so runs the argument, if those responsible for public safety and so on are able to carry out their duties unfettered by the threat of litigation. It must be noted, however, that in the case of *Osman* v. *United Kingdom*, this so-called immunity from suit was held by the European Court of Human Rights to be contrary to Article 6.1 of the European Convention on Human Rights (the protection of the right to a fair trial in both criminal and civil cases).

In *Palmer v Tees Health Authority*, the claimant, whose four year old daughter was murdered by a psychopath, brought an action against the health authority, both in her own name as she had suffered from a psychiatric illness as a result of the murder, and on behalf of the child's estate. Some months prior to committing the murder, the defendant had been an outpatient at the defendant's psychiatric hospital, where he had allegedly told staff that he would kill a child. The Court of Appeal rejected her claims on the basis that there was not sufficient proximity between the defendant and the victim. If, however, the defendant had known his victim and made threats specifically against her then there may well have been sufficient proximity to establish liability.

(f) Liability of the providers of services to the mentally ill

In the case of *Clunis* v. *Camden and Islington Health Authority*, Clunis was a mentally disordered man who had been discharged from a psychiatric hospital and subsequently killed a man in an unprovoked attack. The prosecution accepted his plea of manslaughter on the ground of diminished responsibility. Clunis subsequently sought damages from the health authority alleging that they had failed to treat him in accordance with their common-law duty of care, and that if he had been treated he would not have carried out the killing and would not have been subject to the lengthy period of detention he was now facing. The court invoked the legal maxim of *ex turpi causa non oritur actio*, which means that they found that he could not establish a duty when it stemmed from the claimant's own wrong-doing. The court relied on this as an aspect of public policy, and, somewhat curiously, referred to the importance of the deterrent effect of such a maxim. Given that the claimant was mentally ill, and the motiveless nature of the attack, he was surely incapable of being deterred? It is tempting to consider that *Clunis* might have been decided differently in terms of *ex turpi causa*, if it had not also had implications for fettering the medical and resource-allocation discretion of health authorities to make them liable in negligence in this way.

It is well established that English health authorities can be both primarily liable (when there is an organizational failure) and vicariously liable (where an individual employee is negligent) for acts of negligence. However, the provision of statutory duties by local authorities has raised some difficult issues. The leading authority is *X* v. *Bedfordshire County Council* In this case (a number of different cases listed together) the court had to consider the claims of claimants who alleged damage as a result of the negligence of social workers and psychiatrists involved in child protection cases, and teachers and others involved in the provision of education to children with special educational needs. Applying the threefold test from Caparo the court found that the first two elements were satisfied, that is foreseeability of damage and proximity of relationship between the parties. However, on the third element, the finding was that it would not be 'just and reasonable' to impose a duty of care. In the context of child protection three reasons were given. First, the statutory system set up to protect children cuts across many disciplines: police, education bodies, doctors, and others. To disentangle these relationships and to ascertain where the blame must fall would impose 'impossible problems'. Second, because there is a very fine balance to be drawn between the protection of children and the disruption of removing them from their homes, there is often a conflict to be resolved. The professionals involved in making such decisions should not be further hampered by the threat of litigation. Furthermore, a common-law liability may result in too cautious and defensive an approach on the part of local authorities. Finally, on a practical point, because of the obvious tensions between the local authority and the parents involved, litigation would be commonplace thereby placing a drain on vital resources necessary for the carrying out of the statutory functions.

However, if the social workers and psychiatrists involved *personally* owed duties towards the claimants then, it was said, local authorities might be vicariously liable for any negligence on their part. Furthermore, the House of Lords speculated that educational services might be treated differently in that they were more in the nature of a 'service to the public'.

In *Phelps* v. *London Borough of Hillingdon*[60] the House of Lords confirmed this approach. It concerned a claim for the failure of an educational psychologist to diagnose dyslexia. The court stated that vicarious liability arose on the basis of the personal duty owed by the psychologist, notwithstanding the fact that the educational psychology service was part of the general statutory services provided under the Educations Acts of 1944 and 1981, and that there would be no liability on the part of the local authority for breach of those statutory duties. A similar issue arose in the case of *Clunis* v. *Camden and Islington Health Authority*[73] (the facts of which are outlined above). The court had to consider the extent of the after-care that should be provided under Section 117 of the Mental Health Act. Under this provision, there is a duty upon the district health authority and the local social services authority to provide after-care services for detained patients who leave hospital. It was alleged in this case that the psychiatrist responsible for monitoring the after-care of the claimant after discharge was negligent in failing to admit him to hospital, thereby preventing the act of homicide which followed. However, the Court of Appeal found that the services being provided by the psychiatrist after discharge

were 'essentially in the sphere of administrative activities in pursuance of a scheme of social welfare in the community'. These, it was stated, were different from the duties owed in the context of the doctor–patient relationship, and there was no common-law duty of care. Furthermore, under Section 124 of the Act, an allegation that inadequate Section 117 services are being provided should be dealt with by way of complaint to the Secretary of State. The court found that the wording of the statute indicated a parliamentary intention that breaches of Section 117 should not give rise to a cause of action in private law. In consequence, it seems that the outlook is bleak for claims resulting from the negligence of psychiatrists, psychologists, and social workers in the context of certain statutory functions of local authorities and health authorities alike.

It must be stressed that, although these cases are referring to English statutory provisions, they raise general issues of principle as to how far statutory bodies are to be controlled by the courts, and, in particular, how far scarce public resources should be subject to depletion by litigation.

(g) Liability towards the mentally ill who harm themselves

As indicated above, the issues of consent to treatment, and competence to both consent and refusal of treatment are central to many legal systems. The case of *Re C* illustrates the importance of the right to self-determination in cases where, on the face of it, there might be a strong temptation to find a patient incompetent because of the nature of the mental illness from which he suffers.[22]

Under Section 63 of the Mental Health Act 1983, treatment can be given for mental disorder for which the patient is detained without the consent of the patient, and competency is irrelevant. In *B* v. *Croydon Health Authority* treatment for mental disorder was construed very widely. In that case the patient was clearly a 'self-harmer', and force-feeding due to her refusal of food was held to be treatment for the mental disorder. If she had been incompetent, then treatment (of any sort, not just for the mental disorder) could have been administered in her best interests. The question then arises as to whether an incompetent patient not detained under the Act who, however, refuses food in an attempt to self-harm must be fed, failing which there would be a breach of the duty of care. It must be noted that the courts are extremely reluctant to question medical decisions about treatment. It is left to clinical judgement to decide whether to give, or withdraw, treatment (see the case of *Airedale NHS Trust* v. *Bland*). (In the same case it was confirmed that treatment includes feeding; see also the Californian case of *Bouvia* v. *Superior Court*.) The decision as to whether treatment is necessary is judged by reference to the Bolam test. In other words if there are good clinical reasons for treating or not treating, and these can be justified by reference to a responsible body of medical opinion, then there will be no liability for failure to treat.

The question that arises is whether the health authority would have been liable nevertheless for failing to force-feed the subject of *B* v. *Croydon Health Authority*,[77] even though she was deemed capable of refusing treatment. It would follow from the general principle of self-determination, well established in English law (see, for example, *Re T* (*Adult: Refusal of Treatment*) and *Re MB* (*Adult: Medical Treatment*),[23]) that such a refusal should be respected. However, recent case law in the context of the duty owed by prisons, and by implication hospitals, has considerably muddied the waters.

In the case of *Kirkham* v. *Chief Constable of Greater Manchester Police* the court took the opportunity to look at the difference between negligent acts and negligent omissions (something that has exercised the minds of tort lawyers for some time) and said that, in this case, responsibility for the omission to prevent the suicide had been assumed by the defendant. The prisoner was said to be 'of unsound mind', which presumably meant that, in the context of the right to self-determination, the prisoner was incapable of exercising his autonomy. In those circumstances the decision is not surprising on its facts. However, the judgment suggested that when one person is in the lawful custody of another, whether that be voluntarily such as a hospital, or involuntarily such as a prison, there is a duty to take all reasonable steps to avoid acts or omissions to prevent reasonably foreseeable injury. Consequently in the case of *Reeves* v. *Commissioner of Police of the Metropolis*, another custodial suicide, the same duty was said to be owed to a 'sane' prisoner. Again, the wording is unfortunate by its reference to sanity, but it can be assumed that this prisoner was deemed to be competent. The police surgeon had stated, after examining the claimant, that there was no evidence of mental disturbance. It must be stressed that it will be necessary in all cases including those concerning hospitals, for there to be evidence that it was reasonable to regard the patient as a suicide risk. Of course, in practice, it may be the case that it is undesirable for hospitals to be venues for acts of suicide. However, it is by no means clear that, in the case of someone who has capacity, there should be a positive duty to prevent suicide any more than there should be a duty owed to prevent that person from smoking. These decisions are invoking a strong form of paternalism. Certainly in the case of hospital patients many competent patients discharge themselves against medical advice whereupon they are free to take any form of self-harming action. It is difficult to see why there should be a positive duty to prevent the competent patient from carrying out the acts on hospital premises.

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11.2

Psychosocial causes of offending

David P. Farrington

Introduction

Scope of this chapter

Offending is part of a larger syndrome of antisocial behaviour that arises in childhood and tends to persist into adulthood. There seems to be continuity over time, since the antisocial child tends to become the antisocial teenager and then the antisocial adult, just as the antisocial adult then tends to produce another antisocial child. The main focus of this chapter is on types of antisocial behaviour classified as criminal offences, rather than on types classified for example as conduct disorder or antisocial personality disorder.

In an attempt to identify causes, this chapter reviews risk factors that influence the development of criminal careers. Literally thousands of variables differentiate significantly between official offenders and non-offenders and correlate significantly with reports of offending behaviour by young people. In this chapter, it is only possible to review briefly some of the most important risk factors for offending: individual difference factors such as high impulsivity and low intelligence, family influences such as poor child rearing and criminal parents, and social influences: socio-economic deprivation, peer, school, community, and situational factors.

I will be very selective in focussing on some of the more important and replicable findings obtained in some of the more methodologically adequate studies: especially prospective longitudinal follow-up studies of large community samples, with information from several data sources (e.g. the child, the parent, the teacher, official records) to maximize validity. The emphasis is on offending by males; most research on offending has concentrated on males, because they commit most of the serious predatory and violent offences. The review is limited to research carried out in the United Kingdom, the United States, and similar Western industrialized democracies. More extensive book length reviews of antisocial behaviour and offending are available elsewhere.[1]

I will refer especially to knowledge gained in the Cambridge Study in Delinquent Development,[2] which is a prospective longitudinal survey of over 400 London males from age 8 to age 40. Fortunately, results obtained in British longitudinal surveys of delinquency are highly concordant with those obtained in comparable surveys in North America, the Scandinavian countries, and New Zealand and indeed with results obtained in British cross-sectional surveys. A systematic comparison of the Cambridge Study with the Pittsburgh Youth Study showed numerous replicable predictors of offending over time and place, including impulsivity, attention problems, low school attainment, poor parental supervision, parental conflict, an antisocial parent, a young mother, large family size, low family income, and coming from a broken family.

Measurement and epidemiology

Offending is defined as acts prohibited by the criminal law, such as theft, burglary, robbery, violence, vandalism, and drug use. It is commonly measured using either official records of arrests or convictions or self-reports of delinquency. The advantages and disadvantages of official records and self-reports are to some extent complementary. In general, official records identify the worst offenders and the worst offences, while self-reports include more of the normal range of delinquent activity. The worst offenders may be missing from samples interviewed in self-report studies. Self-reports have the advantage of including undetected offences, but the disadvantages of concealment and forgetting. By normally accepted psychometric criteria of validity, self-reports are valid. Self-reported delinquency predicted later convictions in the Cambridge Study. In the Pittsburgh Youth Study,[3] the seriousness of self-reported delinquency predicted later court referrals. However, predictive validity was enhanced by combining self-report, parent, and teacher information about offending.

The key issue is whether the same results are obtained with both methods. For example, if official records and self-reports both show a link between parental supervision and delinquency, it is likely that supervision is related to delinquent behaviour (rather than to any biases in measurement). Generally, the worst offenders according to self-reports (taking account of frequency and seriousness) tend also to be the worst offenders according to official records. In the Cambridge Study between ages 15 and 18, 11 per cent of the males admitted burglary, and 62 per cent of these males were convicted of burglary. The predictors and correlates of official and self-reported delinquency were very similar.

Much is known about the epidemiology and development of offending and criminal careers, but there is not space to review these topics here.[4] For example, the prevalence of offending tends

to peak in the teenage years, and an early onset of offending predicts a long criminal career. Offenders tend to be versatile rather than specialized, in committing not only different types of offences but also different types of other antisocial acts. While there is considerable continuity over time, in the sense that the most antisocial people at one age tend also to be the most antisocial at another, only about half of antisocial juveniles tend to become antisocial adults.

Risk factors

A risk factor is defined as a variable that predicts an increased risk of offending. For example, children who experience poor parental supervision have an increased risk of committing offences later on. Since risk factors are defined by their ability to predict later offending, it follows that longitudinal data are required to discover them. Risk factors tend to be similar for many different outcomes, including violent and non-violent offending, mental health problems, alcohol and drug problems, school failure, and unemployment. Protective factors are also important. They are defined as factors that predict a low risk of offending or that counteract risk factors.

An obvious problem is that it is not clear to what extent any risk factor is a cause of offending. It is important to investigate causal mechanisms linking risk factors and offending. The best way of establishing a cause is to carry out a prevention experiment tackling that risk factor; preferably a randomized experiment, because the random assignment of people to conditions in principle controls for all other influences on offending. If a prevention experiment was carried out in which parental supervision was improved, and if offending was reduced as a consequence, this would be powerful evidence that the risk factor of parental supervision truly had a causal effect on offending. However, most knowledge about causes comes from quasi-experimental analyses.

Because of the difficulty of establishing causal effects of factors that vary only between individuals (e.g. gender and ethnicity), and because such factors have no practical implications for prevention (e.g. it is not practicable to change males into females), unchanging variables will not be reviewed here. In any case, their effects on offending are usually explained by reference to other, modifiable, factors. For example, gender differences in offending have been explained on the basis of different socialization methods used by parents with boys and girls, or different opportunities for offending by men and women. Similarly, risk factors that are or might be measuring the same underlying construct as delinquency (e.g. physical aggression) will not be reviewed; the focus is on risk factors that might be causes. For simplicity, risk factors are reviewed one by one. Biological factors are not reviewed. I will not attempt to review additive, interactive, independent, or sequential effects of risk factors, although these are important issues. Nor will I review developmental theories of offending.

Individual factors

Hyperactivity and impulsivity

Hyperactivity and impulsivity are among the most important personality or individual difference factors that predict later offending.[5] Hyperactivity usually begins before age 5 and often before age 2, and it tends to persist into adolescence. It is associated with restlessness, impulsivity and a short attention span, and for that reason has been termed the 'hyperactivity-impulsivity-attention deficit' or HIA syndrome. Related concepts include a poor ability to defer gratification and a short future time perspective.

Many investigators have reported a link between hyperactivity or impulsivity and offending. For example, in the Orebro (Sweden) longitudinal survey,[6] hyperactivity at age 13 (rated by teachers) predicted violent offending up to age 26. The highest rate of violence was among males with both motor restlessness and concentration difficulties. The most extensive research on different measures of impulsivity was carried out by Jennifer White and her colleagues in the Pittsburgh Youth Study. This showed that cognitive or verbal impulsivity (e.g. acts without thinking, unable to defer gratification) was more strongly related to delinquency than was behavioural impulsivity (e.g. clumsiness in psychomotor tests).

In the Cambridge Study, a combined measure of hyperactivity-impulsivity-attention deficit was developed at age 8–10, and it significantly predicted juvenile convictions independently of conduct problems at age 8–10. Hence, HIA is not merely another measure of antisocial personality, but it is a possible cause, or an earlier stage in a developmental sequence leading to offending. Similar constructs to hyperactivity, such as sensation seeking, are also related to delinquency. In the Cambridge Study, the extent to which the boy was daring or took risks at age 8–10, as well as restlessness and poor concentration, significantly predicted convictions and high self-reported offending. Daring was consistently one of the strongest independent predictors of offending.

Low intelligence and attainment

Low intelligence is an important predictor of offending, and it can be measured very early in life. In a prospective longitudinal survey of about 120 Stockholm males,[7] low IQ measured at age 3, significantly predicted officially recorded offending up to age 30. Frequent offenders (with 4 or more offences) had an average IQ of 88 at age 3, whereas non-offenders had an average IQ of 101. All of these results held up after controlling for social class. Similarly, low IQ at age 4 predicted arrests up to age 27 in the Perry preschool project.[8]

In the Cambridge Study, twice as many of the boys scoring 90 or less on a non-verbal IQ test (Raven's Progressive Matrices) at age 8–10 were convicted as juveniles as of the remainder. However, it was difficult to disentangle low intelligence and low school attainment. Low non-verbal intelligence was highly correlated with low verbal intelligence (vocabulary, word comprehension, verbal reasoning) and with low school attainment, and all of these measures predicted juvenile convictions to much the same extent. In addition to their poor school performance, delinquents tended to leave school at the earliest possible age (which was then 15) and to take no school examinations.

Low non-verbal intelligence predicted juvenile self-reported offending to almost exactly the same degree as juvenile convictions, suggesting that the link between low intelligence and delinquency was not caused by the less intelligent boys having a greater probability of being caught. Also, measures of intelligence and attainment predicted measures of offending independently of other variables such as family income and family size. Delinquents often do better on non-verbal performance tests, such as object assembly

and block design, than on verbal tests, suggesting that they find it easier to deal with concrete objects than with abstract concepts.

Low IQ may lead to delinquency through the intervening factor of school failure; the association between school failure and delinquency has been demonstrated consistently in longitudinal surveys. In the Pittsburgh Youth Study, Donald Lynam and his colleagues concluded that low verbal IQ led to school failure and subsequently to self-reported delinquency, but only for African-American boys. Another plausible explanatory factor underlying the link between low IQ and delinquency is the ability to manipulate abstract concepts. Children who are poor at this tend to do badly in IQ tests and in school attainment and they also tend to commit offences, mainly because of their poor ability to foresee the consequences of their offending and to appreciate the feelings of victims. Low IQ may be one aspect of cognitive and neuropsychological deficits in the executive functions of the brain.

Family factors

Child rearing

Many different types of child-rearing methods predict offending. The most important dimensions of child rearing are supervision or monitoring of children, discipline or parental reinforcement, warmth or coldness of emotional relationships, and parental involvement with children. Parental supervision refers to the degree of monitoring by parents of the child's activities, and their degree of watchfulness or vigilance. Of all these child-rearing methods, poor parental supervision is usually the strongest and most replicable predictor of offending. Many studies show that parents who do not know where their children are when they are out, and parents who let their children roam the streets unsupervised from an early age, tend to have delinquent children. For example, in Joan McCord's classic Cambridge–Somerville Study in Boston,[9] poor parental supervision in childhood was the best predictor of both violent and property crimes up to age 45.

Parental discipline refers to how parents react to a child's behaviour. It is clear that harsh or punitive discipline (involving physical punishment) predicts offending. In their follow-up study of nearly 700 Nottingham children, John and Elizabeth Newson[10] found that physical punishment at ages 7 and 11 predicted later convictions; 40 per cent of offenders had been smacked or beaten at age 11, compared with 14 per cent of non-offenders. Erratic or inconsistent discipline also predicts delinquency. This can involve either erratic discipline by one parent, sometimes turning a blind eye to bad behaviour and sometimes punishing it severely, or inconsistency between two parents, with one parent being tolerant or indulgent and the other being harshly punitive.

Cold, rejecting parents tend to have delinquent children, as Joan McCord found in the Cambridge–Somerville Study. More recently, she concluded that parental warmth could act as a protective factor against the effects of physical punishment. Whereas 51 per cent of boys with cold physically punishing mothers were convicted in her study, only 21 per cent of boys with warm physically punishing mothers were convicted, similar to the 23 per cent of boys with warm non-punitive mothers who were convicted. The father's warmth was also a protective factor against the father's physical punishment.

Most explanations of the link between child-rearing methods and delinquency focus on attachment or social learning theories. Attachment theory was inspired by the work of John Bowlby, and suggests that children who are not emotionally attached to warm, loving, and law-abiding parents tend to become offenders. Social learning theories suggest that children's behaviour depends on parental rewards and punishments and on the models of behaviour that parents represent. Children will tend to become offenders if parents do not respond consistently and contingently to their antisocial behaviour and if parents themselves behave in an antisocial manner.

Teenage mothers and child abuse

At least in Western industrialized countries, early child-bearing, or teenage pregnancy, predicts many undesirable outcomes for the children, including low school attainment, antisocial school behaviour, substance use, and early sexual intercourse. The children of teenage mothers are also more likely to become offenders. For example, Morash and Rucker[11] analysed results from four surveys in the United States and the United Kingdom (including the Cambridge Study) and found that teenage mothers were associated with low income families, welfare support, and absent biological fathers, that they used poor child-rearing methods, and that their children were characterized by low school attainment and delinquency. However, the presence of the biological father mitigated many of these adverse factors and generally seemed to have a protective effect. In the Cambridge Study, teenage mothers who went on to have large numbers of children were especially likely to have convicted children. In the Newcastle Thousand-Family Study[12] mothers who married as teenagers (a factor strongly related to teenage childbearing) were twice as likely as others to have sons who became offenders by age 32.

There is considerable intergenerational transmission of aggressive and violent behaviour from parents to children, as Maxfield and Widom[13] found in a retrospective study of over 900 abused children in Indianapolis. Children who were physically abused up to age 11 were significantly likely to become violent offenders in the next 15 years. In the Cambridge–Somerville Study in Boston, Joan McCord found that about half of the abused or neglected boys were convicted for serious crimes, became alcoholics or mentally ill, or died before age 35. In the Rochester Youth Development Study,[14] child maltreatment under age 12 (physical, sexual or emotional abuse or neglect) predicted later self-reported and official offending. Furthermore, these results held up after controlling for gender, race, socio-economic status, and family structure.

Numerous theories have been put forward to explain the link between child abuse and later offending. Timothy Brezina described three of the main ones.[15] Social learning theory suggests that children learn to adopt the abusive behaviour patterns of their parents through imitation, modelling, and reinforcement. Attachment or social bonding theory proposes that child maltreatment results in low attachment to parents and hence to low self-control. Strain theory posits that negative treatment by others generates negative emotions such as anger and frustration, which in turn lead to a desire for revenge and increased aggression. Based on analyses of the Youth in Transition Study, Brezina found limited support for all three theories.

Parental conflict and disrupted families

Many studies show that broken homes or disrupted families predict offending. In the Newcastle Thousand-Family Study, marital disruption (divorce or separation) in a boy's first 5 years doubled his risk of later convictions up to age 32. Similarly, in the Dunedin Study in New Zealand,[16] children who were exposed

to parental discord and many changes of the primary caretaker tended to become antisocial and delinquent. The same study showed that single parent families disproportionally tended to have convicted sons; 28 per cent of violent offenders were from single parent families, compared with 17 per cent of non-violent offenders and 9 per cent of unconvicted boys.

The importance of the cause of the broken home is shown in the UK National Survey of Health and Development.[17] Boys from homes broken by divorce or separation had an increased likelihood of being convicted or officially cautioned up to age 21, in comparison with those from homes broken by death or from unbroken homes. Homes broken while the boy was under age 5 especially predicted offending, whereas homes broken while the boy was between ages 11 and 15 were not particularly criminogenic. Remarriage (which happened more often after divorce or separation than after death) was also associated with an increased risk of offending, suggesting a possible negative effect of step-parents. The meta-analysis by Wells and Rankin[18] also shows that broken homes are more strongly related to delinquency when they are caused by parental separation or divorce rather than by death.

Most studies of broken homes have focussed on the loss of the father rather than the mother, simply because the loss of a father is much more common. Joan McCord in Boston carried out an interesting study of the relationship between homes broken by loss of the natural father and later serious offending of the children. She found that the prevalence of offending was high for boys reared in broken homes without affectionate mothers (62 per cent) and for those reared in united homes characterized by parental conflict (52 per cent), irrespective of whether they had affectionate mothers. The prevalence of offending was low for those reared in united homes without conflict (26 per cent) and—importantly—equally low for boys from broken homes with affectionate mothers (22 per cent). These results suggest that it is not so much the broken home which is criminogenic as the parental conflict which often causes it, and that a loving mother might in some sense be able to compensate for the loss of a father.

In the Cambridge Study, both permanent and temporary separations from a biological parent before age 10 (usually from the father) predicted convictions and self-reported delinquency, providing that they were not caused by death or hospitalization. However, homes broken at an early age (under age 5) were not unusually criminogenic. Separation before age 10 predicted both juvenile and adult convictions, independently of all other factors such as low family income or poor school attainment, and was an important predictor of adult social dysfunction.

Explanations of the relationship between disrupted families and delinquency fall into three major classes. Trauma theories suggest that the loss of a parent has a damaging effect on a child, most commonly because of the effect on attachment to the parent. Life course theories focus on separation as a sequence of stressful experiences, and on the effects of multiple stressors such as parental conflict, parental loss, reduced economic circumstances, changes in parent figures, and poor child-rearing methods. Selection theories argue that disrupted families produce delinquent children because of pre-existing differences from other families in risk factors such as parental conflict, criminal or antisocial parents, low family income, or poor child-rearing methods.

Hypotheses derived from the three theories were tested in the Cambridge Study.[19] While boys from broken homes (permanently disrupted families) were more delinquent than boys from intact homes, they were not more delinquent than boys from intact high conflict families. Overall, the most important factor was the post-disruption trajectory. Boys who remained with their mother after the separation had the same delinquency rate as boys from intact low conflict families. Boys who stayed with their father, with relatives, or with others (e.g. foster parents) had high delinquency rates. These living arrangements were more unstable, and other research shows that frequent changes of parent figures predict offending. It was concluded that the results favoured life course theories rather than trauma or selection theories

Criminal parents

Lee Robins and her colleagues showed that criminal, antisocial and alcoholic parents tend to have delinquent sons.[20] She followed up over 200 males in St. Louis and found that arrested parents tended to have arrested children, and that the juvenile records of the parents and children had similar rates and types of offences. Joan McCord also reported that convicted fathers tended to have convicted sons. She found that 29 per cent of fathers convicted for violence had sons convicted for violence, in comparison with 12 per cent of other fathers, but this may reflect the general tendency for convicted fathers to have convicted sons rather than any specific tendency for violent fathers to have violent sons.

In the Cambridge Study, the concentration of offending in a small number of families was remarkable. Less than 6 per cent of the families were responsible for half of the criminal convictions of all members (fathers, mothers, sons, and daughters) of all 400 families. Having a convicted mother, father, brother, or sister significantly predicted a boy's own conviction. As many as 63 per cent of boys with a convicted parent were themselves convicted up to age 40. Furthermore, convicted parents and delinquent siblings predicted self-reported as well as to official offending. Same-sex relationships were stronger than opposite-sex relationships, and older siblings were stronger predictors than younger siblings. Therefore, there is intergenerational continuity in offending.

It is not entirely clear why criminal parents tend to have delinquent children. In the Cambridge Study, there was no evidence that criminal parents directly encouraged their children to commit crimes or taught them criminal techniques. On the contrary, criminal parents were highly critical of their children's offending; for example, 89 per cent of convicted men at age 32 disagreed with the statement that 'I would not mind if my son/daughter committed a criminal offence'. Also, it was extremely rare for a parent and a child to be convicted for an offence committed together. The main link in the chain between criminal parents and delinquent sons seemed to be poor parental supervision.

There are several possible explanations (which are not mutually exclusive) for why offending tends to be concentrated in certain families and transmitted from one generation to the next. First, there may be intergenerational continuities in exposure to multiple risk factors. For example, each successive generation may be entrapped in poverty, disrupted families, single and/or teenage parenting, and living in the most deprived neighbourhoods. Second, the effect of a criminal parent on a child's offending may be mediated by environmental mechanisms such as poor parental supervision. Third, the effect of a criminal parent on a child's offending may be mediated by genetic mechanisms. Fourth, criminal parents may tend to have delinquent children because of official (police and court) bias against criminal families, who also tend to be known to official agencies because of other social

problems. At all levels of self-reported delinquency in the Cambridge Study, boys with convicted fathers were more likely to be convicted themselves than were boys with unconvicted fathers. However, this was not the only explanation for the link between criminal fathers and delinquent sons, because boys with criminal fathers had higher self-reported delinquency scores and higher teacher and peer ratings of bad behaviour.

Large family size

Large family size (a large number of children in the family) is a relatively strong and highly replicable predictor of offending. It was similarly important in the Cambridge and Pittsburgh studies,[21] even though families were on average smaller in Pittsburgh in the 1990s than in London in the 1960s. In the Cambridge Study, if a boy had four or more siblings by his tenth birthday, this doubled his risk of being convicted as a juvenile, and large family size predicted self-reported offending as well as convictions. It was the most important independent predictor of convictions up to age 32 in a logistic regression analysis.

In the National Survey of Health and Development, Michael Wadsworth found that the percentage of boys who were convicted increased from 9 per cent for families containing one child to 24 per cent for families containing four or more children. The Newsons in their Nottingham study also concluded that large family size was one of the most important predictors of offending. A similar link between family size and antisocial behaviour was reported by Israel Kolvin and his colleagues in their follow-up of Newcastle children from birth to age 33.

There are many possible reasons why a large number of siblings might increase the risk of a child's offending. Generally, as the number of children in a family increases, the amount of parental attention that can be given to each child decreases. Also, as the number of children increases, the household tends to become more overcrowded, possibly leading to increases in frustration, irritation, and conflict. In the Cambridge Study, large family size did not predict delinquency for boys living in the least crowded conditions. This suggests that household overcrowding might be an important intervening factor between large family size and delinquency.

Brownfield and Sorenson[22] reviewed several possible explanations for the link between large families and delinquency, including those focussing on features of the parents (e.g. criminal parents, teenage parents), those focussing on parenting (e.g. poor supervision, disrupted families), and those focussing on economic deprivation or family stress. Another interesting theory suggested that the key factor was birth order: large families include more later-born children, who tend to be more delinquent. Based on an analysis of self-reported delinquency in a Seattle survey, they concluded that the most plausible intervening causal mechanism was exposure to delinquent siblings. In the Cambridge Study, co-offending by brothers was surprisingly common; about 20 per cent of boys who had brothers close to them in age, were convicted for a crime committed with their brother.

Social factors

Socio-economic deprivation

The voluminous literature on the relationship between socio-economic status (SES) and offending is characterized by inconsistencies and contradictions, and some reviewers[23] have concluded that there is no relationship between SES and either self-reported or official offending. British studies have reported more consistent links between low social class and offending. In the UK National Survey of Health and Development, the prevalence of official juvenile delinquency in males varied considerably according to the occupational prestige and educational background of their parents, from 3 per cent in the highest category to 19 per cent in the lowest. It has been suggested that low SES families tend to produce delinquent children because their child-rearing tends to be poor.

Numerous indicators of SES were measured in the Cambridge Study, both for the boy's family of origin and for the boy himself as an adult, including occupational prestige, family income, housing, and employment instability. Most of the measures of occupational prestige (based on the Registrar General's scale) were not significantly related to offending. Low SES of the family when the boy was aged 8–10 significantly predicted his later self-reported but not his official delinquency. More consistently, low family income and poor housing predicted official and self-reported, juvenile and adult, offending.

It was interesting that the peak age of offending, at 17–18, coincided with the peak age of affluence for many convicted males. In the Cambridge Study, convicted males tended to come from low income families at age 8 and later tended to have low incomes themselves at age 32. However, at age 18, they were relatively well paid in comparison with non-delinquents. Whereas convicted delinquents might be working as unskilled labourers on building sites and getting the full adult wage for this job, non-delinquents might be in poorly paid jobs with prospects, such as bank clerks, or might still be students. These results show that the link between income and offending is quite complex.

Socio-economic deprivation of parents is usually compared with offending by children. However, when the children grow up, their own socio-economic deprivation can be related to their own offending. In the Cambridge Study, an unstable job record of the boy at age 18 was one of the best independent predictors of his later convictions between ages 21 and 25. Also, having an unskilled manual job at age 18 was an important independent predictor of adult social dysfunction and antisocial personality at age 32.

Between ages 15 and 18, the study boys were convicted at a higher rate when they were unemployed than when they were employed, suggesting that unemployment in some way causes crime, and conversely that employment may lead to desistance from offending.[24] Since crimes involving material gain (e.g. theft, burglary, robbery) especially increased during periods of unemployment, it seems likely that financial need is an important link in the causal chain between unemployment and crime.

Peer influences

Having delinquent friends is an important predictor of later offending; peer delinquency and gang membership predicted self-reported violence in the Seattle Social Development Project.[25] Delinquent acts tend to be committed in small groups (of two or three people, usually) rather than alone. Large gangs are comparatively unusual. In the Cambridge Study, the probability of committing offences with others decreased steadily with age. Before age 17, boys tended to commit their crimes with other boys similar in age and living close by. After age 17, co-offending became less common.[26]

The major problem of interpretation is whether young people are more likely to commit offences while they are in groups than

while they are alone, or whether the high prevalence of co-offending merely reflects the fact that, whenever young people go out, they tend to go out in groups. Do peers tend to encourage and facilitate offending, or is it just that most kinds of activities out of the home (both delinquent and non-delinquent) tend to be committed in groups? Another possibility is that the commission of offences encourages association with other delinquents, perhaps because 'birds of a feather flock together' or because of the stigmatizing and isolating effects of court appearances and institutionalization. Terence Thornberry and his colleagues in the Rochester Youth Development Study concluded that there were reciprocal effects, with delinquent peers causing delinquency and delinquency causing association with delinquent peers.

In the Pittsburgh Youth Study, the relationship between peer delinquency and a boy's offending was studied both between individuals (e.g. comparing peer delinquency and offending of boy X with peer delinquency and offending of boy Y at a particular age and then aggregating these correlations over all ages) and within individuals (e.g. comparing peer delinquency and offending of boy X at different ages and then aggregating these correlations over all individuals). Peer delinquency was the strongest correlate of offending in between-individual correlations but did not predict offending within individuals.[27] In contrast, poor parental supervision, low parental reinforcement, and low involvement of the boy in family activities predicted offending both between and within individuals. It was concluded that these three family variables were the most likely to be causes, whereas having delinquent peers was most likely to be an indicator of the boy's offending.

Associating with delinquent friends at age 14 was an important independent predictor of convictions at the young adult ages in the Cambridge Study. Also, the recidivists at age 19 who ceased offending differed from those who persisted, in that the desisters were more likely to have stopped going round in a group of male friends. Furthermore, spontaneous comments by the youths indicated that withdrawal from the delinquent peer group was an important influence on ceasing to offend. Therefore, continuing to associate with delinquent friends may be a key factor in determining whether juvenile delinquents persist in offending as young adults or desist.

School influences

The prevalence of delinquency among students varies dramatically between different secondary schools, as Michael Power and his colleagues[28] showed many years ago in London. Characteristics of high delinquency-rate schools are well-known. For example, such schools have high levels of distrust between teachers and students, low commitment to school by students, and unclear and inconsistently enforced rules. However, what is far less clear is how much of the variation between schools should be attributed to differences in school organization, climate and practices, and how much to differences in the composition of the student body.

In the Cambridge Study, attending a high delinquency-rate school at age 11 significantly predicted a boy's later offending and antisocial personality scores. The effects of secondary schools on delinquency were investigated by following boys from their primary schools to their secondary schools. The best primary school predictor of juvenile delinquency was the rating of the boy's troublesomeness at age 8–10 by peers and teachers, showing the continuity in antisocial behaviour. The secondary schools differed dramatically in their official delinquency rates, from one school with 21 court appearances per 100 boys per year to another where the corresponding figure was only 0.3. Moreover, going to a high delinquency-rate secondary school was a significant predictor of later convictions. It was, however, very noticeable that the most troublesome boys tended to go to the high delinquency-rate schools, while the least troublesome boys tended to go to the low delinquency-rate schools. Most of the variation between schools in their delinquency rates could be explained by differences in their intakes of troublesome boys. The secondary schools themselves had only a very small effect on the boys' offending.

The most famous study of school effects on delinquency was also carried out in London, by Michael Rutter and his colleagues.[29] They studied 12 comprehensive schools, and again found big differences in official delinquency rates between them. High delinquency-rate schools tended to have high truancy rates, low ability pupils, and low social class parents. However, the differences between the schools in delinquency rates could not be entirely explained by differences in the social class and verbal reasoning scores of the pupils at intake (age 11). Therefore, they must have been caused by some aspect of the schools themselves or by other, unmeasured factors.

In trying to discover which aspects of schools might be encouraging or inhibiting offending, Rutter and his colleagues found that the main school factors that were associated with delinquency were a high amount of punishment and a low amount of praise given by teachers in class. Unfortunately, it is difficult to know whether much punishment and little praise are causes or consequences of antisocial school behaviour, which in turn may be linked to offending outside school. In regard to other outcome measures, they argued that an academic emphasis, good classroom management, the careful use of praise and punishment, and student participation were important features of successful schools.

Community influences

Offending rates vary systematically with area of residence. For example, the classic studies by Shaw and McKay[30] in Chicago and other American cities showed that juvenile delinquency rates (based on where offenders lived) were highest in inner city areas characterized by physical deterioration, neighbourhood disorganization, and high residential mobility. A large proportion of all offenders came from a small proportion of areas, which tended to be the most deprived. Furthermore, these relatively high delinquency rates persisted over time, despite the effect of successive waves of immigration and emigration of different national and ethnic groups in different areas.

Living in a deprived neighbourhood (whether based on parent ratings or on census measures of poverty, unemployment and female-headed households) significantly predicts convictions and self-reported offending. However, it is difficult to establish how much the areas themselves influence antisocial behaviour and how much it is merely the case that antisocial people tend to live in deprived areas (e.g. because of their poverty or public housing allocation policies). It has been suggested that neighbourhoods have only indirect effects on offending because of their effects on individuals and families. However, Robert Sampson and his colleagues[31] argued that a low degree of 'collective efficacy' of a neighbourhood (a low degree of informal social control) caused high crime rates.

One key question is why crime rates of communities change over time, and to what extent this is a function of changes in the

communities or in the individuals living in them. Answering this question requires longitudinal research in which both communities and individuals are followed up. The best way of establishing the impact of the environment is to follow people who move from one area to another. For example, in the Cambridge Study, moving out of London led to a significant decrease in convictions and self-reported offending. This decrease may have occurred because moving out led to a breaking up of co-offending groups, or because there were fewer opportunities for crime outside London.

Situational influences

It might be argued that all the risk factors reviewed so far in this section—individual, family, socio-economic, peer, school, and community—essentially influence the development of a long-term individual potential for offending. In other words, they contribute to between-individual differences: why some people are more likely than others, given the same situational opportunity, to commit a crime. Another set of influences—situational factors—explain how the potential for violence becomes the actuality in any given situation. Essentially, they explain short-term within-individual differences: why a person is more likely to commit crimes in some situations than in others. Situational factors may be specific to particular types of crimes: robberies as opposed to rapes, or even street robberies as opposed to bank robberies.

The most popular theory of offending events is a rational choice theory suggesting that they occur in response to specific opportunities, when their expected benefits (e.g. stolen property, peer approval) outweigh their expected costs (e.g. legal punishment, parental disapproval). For example, Clarke and Cornish[32] suggested that residential burglary depended on such influencing factors as whether a house was occupied, whether it looked affluent, whether there were bushes to hide behind, whether there were nosy neighbours, whether the house had a burglar alarm, and whether it contained a dog. A related theory is the 'routine activities' idea of Cohen and Felson.[33] They suggested that, for a predatory crime to occur, the minimum requirement was the convergence in time and place of a motivated offender and a suitable target, in the absence of a capable guardian. They argued that predatory crime rates were influenced by routine activities that satisfied basic needs such as food and shelter. Changes in routine activities led to changing opportunities for crime. For example, the increasing number of working women meant that more homes were left unattended during the day.

Much work on describing situations leading to violence has been carried out in the United Kingdom under the heading of crime analysis. This begins with a detailed analysis of patterns and circumstances of crimes and then proceeds to devise, implement, and evaluate crime reduction strategies. For example, it was found that most street robberies in London occurred in predominantly ethnic minority areas, and most offenders were 16–19 year old Afro-Caribbean males.[34] The victims were mostly Caucasian females, alone, and on foot. Most offences occurred at night, near the victim's home. The main motive for robbery was to get money, and the main factor in choosing victims was whether they had a wealthy appearance.

In their Montreal longitudinal study of delinquents, LeBlanc and Frechette[35] provided detailed information about motives and methods used in different offences at different ages. For example, for violence at age 17, the main motivation was utilitarian or rational. For all crimes, however, the primary motivation changed from hedonistic (searching for excitement, with co-offenders) in the teenage years to utilitarian (with planning, psychological intimidation, and use of instruments such as weapons) in the twenties. In the Cambridge Study, motives for physical fights depended on whether the boy fought alone or with others.[36] In individual fights, the boy was usually provoked, became angry, and hit out to hurt his opponent and to discharge his own internal feelings of tension. In group fights, the boy often said that he became involved to help a friend or because he was attacked, and rarely said that he was angry. The group fights were more serious, occurring in bars or streets, and they were more likely to involve weapons, produce injuries, and lead to police intervention. Fights often occurred when minor incidents escalated, because both sides wanted to demonstrate their toughness and masculinity and were unwilling to react in a conciliatory way.

Much is known about the situations in which violence occurs. For example, in Sweden, violence preceded by situational arguments typically occurs in streets or restaurants, while violence preceded by relationship arguments typically occurs in homes.[37] In England, stranger assaults typically occur in streets, bars, or discotheques, non-stranger assaults typically occur at home or work, and robberies typically occur in the street or on public transport. Most violence occurs on weekend nights around pubs and clubs, and involves young males who have been drinking.[38] More research on situational influences on offending needs to be incorporated in prospective longitudinal studies, in order to link up the developmental and situational perspectives.

Conclusions

Offending is one element of a larger syndrome of antisocial behaviour that arises in childhood and tends to persist into adulthood, with numerous different behavioural manifestations. However, while there is continuity over time in antisocial behaviour, changes are also occurring. It is commonly found that about half of a sample of antisocial children go on to become antisocial teenagers, and about half of antisocial teenagers go on to become antisocial adults. More research is needed on factors that predict these changes over time. Research is especially needed on changing behavioural manifestations and developmental sequences at different ages. More efforts should especially be made to identify factors that protect vulnerable children from developing into antisocial teenagers.

A great deal has been learned in the last 20 years, particularly from longitudinal surveys, about risk factors for offending and other types of antisocial behaviour. Offenders differ significantly from non-offenders in many respects, including impulsivity, intelligence, family background, and socio-economic deprivation. These differences are present before, during, and after criminal careers. While the precise causal chains that link these factors with antisocial behaviour, and the ways in which these factors have independent, interactive, or sequential effects, are not known, it is clear that individuals at risk can be identified with reasonable accuracy. In order to advance knowledge about human development and criminal careers, new multiple-cohort longitudinal studies are needed.[39]

The identified risk factors for offending should be targeted in prevention programmes. Risk-focussed prevention has great

potential for crime reduction.[40] The continuity of antisocial behaviour from childhood to adulthood suggests that prevention efforts should be implemented early in life. Because of the link between offending and numerous other social problems, any measure that succeeds in reducing offending will have benefits that go far beyond this. Any measure that reduces offending will probably also reduce alcohol abuse, drunk driving, drug abuse, sexual promiscuity, family violence, truancy, school failure, unemployment, marital disharmony, and divorce. It is clear that problem children tend to grow up into problem adults, and that problem adults tend to produce more problem children. Continued efforts are urgently needed to advance knowledge about offending and antisocial behaviour, and to tackle the roots of crime.

Further information

For extensive reviews of risk factors and interventions, see *Saving Children from a Life of Crime: Early Risk Factors and Effective Interventions* by D. P. Farrington and B. C. Welsh (Oxford University Press, 2007).

For extensive information about the Cambridge Study, see *Criminal Careers up to Age 50 and Life Success up to Age 48: New Findings from the Cambridge Study in Delinquent Development* by D. P. Farrington and colleagues (Home Office Research Study No. 299), available from www.homeoffice,gov.uk/rds.

For other reviews of psychosocial causes of offending, see *Antisocial Behaviour by Young People*, by M. Rutter, H. Giller, and A. Hagell (Cambridge University Press, 1998).

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11.3

Associations between psychiatric disorder and offending

Contents

11.3.1 Associations between psychiatric disorder and offending

Lindsay Thomson and Rajan Darjee

Introduction

The associations between psychiatric disorder and offending are complex. There has been a great deal of research into certain disorders and violent offending particularly over the last two decades. In summary, this has found a clear and consistent association between schizophreniform psychoses and violence, the importance of premorbid antisocial behaviour in predicting future violence, and the adjunctive effect of co-morbid substance misuse and antisocial personality disorder in the prevalence of violence. In addition, it has allowed the development of neuropsychiatric models to begin to explain violence in the context of mental disorder. Substance use disorders and learning disability are discussed in Chapters 11.3.2 and 11.3.3.

Mental disorder and offending: a problematic relationship

Criminal behaviour is common in our society but there is evidence that violent crime rates have declined in Europe and North America over the last decade.[1] Mental disorders are also common. It is important to study the overlap between mental disorder and offending to consider those mental health and criminogenic factors that may be amenable to change. The social and economic factors relevant to offending are discussed in Chapter 11.2.

Before considering any associations between mental disorder and offending it is useful to consider the methodological problems in studying these:

- Offences are man-made concepts and not static. For example, many jurisdictions have created laws against stalking in the last twenty years which did not previously exist.
- Psychiatrists see a limited range of offenders but often base their research on these.
- Research is generally carried out on a captive population in prison or secure hospital. Offenders in these settings are likely to include those with characteristics that disadvantage them in the criminal justice system, for example ethnic status, low economic status, homelessness, unemployment, and mental illness.
- The generalizability of any findings must be queried given that offending is dependent on the wider social context such as rates of unemployment or crime, prevalence of substance misuse, and weapon carrying culture.
- It can be difficult to standardize populations studied, and severity of crimes.
- Criminal and mental health records may be unreliable.

Evidence for neurobiological determinants of offending or aggressive behaviour

There is resistance to any oversimplified idea of seeking a genetic or neuropsychological explanation to offending behaviour as a whole but research in this area is expanding slowly. Neuropsychological abnormalities are commonly found in offenders and there is evidence for specific brain deficits in aggressive or violent behaviour.[2] These findings include:

- An association between specific traumatic damage to the frontal lobes, particularly orbitofrontal injury, and poor impulse control and aggressive outbursts.
- Abnormalities on neuropsychological tests of frontal lobe function in aggressive and antisocial subjects, indicating prefrontal executive dysfunction.

- Abnormalities found in clinical neurological testing of offenders. Antisocial behaviour is associated with EEG abnormalities particularly frontal slowing and these are commonly found in more than half of prisoners with a history of repetitive violence. Clinical signs of frontal lobe dysfunction are also associated with recurrent aggression.
- Neuroimaging changes. Structural and functional studies examining patients in forensic services and patients with antisocial personality disorder have consistently found changes in the frontal lobes of aggressive patients, typically reduced prefrontal cortical size and activity. Predatory rather than impulsive, emotionally charged (affective) perpetrators of homicide show functional patterns of blood flow similar to controls suggesting that these neuroimaging findings are relevant to impulsive or affective aggression rather than premeditated, purposeful violence. Two groups of people with aggressive behaviour are postulated: the first with an acquired frontal lobe lesion due to injury or disease which impairs social judgement, risk avoidance, and empathy; the second, shows increased aggressive behaviour associated with deficits in executive functioning correlating with dorsolateral prefrontal dysfunction which may occur in foetal or birth brain injury, developmental learning disorders, attention deficit hyperactivity disorder, substance misuse, and antisocial personality disorder with episodic aggressive dyscontrol.
- In addition, there is evidence for biochemical abnormalities. Reduced serotonin function is largely related to impulsivity rather than directly to violence.[3] Serotonin has a role in emotional states such as impulsiveness, aggression, anxiety, and depression. This has provided potential avenues for treatment with selective serotonin reuptake inhibitor medication.[4] Cortisol abnormalities have been recognized for a long time and more recently dietary insufficiencies have been explored.[5]
- Lastly, the role of genetics in offending behaviour has been examined through family, twin, and adoption studies.[6] Adoption studies have found a consistent association between biological parents and adoptee for property offences but a more complex association for violent crime with a relationship discovered in the Danish birth cohort study between paternal violence and adoptee schizophrenia. A link between a genotype and disturbed behaviour was found in maltreated male children. Those with the gene encoding the neurotransmitter metabolizing enzyme monoamine oxidase A (MAOA) moderated the effect of maltreatment and had reduced antisocial behaviour in later life.[7] MAOA genes have a known association with aggression in animals and humans. The low expression variant leads to increased aggression, limbic volume reductions and hyper-responsive amygdala during emotional arousal with decreased reactivity of regulatory prefrontal regions.[8] Twin studies have shown that antisocial behaviour in early childhood particularly when associated with callous unemotional traits has a strong genetic influence which weakens in antisocial behaviour displayed initially in adolescence when environmental influences are important.[9]

Such changes are not a necessarily a cause for aggressive behaviour which is dependent on so many environmental factors, nor do they necessarily predict aggressive behaviour but they are important to study in that they indicate potential management strategies.

Clinical implications of the relationship between mental disorder and offending

The nature of the relationship between psychiatric disorder and offending is complex both at an individual and population level, and has important implications at both. Epidemiological data point to the following conclusions:

- Schizophrenia, personality disorder, and substance related disorders are significantly overrepresented in offenders.
- A number of factors associated with violence and offending in the non-mentally disordered are relevant to violence and offending in the mentally disordered.
- Amongst offenders there is the need to provide psychiatric assessment and treatment for significant numbers of people with mental disorder.
- Amongst mentally disordered offenders it is important to address criminogenic factors as well as providing traditional clinical treatment
- Appropriate treatment and preventative measures may prevent some offending and violence by individuals with mental disorders

It is important to know and understand the epidemiological research, but also to know and understand the patient that is being assessed. Not all factors of relevance to offending in people with mental disorders act in the same way in every case, and some factors not found to be of relevance in study samples may be crucial in individual cases. There is no simple or straightforward approach to such an assessment, and narrowing one's focus to a tick list of a few factors (perhaps from an actuarial tool) is at best lazy and at worst negligent. The psychiatrist should be able to articulate a formulation incorporating the factors which account for previous offending and which may be of relevance to future offending.

Every violent act involves a perpetrator, a victim, and a context, and this is no different where mental disorder is involved. A number of factors in the perpetrator, victim, and context in any violent incident are relevant to that violent act, and these three interact with each other. A simple example is that a drunk aggressive victim may frighten a suspicious impulsive perpetrator in the context of alcohol intoxication and an available knife. Where a person with mental disorder is violent one should not assume the former straightforwardly causes the latter. Sometimes mental disorder is the major determinant of an offence and at others mental disorder is entirely coincidental. In most cases mental disorder is one of a number of interacting factors (Fig. 11.3.1.1). Even where an offence seems explicable by psychotic hallucinations or delusions it is essential to consider wider factors (e.g. alcohol and substance misuse, social networks and personality).[10] A thorough assessment of these factors is crucial when evaluating criminal responsibility (see Chapter 11.1), assessing risk of future offending (see Chapter 11.15) and planning treatment for patients who have been violent. Treating mental disorder without addressing these factors will not adequately address the risk of further offending.

Scizophrenia and offending

For over a decade, studies have consistently shown a small but significant association between schizophrenia and violence with an

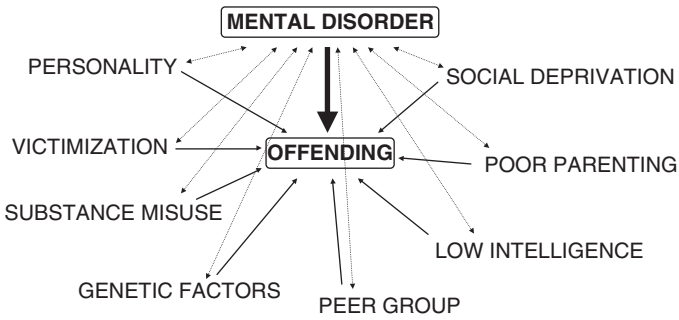

Fig. 11.3.1.1 A schematic representation of the complex interplay between factors of relevance to offending in people with mental disorder.

increased risk of violence of between 2–4 for men and 6–8 for women after controlling for marital status, socio-economic background and substance abuse.[11] The proportion of violent crimes in the population of Northern European countries attributable to individuals with severe mental illness is around 5 per cent.[12] A systematic review of mental disorder in 23,000 prisoners found psychoses in 3.7 per cent of men and 4 per cent of women which was 2–4 times more than in the general population. Aggression in first episode psychosis occurs consistently in one-third of cases.[13]

Two patterns of aggression associated with schizophrenia are seen: firstly, those in whom features of conduct disorder (20-40 per cent) and later antisocial personality disorder predate the onset of overt schizophrenia; and secondly, those in whom violence occurs at a later stage.[11] Psychotic symptoms and violence in childhood are strongly related to later violence, as is a history of conduct disorder.[14]

Studies of people convicted of homicide have consistently shown an excess of schizophrenia with rates of 5–15 per cent.[15,16] Family members are significantly more likely to be the victim than strangers. Those with comorbid antisocial personality disorder are less likely to be actively psychotic at the time of the offence, and more likely to be intoxicated and to kill non-relatives.

Neurobiological correlates of schizophrenia and violence

Naudts and Hodgins[17] examined neurobiological correlates of schizophrenia and violence. The literature is small, as are the sample sizes, and the measures used are diverse. At least one-fifth of men with schizophrenia show antisocial behaviour from childhood onwards. Overall, these men perform better on tests of specific executive functions and verbal skills despite greater impulsivity; and worse on tests of orbitofrontal functions than men with schizophrenia alone. Structural brain imaging studies of men with schizophrenia and a history of repetitive violence found reduced whole brain and hippocampal volumes; impaired connectivity between the orbitofrontal cortex and the amygdala; and structural abnormalities of the amygdala. Functional neuroimaging studies found reduced prefrontal cerebral blood flow during completion of a test of executive function in violent patients. This reduction in blood flow may result in loss of inhibition and therefore aggression, or it may reflect that these violent men found the test easier and did not need increased blood flow to complete it. Studies of acquired brain lesions show that ventromedial orbitofrontal cortex is necessary for inhibiting impulsive decision-making and behaviour and for physiological anticipation of secondary inducers such as punishment. Studies at different ages suggest that an intact amygdala in early life is necessary for the normal development of this orbitofrontal system to recognize and process emotions. This fits with the work of Silver *et al.*[18] who found that patients with schizophrenia with a history of severe violence differ from non-violent patients with schizophrenia in their perception of the intensity of emotions but not cognitive function. Failure to assess the intensity of emotions may contribute to conflict generation, failure to recognize resolution signals, conflict escalation, and violence.

Clinical features

People with schizophrenia may be violent because of their psychotic experiences, impaired judgment and impulse control, or situational factors. The evidence of the effect of symptoms is conflicting however, probably because of the way questions are asked; failure to take account of affective symptoms; variation in the timing between symptoms and violence; failure to control for medication, compulsion and previous violence; and variations in statistical procedures.[19] Swanson *et al.*[20] in a study of 1410 people with schizophrenia in a six month period found 19.1 per cent had been violent and this was serious in 3.6 per cent of cases. Positive symptoms increased the risk of minor and serious violence whereas negative symptoms decreased the risk of serious violence perhaps because these individuals lived alone. Serious violence was associated with psychotic and depressive symptoms, childhood conduct problems, and victimization. Severe psychotic symptoms and threat-control override (TCO) were antecedents of violent behaviour of patients in the community even after controlling for psychopathy and substance abuse.[21] TCO consists of persecutory delusions, passivity phenomena and thought insertion, resulting in perceived personal threat combined with loss of self control. Excessive perceptions of threat explained violence in people with schizophrenia-spectrum disorder alongside a history of conduct disorder.[22]

Hallucinations, acute suicidal ideation, acute conflict and stressors such as separations or housing problems, and lack of insight have all been associated with an increased risk of violence.[23]

Co-morbidity

The likelihood of violence in patients with schizophrenia increases in the presence of comorbid substance use (3-fold in men, 16-fold in women) or personality disorder (4-fold men, 18-fold women).[11] Swanson *et al.*[20] found however, that the effects of substance abuse were non significant after controlling for age, positive symptoms of schizophrenia, childhood conduct problems and recent victimization thereby suggesting that the effects of substance abuse on violence may be mediated by these factors. There is a strong association between comorbid antisocial personality disorder and substance abuse in patients with schizophrenia with common origins in conduct disorder.[24] Further evidence for this is found in neuropsychological tests. Patients with comorbid schizophrenia and substance abuse perform as well as those with schizophrenia alone on neurological tests although it is known that the brains of men with schizophrenia are particularly sensitive to effects of drugs and alcohol. It has been suggested that these men belong to the antisocial group who do better on these tests at the onset of their schizophrenia.[17]

Management

The care of patients with schizophrenia and a history of serious violence is centred around ongoing assessment and management of factors contributing to risk of violence and treatment of the schizophrenic illness. Predictors of violence include recent assault, a previous violent conviction, lower educational attainment and attending special education, a personal and family history of alcohol abuse, and lower but normal range IQ.[25,26] The management of schizophrenia is discussed in Chapter 4.3.8 but in patients with a history of violence methods of legal compulsion for detention and treatment, and programmes to address comorbid and criminogenic needs are particularly important. There is evidence that cognitive behavioural therapy can modify delusional beliefs that have lead to violence[27] and that clozapine has a particular role in the treatment of aggression in patients with schizophrenia separate from its antipsychotic or sedative components.[28]

Outcome

There is no indication that deinstitutionalization has lead to increased offending by patients with schizophrenia. Rates of violence have increased but in proportion to the increase in society as a whole and with increased comorbid alcohol abuse.[29] Outcome studies of patients transferred from high security found a recidivism rate of 34 per cent and 31 per cent in England and Scotland respectively, and a violent recidivism rate of 15 per cent and 19 per cent after ten years.[30, 31] Patients with a primary diagnosis of schizophrenia during a follow up period of 8 years had a recidivism rate of 15 per cent and a violent recidivism rate of 5 per cent.

Delusional disorders

Delusional disorders are described in Chapter 4.4. There are a number of subtypes including somatic, grandiose, mixed and unspecified but it is particularly the jealous, erotomanic and persecutory forms that are associated with offending and violence. Individuals with delusional disorders remain organized allowing them to target and plan any violence more effectively. Sixty percent of people with delusional jealousy are violent towards their partners. In erotomania the patient has the delusional belief that s/he is loved from afar by another and may attack individuals perceived as standing between them and their loved one, or the object of their affections if they feel slighted. Erotomania is associated with anger, harassment, stalking, and violence.[32]

Delusional disorders are found in excess in homicide (6-fold increase) and are associated with stalking. Stalking is persistent harassment in which a person repeatedly intrudes on another in an unwelcome manner that evokes fear or disquiet. One study of stalkers found that 30 per cent had a delusional disorder, 10 per cent schizophrenia, less than 5 per cent bipolar disorder or anxiety; 50 per cent had a personality disorder and 25 per cent abused substances. The classification of stalkers is based on their motivation. It is the rejected stalkers who pursue ex-intimates for reconciliation, revenge or both (includes delusional jealousy); intimacy seekers (includes erotomania); and the resentful stalker who pursue victims as revenge for an actual or perceived injury (includes persecutory) who are most likely to have a delusional disorder. This is not the case for incompetent suitors or predatory stalkers. See Chapter 11.10

Risk of harm to others can be reduced by treating the individual's delusional disorder with medication; controlling his or her environment by use of hospital or legal orders to restrict movement; and by advice to any potential victim on protective measures. There have been no randomized controlled trials of the treatment of delusional disorders but novel antipsychotics are now most commonly used due to their lower side-effect profile and better compliance, and it is recognized that pimozide has no particular role in the treatment of delusional disorder.[33] Cognitive behavioural therapy may assist compliance and insight but this has not been systematically demonstrated. Eye movement desensitization and reprocessing (EMDR) interwoven with cognitive therapy has been reported as a treatment of morbid jealousy. It is essential to warn any identified potential victims and such a breach of confidentiality is permitted by medical governing bodies.

Organic disorders

Organic disorders may be related to offending directly because of the brain injury with disinhibition and impaired judgement, or secondarily to socio-economic deprivation and exclusion.

Traumatic brain injury (TBI)

There is a 4-fold increase in the relative risk of developing any psychiatric illness in the six months post moderate to severe TBI and almost 3-fold in mild TBI excluding those with a history of mental illness. Common psychiatric sequelae of traumatic brain injury include mood (50 per cent), anxiety (25 per cent) and substance use disorders (28 per cent) along with post traumatic stress disorder and post traumatic brain injury attention deficit hyperactivity disorder in children, in addition to any cognitive problems.[34]

Posttraumatic irritability occurs in up to 70 per cent of people with TBI. Aggressive behaviour occurs in approximately one-quarter and this is significantly increased by the presence of major depression or substance abuse. Psychological tests of executive function and neuroimaging suggest that defective pre-frontal modulation of medial limbic structures may explain aggressive and impulsive behaviour seen in these patients. TBI patients who display aggression are characterized by significantly more impulsivity, disinhibition and social withdrawal; and poorer drive and motivation.[35] A study of sex offenders found that half had a history of head injury leading to loss of consciousness with significant neurological impairment in a quarter.[36] The head injury group offended more against adults than children, had more exhibiting and wide ranging sexual behaviours, and abused more substances than their non TBI sex offender controls.

Epilepsy

Contrary to previous views, the evidence now suggests that there is no association between epilepsy and criminal behaviour. A systematic review of epilepsy in prisoners found a prevalence rate of about 1 per cent which is comparable with a community sample of a similar age and gender.[37] Epilepsy is considered in detail in Chapter 11.3.3.

Dementia

A diagnosis of dementia and being charged with a sexual offence were factors most likely to distinguish those over 60 years of age referred by courts for a psychiatric assessment in Sweden compared to their younger counterparts although all forms of psychoses

(31 per cent), personality disorder (20 per cent), and substance use disorders (14.8 per cent) were more frequently diagnosed than dementia (7 per cent).[38] Offending is much less common in the elderly. Physical aggression in dementia however, is not uncommon and may be associated with exposure of intellectual deficits during testing (catastrophic reaction), severe cognitive impairment, impaired expression and comprehension, a history of premorbid aggression, physical illness particularly involving delirium or pain, post ictal confusion, depression and changes within the environment. Frontotemporal or orbitotemporal degeneration are more frequently associated with aggression in dementia, as are reduced serotonin and acetylcholine.[39]

Autism spectrum disorders

Autism spectrum disorders (ASD) (including autism and Asperger's syndrome) are described in Chapter 9.2.3. In the severe form (autism) incapacity is frequently major and affected individuals are consequently unlikely to come into contact with the judicial system. The frequency of the lesser forms is inadequately researched. Although the majority of individuals with ASD are law abiding—and many indeed may be less likely to offend as a result of 'concrete' rule-based thinking—they are found in excess in high security psychiatric settings. Those who offend may do so because of feelings of resentment caused by bullying or rejection at school or in the community, because of an over-sensitivity to sound, occasionally because of a powerful interest in guns, fire or killing[40] or because of a lack of appreciation of social norms. Theft may occur in order to pursue a particular interest or obsession.

Personality disorder

The link between personality disorder and offending and the role of psychiatry in managing personality disordered offenders is complicated by a number of issues: poor diagnostic systems; variable and subjective approaches to assessment; confusing legislative approaches seeking to define and impose measures on certain individuals; societal preoccupation with 'psychopaths' and 'sexual predators'; and uncertainty as to what treatment, if any, is effective. The concept of anti-social personality disorder, introduced in DSM-III and rehashed in DSM-IIIR and DSM-IV, has been unhelpful. Its reliance on socially deviant behaviour, rather than personality traits, reliably demarcates a broad heterogeneous group with varying underlying psychopathology and social problems. Personality disorder should not be diagnosed primarily on the basis of repeated anti-social behaviour. Assessment of personality in offenders should be based on pervasive and persistent emotional experience and expression, thoughts about self and others, interactions with others and behavioural control. Personality disordered offenders show a range of personality pathology rarely falling neatly into the diagnostic boxes of ICD 10 or DSM-IV. Focussing on personality disorder as a clinical entity misses relevant developments in assessment and management of such individuals from research and practice in criminal justice.

Psychopathy

The term psychopathy has a long and chequered history (see Chapter 4.12.1). Its clinical use should probably be reserved for individuals fulfilling Hare's criteria[41] (based on Cleckley's 'Mask of Sanity'[42] describing superficial, self-centred, callous, parasitic, impulsive, aggressive, predatory individuals. Hare operationalized psychopathy with the Psychopathy Check List-Revised (PCL-R). Interview and case file material is used to assess 20 items on a 3-point scale giving a maximum total score of 40. In North America a cut-off of 30 is used to diagnose psychopathy. In Europe the cut-off may be lower, but this is a contentious area. The total score may be useful for research purposes, but the profile of characteristics is important in clinical assessment and formulation. Underlying the construct are emotional, interpersonal and behavioural domains. Hare originally described two underlying factors: Factor 1 (emotional and interpersonal) correlated with narcissistic and histrionic disorders, and Factor 2 (socially deviant life style and behaviour) correlated with anti-social and borderline disorders. This has been refined into a four factor model subdividing factor 1 into interpersonal and affective domains, and factor 2 into lifestyle and antisocial behaviour domains. It has been suggested that the antisocial behaviour domain is not core to the condition.[43] Psychopathy is a narrower construct than dissocial (ICD 10) or antisocial (DSM-IV) personality disorder, so research findings cannot be extrapolated. It is an important concept in theory, research and practice concerning offender management and recidivism. But psychopathy only focuses on a limited, albeit important, set of personality traits of relevance in assessing offenders. Comprehensive consideration of personality pathology involves broader assessment. Patrick's book[44] gives an authoritative account of psychopathy.

Relationship between personality disorder and offending

Personality disorder rates in prisoners range from 10 per cent[45] to 78 per cent[46] depending on study method. Lower rates are found with comprehensive clinical assessment, higher rates with structured tools administered by non-clinicians. In male prisoners the most prevalent disorder category is, unsurprisingly, antisocial followed by paranoid, then borderline, then obsessive compulsive, avoidant and narcissistic at similar rates.[46] In Fazel and Danesh's systematic review[37] 65 per cent of male prisoners were personality disordered (47 per cent anti-social); amongst female prisoners the rate was 42 per cent (25 per cent borderline, 21 per cent anti-social). Psychopathy has been found in 10 to 30 per cent of prisoners.[47]

Blackburn and Coid highlighted the heterogeneity of personality pathology in violent offenders.[48] Using cluster analysis they found six diagnostic patterns: antisocial-narcissistic, antisocial-paranoid, borderline-antisocial-passive aggressive, borderline, compulsive-borderline, and schizoid. The three antisocial groups displayed more psychopathy. Half of homicide offenders in Sweden were personality disordered[49] but the rate in the UK was about 10 per cent[15] (an underestimate due to the method of the study). Rates of personality disorder are high in sexual offenders.[50] Adult rapists have higher rates of psychopathy than child molesters who have higher rates of avoidant and dependent disorders. Sexual homicide offenders have high rates of psychopathy and other personality disorders, particularly in serial offenders and sexual sadists. Sadistic personality disorder (defined in the DSM-IIIR appendix) is found in up to a quarter of violent and sexual offenders, but is present with other personality pathology and is associated with sexual sadism.[51] There is little support for its recognition as a discrete disorder. Most serial arsonists have personality disorders. Borderline, narcissistic and antisocial subtypes of domestically violent men have been

identified.[52] Personality disorder of various types is common in stalkers.[53] Psychopathy is particularly associated with instrumental violence, but reactive violence is also displayed.[44]

Population based cross sectional and birth cohort studies show offending and violence to be increased in individuals with personality disorders, particularly cluster B disorders.[54,55] Follow-up studies show higher rates of offending and violence in personality disordered subjects in general and forensic samples, particularly associated with psychopathy.[56,57,58] High rates of comorbid personality disorder are found in offenders with mental illness[59] and learning disability.[60] A range of personality pathology is found amongst patients detained under 'psychopathic disorder' and 'mental illness' legal categories in English secure hospitals.[59] Most legal 'psychopathic disorder' patients are not psychopaths.[61]

Various traits, individually and combined, are relevant to offending. Some lead to interpersonal conflict (suspiciousness, hostility, argumentativeness, rigidity, arrogance, clinginess), others to behavioural dyscontrol, others to angry emotional reactions, others to not considering consequences for self or others, others to taking pleasure in violating rules and others. Personality pathology may lead to inability to form intimate relationships, maintain work, establish a stable lifestyle or meet basic needs, which may predispose to offending. Impulsivity, need for stimulation, intolerance of dysphoric affect and inability to regulate affect predispose to drug and alcohol misuse leading to offending (see Chapter 11.7).

Assessment

Assessment, as in other cases, involves a comprehensive history, current mental state examination, and information from records and informants. Structured approaches to personality assessment (e.g. PCL-R and International Personality Disorders Examination (IPDE)), may be incorporated but 'psychometrics' should not be used in isolation. Comorbid mental illness should be considered and structured approaches to risk assessment should be used, especially with serious violent or sexual offenders. A comprehensive formulation should consider the relationship between dysfunctional personality traits and offending.

Management

Discussion of treatment is easily hijacked by the political agenda (largely focussed on protecting the public from dangerous psychopaths), the criminal justice context and legal issues. Although it would be naïve to consider treatment in isolation from these other issues, clinical management should be the starting point and primary concern of psychiatrists.

Most personality disordered offenders are not serious offenders, and their treatment needs are similar to individuals with personality disorder generally (see Chapter 4.12.7). General (e.g. long-term support and attention to relationships) and specific (e.g. psychological therapies and medication) approaches are relevant to many offenders. The application of such approaches within the criminal justice system or secure hospitals is complicated by coercion, the legal context and institutional factors. How should the success of treatment be measured? Amelioration of distress, improved social functioning or diminished reoffending? The three may be related, but the public, understandably, expects treatment to prevent offending.

Studies of consenting community patients cannot be extrapolated to serious offenders. The widespread view that psychological treatment programmes increase risk in psychopathic offenders is not backed by the literature, with mixed views and empirical findings.[62] The answer to 'Is psychopathy treatable?' is that the jury is still out and will be for some time. This does not mean treatment is ineffective or inappropriate for the range of personality pathology in serious offenders.

Cognitive behavioural programmes for offending behaviour have a moderate positive impact on recidivism.[63] Many of the areas targeted in such treatments relate to dysfunctional personality traits and many who go through these programmes have personality disorders. Adaptations for individuals with high levels of personality dysfunction and psychopathy have been made incorporating: greater focus on motivation, engagement, maintaining participation; using more appropriate learning styles; addressing underlying core beliefs; greater flexibility; emphasis on individual formulation, and positive psychology.[41] Whether such approaches lead to reduced recidivism awaits evaluation.

Although treatment in hospital may seem appropriate if there is to be a therapeutic approach, there are descriptions of prisons where a therapeutic environment has been achieved.[64] Detention in hospital primarily for indefinite incapacitation of dangerous offenders is controversial.

Personality disordered violent and sexual offenders have treatment needs, but they do not fit into traditional psychiatric systems, geared towards psychotic offenders. The approach with the latter is diversion from criminal justice to health care. With personality disordered offenders, the appropriate approach is probably to provide assessment and treatment within a criminal justice framework, in prison or community. Hospital treatment, as a scarce and expensive resource, should be reserved for those who have the potential to engage and benefit. Meux *et al* comprehensively describe hospital treatment approaches.[65] Staff working with personality disordered offenders, particularly with more severe pathology, need appropriate selection, training, support and supervision. Psychodynamic supervision of staff is important.

Management in the community involves monitoring, supervision and treatment by various agencies. In the UK Multi-Agency Public Protection Arrangements (MAPPA) are used to co-ordinate the management of violent and sexual offenders in the community. Within this mental health professionals can focus primarily on clinical assessment and treatment whilst criminal justice agencies focus on monitoring and supervision.

Outcome

Certain personality disorders are associated with risk of recidivism. Psychopathy is associated with high rates of general and violent recidivism.[41] Amongst sex offenders psychopathy predicts violent rather than sexual offending, although sexual deviation with psychopathy is a malignant combination.[66] Psychopathy is associated with violence towards victims and diverse offending. Successful social integration following discharge has been found to be associated with not being reconvicted.[67] Personality disorder is associated with suicide in offenders.[68]

Legal issues

An insanity defence, although historically relevant in some jurisdictions, is not usually available where the primary diagnosis is personality disorder. The partial defence of diminished responsibility is open to personality disordered homicide perpetrators in

England and Wales. Hospitalization does not often follow such a finding.

Detention in hospital is rather arbitrarily applied to some personality disordered offenders in England and Wales. Indefinite detention in hospital as a sentence can become lifelong incapacitation in a clinical setting.[69] To ensure that hospital detention is treatment focussed there should be prolonged assessment first. Some argue treatment in hospital should only occur via transfer to hospital during a prison sentence. In the Netherlands and Germany, personality disordered offenders are routinely detained in forensic hospitals.

Sexually violent predator laws allow civil commitment (and hospital detention) of mentally disordered high risk sex offenders at the end of a prison sentence in some US states.[70] Most offenders are personality disordered and these laws have survived constitutional challenge.

In many jurisdictions indeterminate sentences are applied to some offenders who are considered to pose an ongoing risk of serious offending.[71] Many such offenders are personality disordered. Psychiatric assessment of risk in such cases is considered unethical by some. Assessments of risk and personality disorder by psychiatrists have been used to under-write the application of the death penalty in some US States.[72]

Mood disorders

Mood disorders are less related to offending than schizophrenia. Disinhibition and grandiosity in individuals with mania leads to public order offences, driving offences, theft, fraud and minor violent or sexual offences. Serious violent or sexual offending is rare. Treatment non-compliance and comorbid substance misuse are associated with offending.

Depression is not uncommon in prisoners (10 per cent in Fazel and Danesh's systematic review[37]) but rarely leads to offending. The association between depression and shoplifting in the historical literature is probably spurious. When depression is encountered in an offender, it may not have been present prior to the offence. If it was it is unlikely to have played a direct role. High life-time rates of depression are reported in sex offenders, and low mood may be one factor of importance in the path to re-offending in some.

Suicide follows 5 per cent of homicides in the UK.[73] Depression sometimes plays a role but other psychopathology is also seen. Types of offences include:

- Spousal: with pathological jealousy.
- Spousal: elderly men with poor health and/or ailing spouses who feel either or both cannot cope with declining health, adversity, or loneliness.
- Filicide-suicide: depressed mother kills her child(ren) and herself to save them from a worse fate.
- Familicide-suicide: depressed, paranoid, or intoxicated male kills the family and himself in the context of financial, marital, or other social stresses.
- Extra-familial murder-suicide: disgruntled, paranoid, narcissistic individuals who feel slighted or humiliated, take revenge on single or multiple victims, either as specific targets or as bystanders. This pattern is seen in spree killings.

Altruistic homicide and extended suicide are terms used where a depressed person kills one or more family members to 'spare them suffering' and commits or attempts suicide. Most are filicide-suicide and familicide-suicide cases. Depressive psychosis with nihilistic delusions is seen in a minority. It is probably impossible to identify these cases in advance, but it is prudent to explore thoughts about children in parents presenting with suicidal ideation.

Publicity has been given to the association between SSRI anti-depressants and aggression and suicide. There is no epidemiological data to back these claims, and some evidence SSRIs reduce impulsivity and associated aggression.[74]

Severe depression or mania may make an accused unfit to plead; affective psychosis may be accepted as the basis of an insanity defence; and milder forms of mood disorder may found a defence of diminished responsibility in homicide cases. Very few patients in secure hospitals are detained on the basis of mood disorder. In England and Wales, mothers with postpartum disorders who kill their children are usually convicted of infanticide rather than murder.

Neurotic disorders

Neurotic symptoms are common in offenders, but it is unusual to find a close relationship between an offence and an ICD-10 neurotic, stress-related, or somatoform disorder. In many offenders it is possible to identify neurotic conflicts and find symbolic meaning in criminal behaviour.[75] Acute stress reactions and adjustment disorders may be associated with offending, particularly where the underlying personality is abnormal or substance misuse occurs. Social phobia may be relevant through its association with alcohol misuse. Although violent and sexual themes often feature in obsessions, obsessive compulsive disorder is negatively correlated with violence and offending. Obsessional features are sometimes seen in the paraphilic behaviour of some sexual offenders.

Post-traumatic stress disorder (PTSD) may be a contributory factor to offending. Offending rarely results directly from flashbacks or other re-experiencing phenomena, although there are cases where rape victims have attacked sexual partners. PTSD may contribute to the violence of women towards abusive partners. 'Battered Woman Syndrome' has been suggested as a sub-category of PTSD in such cases.[76] PTSD rarely leads to a defence of insanity, but may lead to diminished responsibility.

UK psychiatrist read with astonishment and incredulity accounts from the US of multiple personality disorder,[77] where the diagnosis has justified separate legal representation for alters, separate testimony from alters, unfitness to plead, the insanity defence, and malpractice suits when the diagnosis is 'missed'. James and Schramm[78] give guidance to avoid repetition of the US experience in the UK. Psychiatrists are advised not to collude by looking for further alters, investigating those that 'appear', and treating the patient as if he were more than one person. Frankel and Dalenberg[79] give a comprehensive account of dissociation and dissociative identity disorder in the US context.

Factitious illness by proxy

Also called Munchausen syndrome by proxy, this is the fabrication of symptoms in, or the injury of, a child by its carer who presents

the child for medical attention.[80] There is a wide range of injurious behaviours from fabricating symptoms and tampering with specimens and charts to poisoning, smothering, and withholding of nutrients. It is a behaviour rather than a psychiatric disorder. It may feature in systematic and serial child abuse, and in serial killings by health-care workers. Underlying disorders include personality disorders (narcissitic, borderline, and antisocial), somatization, mood, eating and substance misuse disorders. Psychodynamic literature offers various perspectives. In a number of highly publicized cases in the UK, expert evidence regarding families with multiple unexplained child deaths has led to mothers being convicted of murder. Several cases have been overturned on appeal with subsequent disciplinary action against the experts involved.

Impulse control disorders

Impulse control disorders are described in Chapter 4.13.1. Two specific 'disorders' have relevance in forensic psychiatry: kleptomania and pyromania. Psychiatric aspects of arson are described in Chapter 11.9

In clinical practice, it is rare to find a shoplifter who meets criteria for kleptomania without underlying psychiatric disorder (mood, anxiety or eating disorder).[81] Apparently inexplicable acts of shoplifting may be perpetrated by people who have no features of kleptomania. Goldman[82] in an extensive review concluded that the disorder is more common than previously suggested.

The diagnosis of pyromania is of equally dubious validity. Most repeated arson offenders with the clinical features are excluded as they have other underlying disorders (psychosis, intellectual disability, or personality disorder) or are intoxicated when they set fires.[83]

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11.3.2 Offending, substance misuse, and mental disorder

Andrew Johns

This chapter deals with the relationship between offending, substance misuse, and mental disorder, and also describes approaches to clinical and medico-legal assessment.

Relationship between offending, substance misuse, and mental disorder

The nature of this relationship is complex, yet has to be understood in order to manage the health and risk of offending of individual patients. Offences related to substance misuse can be categorized as (i) violent offences, often involving an altered mental state, (ii) acquisitive offences, and (iii) miscellaneous offences such as breaking laws to control the misuse of drugs, driving under the influence of alcohol, and impact of substance misuse on parenting.

Violent offences

Aggression is not an inevitable pharmacological consequence of misusing alcohol or any particular drug, but arises from many possible factors including expectancy effects, pattern of consumption, individual responses to intoxication or withdrawal, peer influences, and interpersonal issues.

Alcohol consumption is the single factor most associated with violence. It is a repeated finding from the British Crime Surveys that alcohol is a key factor in at least half of interpersonal assaults, with a greater contribution to assaults on strangers and domestic violence. By comparison, drug misusers are overwhelmingly more likely to commit acquisitive offences.

The co-morbidity of major mental illness and substance misuse further increases the risk of violence. For example, in the Epidemiological Catchment Area (ECA) survey of 10 000 individuals,[1] the prevalence of violent behaviour in the previous year was 2 per cent for those with no mental disorder, 7 per cent in 'major mental illness', 20 per cent for 'substance misuse disorder', and 22 per cent for co-morbid respondents. Among patients with first-episode psychoses,[2] just under 10 per cent demonstrated serious aggression when psychotic and 23 per cent showed lesser degrees of aggression. Those co-morbid for drug misuse were nine times more likely to show aggression after service contact—primary drug-related psychoses or alcohol misuse were not so associated.

There is particular concern in the UK about the risk to the public from serious violence by the mentally disordered. Nationally,[3] alcohol or drug misuse contributes to two-fifths of homicides and 17 per cent were committed by patients with severe mental illness and substance misuse. Alcohol- and drug-related homicides were generally associated with male perpetrators who had a history of violence, personality disorders, mental health service contact, and with stranger victims.

However, these epidemiological studies cannot define a causal link between substance misuse and mental disorder. There are many ways in which violence may arise in substance misusers and in co-morbid individuals. Simple intoxication on alcohol or other depressants such as benzodiazepines or barbiturates, leads initially to apparently excited behaviour. Stimulants such as cocaine or amphetamines, may produce arousal and irritability. Most forms of intoxication are also associated with impaired judgement, perception, and impulse control. Severe intoxication on alcohol, Cannabis, sedatives, or stimulants can lead to a toxic psychosis and highly disturbed behaviour. Even at levels of consumption insufficient to intoxicate, disinhibition, and autonomic arousal may facilitate recklessness and aggression. Pathological intoxication, in which aggression is supposed to occur within minutes of consuming moderate amounts of alcohol, is of doubtful validity and in most cases, better explained by alcohol-induced hypoglycaemia, head injury, or other organic disorder.

The association between withdrawal effects and potential for violence is often overlooked. Withdrawal from alcohol and most drugs of dependence, is a highly aversive state in which irritability and aggression may occur. Cessation from alcohol or sedatives, may lead to more severe withdrawal syndromes such as delirium tremens which are commonly associated with impaired perception, affect, judgement, and impulse control.

Acquisitive offending

The relationship between acquisitive crime and drug misuse problems was studied among 753 clients recruited to the National Treatment Outcome Research Study (NTORS).[4] More than 17 000 offences were reported during the 90-day period prior to treatment. Half of the clients committed no acquisitive crimes during this period, whereas 10 per cent committed 76 per cent of the crimes.

Such work does not demonstrate a causal relationship between illicit drug use and acquisitive crime. From a large survey of British youth,[5] the average age of onset for truancy and crime are 13.8 and 14.5 years respectively, compared with 16.2 for drugs generally and 19.9 years for 'hard' drugs. Thus, crime tends to precede drug use rather than vice versa. It is clear that heavy drug use is strongly associated with impulsive acquisitive offending, including street robbery, and burglary, which involve violence.

Other offences

In Britain, the non-medical use of drugs is subject to the Misuse of Drugs Act 1971, as subsequently amended, and which contains a classification based on perceived harm. Class A drugs include Ecstasy, LSD, heroin, cocaine, crack, magic mushrooms (if prepared for use), amphetamines (if prepared for injection); Class B drugs include amphetamines and methylphenidate (ritalin); Class C drugs include Cannabis, tranquilizers, some painkillers, Gamma hydroxybutyrate (GHB), ketamine. In January 2004, Cannabis was reclassified from a Class B to a Class C drug, it is still illegal. This legislation defines the penalties for supply, dealing, production, trafficking, and also possession.

Other offences include driving cars, or public conveyances such as trains whilst under the influence of alcohol or other drugs.

Responding to the drug or alcohol using offender

Clinical- and risk-assessment

The following is a practical guide to assessing the drug or alcohol-using offender.

1 Obtain a detailed life history, with corroboration from other informants and agencies. This should include relationships, work record, and current social situation.

2 Take a detailed history of all of the substances of misuse, including onset of regular use, dosage, route of administration, and pattern of use in a typical week. Ask about the desired effects of substance misuse, and also the actual effects. If there is also a serious mental illness, ascertain the effect of substance misuse on symptoms and behaviour. Has previous or recent substance misuse been associated with self-harm or aggression? Note any history of substance misuse treatment and the effects of this.

3 Take a detailed history of previous and recent offending with reference to the effects of substance misuse and mental illness, on mental state and behaviour before, during, and after each offence. Corroborate where possible from witness statements and independent sources.

4 Take a detailed mental state including some assessment of intelligence and personality, and also degree of insight into their offending, illness, and substance misuse.

5 Consider with the patient, the practical implications of this assessment for immediate clinical management and any need for medico-legal reporting that may arise.

There is increasing recognition of the role of actuarial instruments such as the HCR-20 (Historical/Clinical/Risk-management 20-item scale)[6] which allow for previous and current substance misuse to evaluated in the context of other significant risk factors.

Informed by the above, carry out a risk-assessment, firstly by defining the nature of any risk such as self-harm or relapse in substance misuse or of a primary mental illness. If a risk of violence is identified, this may involve particular individuals such as family, partners, or carers. Assess the probability and severity of each risk, and whether there are any early warning signs, such as particular behaviours or symptoms, or non-compliance with treatment.

Risk- and clinical management

Maden[7] (2007) argues 'the first step in improving risk-management is to recognize that that the prevention of violence is a central task of mental health services'. As a general approach to risk-management of the drug or alcohol misusing offender, ascertain what risk factors may be changed, and how the provision of support, care, or security may reduce the risk.

It is clearly important to achieve cessation or control of drug or alcohol misuse. These are not easy aims, but it is important to dispel therapeutic nihilism and to appreciate that a range of interventions have been shown to be effective. Details of specific interventions are given in Chapter 4.2.2.4 and Chapters 4.2.3.1–4.2.3.7.

Treatment can reduce re-offending. The NTORS[4] found that 5 years after treatment, convictions for acquisitive, drug selling, and violent crimes had reduced.

The National Confidential Inquiry[8] has concluded that provision for dual diagnosis should be central to modern mental health care and should include: staff training in substance misuse management, joint working with drug and alcohol teams, local clinical leadership and use of enhanced Care Programme Approach for all those with severe mental illness, and a destabilizing substance misuse problem.

Medico-legal issues

Possible defences related to substance misuse

Generally speaking, the acute effects of having voluntarily taken drugs or alcohol are not a mitigating factor and it is argued that a drunken intent is still an intent. There are however narrowly defined circumstances in which an altered mental state due to substance misuse can raise the question of a possible defence.[9]

Amnesia

Amnesia is common after violent offending, may relate to acute intoxication especially on alcohol or sedatives. In the absence of organic disease, amnesia does not affect fitness to plead, though it clearly complicates assessment of the perpetrators mental state at the time of an offence.

Simple intoxication

Self-induced intoxication is generally no defence to a criminal charge. However, in England and Wales, case-law has determined

that crimes such as murder, wounding with intent, theft, and burglary, require a *specific intent*, for which self-induced intoxication on alcohol or drugs may be a defence, but only if it can be shown that the accused was so intoxicated as to be unable to form the necessary intent. The psychiatrist can only comment as to whether the accused had the capacity to form the specific intent. It is a matter for the jury to determine whether the specific intent was present or not. If the specific intent is not demonstrated, then the accused may still be convicted of a lesser offence, so that acquittal on a charge of murder may lead to a conviction for manslaughter. It is a matter of clinical judgement as to whether an individual was so intoxicated as to be unable to form a specific intent, and the degree of purposiveness before, during, and after the offence, may be a useful indication.

Other crimes such as manslaughter, rape, and unlawful wounding, require only a *basic intent*, which cannot be negatived by intoxication. For these offences, the recklessness of voluntary intoxication may provide the necessary mental guilt.

Insanity

Alcohol or drug misuse may give rise to a psychotic illness, such as delirium tremens, which may meet the requirements of the McNaughton rules, but the inanity defence is rarely used. In theory, consumption of drugs or alcohol could lead to a state of insane automatism, but the defence of insanity is not available if the consumption has been voluntary.

Diminished responsibility

In England and Wales, Section 2 of the Homicide Act 1957 provides a defence of diminished responsibility in a charge of murder. The defence has to demonstrate that an *abnormality of mind* arises from one of the causes specified in the Act and those of possible relevance to substance misuse are *disease*, *injury* or *inherent causes*. An abnormality of mind due to intoxication is no defence. Alcohol dependence could meet criteria for *disease*, provided that the first drink of the day was shown to be involuntary. Diminished responsibility may become an issue when the effects of substance misuse interact with other factors such as organic brain damage, depression, or personality disorder. For legal purposes, the effect of intoxication has to be set aside and the defence must show that the associated condition was in itself severe enough to lead to an abnormality of mind.

Psychiatric recommendations to the courts

In reporting to the court, the task of the psychiatrist is to explain the possible contribution of substance misuse to a particular offence, in the context of the life history, psychiatric, and offending history of the individual. The aim of such a report is (i) to consider relevant psychiatric issues and their bearing on the offence, (ii) to indicate whether treatment could usefully prevent re-offending, and (iii) to help the court to protect society. There are a range of legal interventions that can facilitate engagement in treatment in community settings.

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11.3.3 Cognitive disorders, epilepsy, ADHD, and offending

Norbert Nedopil

'Cognitive disorders' is a broad and heterogeneous diagnostic category, which includes different disorders, each with a distinct aetiology. They affect individuals in different ways depending on the age in which they occur. The term may be applied to a child, who has experienced perinatal trauma as well as to an older person with a beginning dementia of the Alzheimer type. The scientific literature on offenders with cognitive disorders is sparse. Most authors in forensic psychiatry do not systematically differentiate between the diagnostic subcategories and tend to use broad terms, such as organic disorder, organic psychosis, organic brain syndrome, neuropsychological deficit, dementia, mental handicap, mental retardation to include a number of different disorders in their studies. The number of patients with any kind of brain disorder in forensic hospitals and institutions is comparatively small and ranges from 1 to 10 per cent of all forensic inpatients. The same numbers apply for individuals assessed for criminal responsibility or risk of reoffending.[1–3] Compared to major mental disorders like schizophrenia or affective disorders or to personality disorders, patients with cognitive disorders account for only a small proportion of individuals seen by forensic psychiatrists. Subdividing this group any further would be statistically irrelevant. The way forensic psychiatry and the law deals with offenders suffering from organic brain disorders is rather derived from case reports and convention than from empirical knowledge.

DSM-IV-TR cites several disorders where aggression is either a diagnostic or associated feature and among them are four with an organic aetiology.

- Dementia of the Alzheimer type (DAT)
- Dementia caused by head trauma

- Personality change due to general medical condition (aggressive type)
- Postconcussional disorder

The psychiatric and general medical literature lists several other organic brain disorders that are either believed to be or in fact are associated with violence and offending, although their link is not as well proven.[4]

- Epilepsy
- Huntington's chorea
- Korsakow psychosis
- Brain tumours
- Mental retardation

From the experience of the author two other disorders should be added to this list:

- Traumatic brain injury
- Frontotemporal dementia

Systematic analyses of epidemiological data and of other research findings show that patients with clinically relevant brain damage do not commit violent crimes more often than would be expected according to their proportion in the general population.[5–7] These findings do not contradict the knowledge we have about aggressive and disruptive behaviour of certain patients with brain damage. The estimates of the frequency of such behaviours range from 18 per cent in demented patients to 60 per cent in patients with frontal lobe injuries.[8] Most of these patients are not seen by forensic psychiatrists, but are treated in special institutions or in outpatient settings. Apparently, the violent behaviour of patients with brain damage does not lead to interventions by the criminal justice system as often as could be expected from the above mentioned numbers. Similar findings are reported from demented patients: although not appearing in criminal court files, aggression and agitation of demented patients is a major problem in nursing homes and for caregivers of the elderly in outpatient settings. Again, exact definitions and robust data on how much violence really occurs are lacking, but estimates range from 18 to 48 per cent.[8] Rabins *et al.*[9] reported that 75 per cent of caregivers considered aggression as the most serious problem in agitated demented patients.

Offending and contact with the criminal justice system can be expected to be more frequent in patients who suffer less from cognitive impairments—which would prevent skilled or planned criminal activity—but rather from personality changes, like irritability, impulsivity, lack of concern for others, and for the consequences of one's action which is the case in frontotemporal dementia. Offending, but rarely violent offending, occurs sometimes as a first sign of this disorder.[10] Violent crimes are sometimes associated with cognitive disorders when delusions are among the first symptoms of a beginning dementia. Especially delusions of jealousy, envy, or revenge are prone to result in violent acts, which may leave partners or neighbours as victims. These crimes contradict the previous occupational and social life of the perpetrators and are paradigmatic examples of offending as a result of a mental disorder, leading to inculpability of the patient.

Offending can also be expected to be more frequent in patients between 18 and 35 years old and therefore in an age, where offending is statistically more frequent than in other age groups. Males of the same age group have the highest rate of traumatic brain injury. They also belong to the age group with the highest rate of criminality and especially of violent criminality. This same age and sex group also has the highest rate of substance abuse. Given the high prevalence of brain injury among young men and their propensity to use alcohol and drugs it is surprising how few are seen by forensic psychiatrists or sentenced to prison. The actual numbers of such patients found in forensic hospitals and in prisons do not reflect the high risk of violent crime by persons with brain injury. Hodgins[11] found that only 0.4 per cent of male penitentiary inmates warranted a diagnosis of organic brain syndrome (which is a much broader term than traumatic brain injury). Similarly the proportions of patients with organic brain syndrome in forensic hospitals is below 10 per cent and not greater than that in general psychiatric hospitals.[2,5]

Several studies suggest that the criminality of individuals with brain injury may, to a large extent, be attributed to premorbid personality traits, to the social disintegration which follows the injury, and hence not only to the injury itself. Kreutzer *et al.*[12] studied a sample of 327 patients with varying severities of traumatic brain injury. Those arrested after the brain injury were more likely to have had a history of police contacts before the brain injury, than those who were not arrested.

Two disorders have to be presented in greater detail:

Epilepsy, because it was historically one of the disorders of great concern for forensic psychiatrists and served as a model of the mentally ill offender not responsible for his crimes, and ADHD, because it is one of the disorders for which a relationship to antisocial behaviour and offending is most intensively researched.

Epilepsy and offending

Throughout history epilepsy has been associated with violence. Devinsky and Bear[13] observed 'it would be difficult to cite, either from case reports or a literature review, another medical or neurologic illness in which aggressive behaviour is described so regularly'. Not only seizures were frightening for lay people and caused them to consider epileptics as being cursed by gods or being possessed by witches (Malleus Maleficarum, 1487) and dangerous to others, these patients were seen as threat because of their personality changes. At the turn of the twentieth century most lay persons and professionals believed that people with epilepsy had pathological personality traits and displayed aggression, sociopathy, and psychosis.[14] Kraepelin too reported aggression in epileptic patients and mentioned that almost always an intensification of mental irritability occurs. Jackson took it as given that epilepsy was a cause of insanity '…often of a kind that brings epileptics in conflict with the law'.[15] Even in 1973 Sjöbring[16] noted, that patients suffering from epileptic seizures become torpid and circumstantial, sticky and adhesive, effectively tense, and 'suffer from explosive outburst of rage, anxiety and so on'.

Epidemiologic research,[17] literature reviews,[18] and experimental studies[19] have not supported these beliefs. Although epilepsy was found to be three to four times more frequent among prisoners in the United Kingdom than in the general population,[17] their offences did not differ from those of the rest of the prison population. Similar findings were reported from the United States (King and Young, 1978). In a extensive survey of mentally ill

offenders in Germany, who had committed acts of violence, Häfner and Böker[5] found only 29 patients with epilepsy out of 533 hospitalized violent offenders (5.4 per cent of the total sample). They compared their sample to an unselected population of 3392 non-violent mentally ill hospital patients and reported that 5.2 per cent of them had also received the diagnosis of epilepsy. They concluded that epilepsy was statistically not a risk factor for violence. A thorough analysis of the crimes of the epileptic patients showed that marital status (single), educational level, socio-economic state, and alcohol consumption were more important risk factors than epilepsy. This is in accordance with studies in other countries. Eight of the 29 patients in the Häfner and Böker study had committed their crimes in an epileptic confusional state (which corresponds to the medico-legal term of organic automatism), but 11 had a quarrel with their victim before their offence.

Although specific personality changes were not confirmed for epileptics in general, and some authors attributed them rather to institutionalization, brain damage, comorbid disorders, medication, or social changes than to epilepsy itself, a specific association has been made between temporal lobe epilepsy (TLE) and special personality traits. 18 characteristic personality traits were summarized from the literature to constitute the Gastaut–Geschwind syndrome,[20] among them aggression, emotional lability, and 'hypomoralism', traits that must be considered as risk factors for offending. The empirical and neurobiological database to support an increased risk of criminality or violence even in these patients is, however, small. MRI studies found that severely aggressive epileptic patients were characterized by severe amygdalar atrophy or by left temporal lesions affecting the amygdala and the brain regions around them,[21] confirming the assumption that violence has to be attributed to specific brain damage rather than to epilepsy itself.

Epilepsy is associated with a number of psychopathological and behavioural symptoms, among them mood changes, anxiety, rigidity, and aggression. Epidemiologic studies show a high comorbidity with cognitive impairment, ADHD, personality disorders and psychotic disorders. Behavioural abnormalities and comorbid disorders, as well as epilepsy itself can contribute to the risk of offending in these patients.

Summarizing the results from newer empirical studies, epilepsy does not increase the risk for offending or violence, and the number of cases, in which epilepsy was successfully used to claim inculpability after offending violently is small.[22] Forensic psychiatrists confronted with the assessment of epileptic patients who have committed crimes have to consider the following key questions:[23]

- Is the association between offending and epilepsy due to the occurrence of the epileptic seizure itself?
- Is it due to the associated brain damage that may be the cause of the seizure?
- Is it the result of socio-economic factors or of medication?
- Is the offending independent of the epilepsy and due to other criminogenic factors?

There are theoretically several possibilities, why epileptic patients could offend or act violently as a consequence of the disorder:

1 Aggression or impulsive acts could be a manifestation of a seizure or an equivalent to a seizure (violent automatism); it could also be the reaction of the patient to negative aura experiences. Delgado-Escueta *et al.*[24] collected 5400 videotaped seizures and concluded that violence appears to be extremely rare event in epileptic seizures. Only 13 individuals in their sample acted violently and only three attacked other people. This study was criticized because it did not take the patient environment interaction into account, which also plays a major role in outbreaks of aggression and which can be modified by epileptic seizures. Nevertheless, the number of cases of ictal aggression reported in the literature is small. Ictal violence erupts out of a normal non-aggressive situation within seconds, appears to be inappropriate to the circumstances, lasts for 1 to 3 min and subsides as suddenly as it has erupted. The patient returns to consciousness immediately or returns to normality after a few minutes of confusion, appears to be puzzled over what has happened and has no memory of it. Complex criminal acts cannot be attributed to seizures, seizure equivalents, or epileptic automatism.

2 Violence can be the result of tension and irritability in the prodromal phase of an epileptic seizure. In this case the irritability usually proceeds in waves, often triggered by the reactions of bystanders. Violence appears to be goal-directed and limited to avoid personal harm. The aggressive tension may last for several minutes up to an hour. Sometimes the patients seem out of control and do not remember their behaviour at al.

3 Postictal confusion can be associated with poriomania, somnambulism, and offending. Again, complex acts and adequate reactions to new situations cannot be attributed to postictal confusion or twilight states.

4 Interictal offending is quite often not associated with the disorder itself but with a criminal or aggressive family background, lower socio-economic status, and brain trauma.[23,25] Violence can be due to epileptic psychosis, to cognitive impairment, to impulse control disorder or to personality disorder, all of which could be consequences of epilepsy, although the data on these sequels of epilepsy are not undisputed.

A recent review of the literature by Schachter[26] can be summarized to the fact that postictal aggression was rarely due to confusion but significantly more frequent in postictal psychosis, and interictal aggression was associated rather with male sex, brain damage, social disadvantages, and chronic behavioural difficulties than with specific EEG findings or characteristic brain scans. In conclusion several risk factors have to be taken into account when the offending or violence of epileptic patients is considered:

Disorder-related risk factors

- Brain damage
- Early onset of epileptic seizures
- Postictal psychosis

Disorder-unrelated risk factors

- Male sex
- Growing up in a criminal or aggressive environment
- Low socio-economic class
- Antisocial personality disorder
- Alcohol abuse

In most legislations forensic psychiatrists and courts would agree, that the criteria proposed by Hindler,[27] would be minimal requirements to relate offending or violence to epilepsy:

- An unequivocal past history of epileptic attacks
- The crime is out of character with the person's previous personality
- The crime is motiveless and unpremeditated
- EEG studies are compatible
- An altered state of consciousness during the event
- Total or partial amnesia for the crime

More stringent criteria were proposed by a panel of epileptologists in the United States in 1981.[24] They included videotaped documentations of epileptic automatism and of the presence of aggression during such seizures; also the aggressive or violent acts should be characteristic of the patient's habitual seizures. In most Central European countries videotaped proof would not be necessary to attribute the offending of an epileptic to his disorder.

Attention-deficit/hyperactivity disorder (ADHD) and offending

Attention-deficit/hyperactivity disorder (ADHD) is a relatively new diagnostic term. It has been introduced in the current form in DSM-III-R in 1987, although the condition has been known to psychiatrists for more than 100 years.[28] Only with DSM-IV-TR some allowance was made to extend the diagnosis to adults[29] but DSM-IV-TR continued to assert that the majority of the patients loose their symptoms during late adolescence. The diagnostic criteria were empirically evaluated in school-children and it can be questioned whether they are appropriate for adolescents and adults. It can be questioned even more, how the association between offending and ADHD can be adequately established. The core symptoms of ADHD, inattention, hyperactivity, and impulsivity, are likely to cause social conflicts with peers and caregivers and to lead to resentment and aggressive interactions. This dissocial functioning may appear as the precursor of offending in later life. A number of studies tried to investigate the relationship between ADHD in childhood and the diagnosis of antisocial personality disorder (APD) in adulthood. Although there is converging evidence that about 25 per cent of children with ADHD will later receive the diagnosis of APD,[30,31] and that in individuals who met the criteria of psychopathy according to Hare,[32] ADHD was diagnosed retrospectively four times more often than in the general population.[33] Most of the studies ignore the possible impact of comorbid disorders, which are especially frequent with ADHD. Conduct disorder (CD), oppositional defiant disorder (ODD), alcohol dependence or abuse and other drug dependence or abuse are significantly more frequent in ADHD individuals than in control groups,[34] and all contribute substantially to offending and to the diagnosis of APD. The longitudinal study of Satterfield & Schell[35] found that half of the children with a combination of ADHD and CD were later diagnosed as APD, while APD was only found in 12 per cent of those who had ADHD without CD in their childhood.[36]

A number of studies found ADHD largely overrepresented in prison populations, where it is calculated to be up to 10 times more frequent than in the general population.[37,38] Except for drug-related crimes,[30] no special criminal profile could be attributed to the disorder.

In psychiatric assessments for courts and tribunals ADHD is rarely considered as a cause for diminished responsibility, and most offenders with this disorder will not be sent to forensic psychiatric institutions. ADHD has, however, to be regarded as risk factor for reoffending, especially if it is or was combined with a conduct disorder. Prisoners with ADHD had been reconvicted four times more often than other prisoners.[39]

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11.4

Mental disorders among offenders in correctional settings

James R. P. Ogloff

Incontrovertible evidence now exists to show that the prevalence of mental disorders among prisoners far exceeds that found in the general community. A surprising concordance is emerging from several large international studies to show that, in western developed societies at least, the rates of major mental disorders in prisons are quite consistent. This chapter will provide an overview of relevant research examining rates of mental illness in prisons with those found in the community. Some observations regarding trends and implications for prisons also will be provided.

At the outset it is useful to reflect on the scope of illnesses which have been subsumed under the 'mental disorder' umbrella as it has been applied to the prison research. Most of the research that exists has focussed serious mental illnesses within the Axis I disorders—namely psychotic illnesses, mood disorders, and anxiety disorders. Considerable attention has been paid regarding the prevalence of personality disorders within prisons. Over the past 20 years much of that work has investigated antisocial or dissocial personality disorder and psychopathy.[1] By comparison, relatively little attention has been paid to other personality disorders. A growing area of importance concerns substance abuse and dependence disorders and, of course, co-occurring substance use and mental illness disorders. Considerable research also exists exploring the prevalence of mental retardation or intellectual disabilities in prison. Thereafter, fragments of research exist exploring any number of mental syndromes and conditions. The focus of this chapter will be on the major mental disorders which fall into Axis I. Some mention will be made of substance use disorders and personality disorders. In addition, with the growing number of women in prisons, information will be provided regarding this important group.

The prevalence of mental illness among male and female prisoners

Recent research exists from Britain and Wales that shows that the prevalence of mental illness in prisons is many times greater than that found in the community. Brugha and colleagues[2] compared rates of mental illness among some 3,000 remanded and sentenced male and female prisoners in Britain and Wales and more than 10,000 community residents in Great Britain. The rate of psychotic illnesses in the community was 4.5 per 1,000 (0.045 per cent) compared with 52 per 1,000 in prisons (0.52 per cent). While the ten-fold increase in prevalence from the community to the prisons was remarkable, the results revealed further that the prevalence rate of psychotic illness for female prisoners was an astonishing 110 per 1,000 (0.11 per cent), compared to 50 per 1,000 for males (0.05 per cent).

One of the key studies that has helped provide information regarding the rate of mental illness in gaols and prisons is a meta-analysis conducted by Fazel and Danesh[3] that was published in *The Lancet*. Their analyses included 62 studies that included 22,790 prisoners. The majority of prisoners (81 per cent) were male. Nonetheless, enough studies that included women prisoners were available to provide information regarding the rate of mental disorder among them. Data from the Fazel and Danesh meta-analysis are presented in Table 11.4.1. The results show that approximately one in seven prisoners have a psychotic illness or major depression. As the authors report, this is between two and four times greater than would be expected in the general population.

Table 11.4.1 Representative prevalence of mental illness and personality disorder among male and female prisoners (international samples)

Disorder	Males per cent (95 per cent C.I.)	Females per cent (95 per cent C.I.)
Psychotic Illness (k = 49, N = 19,011)	3.7 (3.3–4.1)	4 (3.2–5.1)
Major Depression (k = 31, N = 10,529)	10 (9–11)	12 (11–14)
Personality Disorder (k = 28, N = 13,844)	65 (61–68)	42 (38–45)
Antisocial Personality Disorder	47 (46–48)	21 (19–23)

k = number of studies; N = number of subjects; C.I. = Confidence Intervals.

Source: Fazel & Danesh (2002).

With respect to personality disorders, half of males and approximately 20 per cent of females are found to have a personality disorder—which is ten times greater than would be seen in the community.

There was some variability across studies, some (but not all) of which was explained by differences between research that used validated diagnostic procedures (3.5 per cent) and those that did not (4.3 per cent). Studies from the USA also showed higher prevalence rates than elsewhere. Psychosis among female prisoners was found to be slightly higher than that in males (4.0 per cent *c.f.* 3.7 per cent).[3]

A limitation of the Fazel and Danesh[3] meta-analysis is that relatively limited information was provided regarding the type and nature of mental illness. Brinded, Simpson, Laidlaw, Fairley, and Malcolm[4] reported the results of one of the most well conducted studies on the prevalence of mental illnesses among inmates ever published. *All* female sentenced and remanded inmates and a random sample of 18 per cent of sentenced male inmates in New Zealand were interviewed. Interviewers used standardized measures to identify inmates with mental illnesses and personality disorders. The final sample consisted of approximately 1 200 inmates. The results of prevalence rates for mental disorder in the last month are presented in Table 11.4.2.

As the results in Table 11.4.2 show, the prevalence rates obtained by Brinded and colleagues[4] in New Zealand essentially parallel those obtained by the Fazel and Danesh[3] meta-analysis. The New Zealand results, however, include data for post-traumatic stress disorder, substance abuse, and dependence disorders. As with psychosis and major depression, the prevalence rates of the other disorders is significantly greater than what would be seen in the general population.

Of late, increased attention is being paid to the prevalence of mental disorders among female inmates.[5] In recent years, the rate of growth among women in custody has far surpassed the growth rate for male prisoners. For example, data show that in the 10 years ending 2005, the percentage of women in prisons in the United States has increased by 57 per cent, compared to a growth rate of 34 per cent for men during the same period.[6] Similar findings exist in Australia, where research shows that the number of women in prison increased by 66 per cent from 1991 to 1999, while it increased by 24 per cent for men during the same period.[7]

Even more than for male prisoners, 'the prevalence of childhood and adulthood sexual and violent victimization, poverty, and poor educational and employment attainment reported by female inmates is nothing short of alarming'.[8] While a comprehensive review of the studies of mental illness among women offenders is beyond the scope of this chapter, Ogloff and Tye[5] have shown that the prevalence rates of mental disorders for women is now surpassing those identified for men in most published studies. This is particularly the case for mood disorders and anxiety disorders.

Table 11.4.2 Prevalence rates for mental disorder in last month (New Zealand samples)

Diagnosis	Women N = 167 n (per cent)	Remanded Men N = 441 n (per cent)	Sentenced Men N = 636 n (per cent)
Mental Illness			
Schizophrenia and related disorders	7 (4.2)	15 (3.4)	14 (2.2)
Bipolar affetive disorder	2 (1.2)	4 (1.0)	7 (1.1)
Major depression	18 (11.1)	47 (10.7)	38 (5.9)
Obsessive-compulsive disorder	7 (4.3)	22 (5.0)	21 (4.8)
Posttraumatic stress disorder	27 (16.6)	42 (9.5)	55 (8.5)
Substance-Related Disorders			
Alcohol abuse	7 (4.3)	25 (5.7)	8 (1.2)
Alcohol dependence	4 (2.5)	19 (4.3)	3 (0.5)
Cannabis abuse	6 (3.7)	38 (8.6)	27 (4.2)
Cannabis dependence	0 (0)	0 (0)	0 (0)
Other abuse/dependence .	6 (3.7)	27 (6.1)	12 (1.9)

Reproduced from Brinded *et al.* Prevalaence of pyschiatric disorders in New Zealand Prisons: A national study. *Australian and New Zealand Journal of Psychiatry*, **35**, 166–73, copyright 2001, John Wiley & Sons, Inc.

Implications, service needs, opportunities

The cause of the relatively high prevalence of mental illness among people in the prison system has been sometimes attributed to the deinstitutionalization movement that has occurred in mental health over the past 20 years. The contention that the mentally ill are entering gaols in increasing numbers has not been accepted by all, however.[9] It has been proposed that it is simply heightened awareness among professionals and the public of the problem of mentally ill in the gaols that has resulted in the perception that they are entering in increasing numbers.[10] In a recent study investigating the criminal offence history of every person in Victoria with schizophrenia in the public mental health registry in five year cohorts from 1975 to 1995, Wallace, Mullen, and Burgess[11] found that there was no subsequent increase in offence rate by year for those with schizophrenia, while the offence rate for the matched comparison group of people in the community without a mental illness increased significantly over the period. This is particularly interesting since during that time the process of deinstitutionalization was completed in Victoria. Indeed, there are no more psychiatric hospitals in Victoria (except for a 100 bed secure forensic psychiatric hospital).

A number of contributing factors have been identified that help explain the high numbers of people with mental illnesses in the criminal justice system. Considerable concern has been raised about the capacity of community-based mental health services to address the needs of mentally ill offenders. Community-based mental health services work best for those who have reasonable connections and support within the community. Unfortunately, offenders (especially imprisoned offenders) tend to be poorly integrated into the community[12] and have poor access to a range of support services including accommodation, income support, health and mental health.[13, 14]

While the presence of mentally ill people in the criminal justice system presents challenges and raises concerns, the fact is that the justice system provides an opportunity to identify and deliver treatment to people who are otherwise likely to remain outside the reach of services. As such, it has been suggested that justice mental health services present an opportunity for identifying those with mental illnesses and making services available to them that would otherwise be non-existent.[10] Accordingly, taking a population

health perspective, efforts to identify and treat those with mental illnesses who are entering the criminal justice system can help provide much needed services to this otherwise under-serviced population.

Service development and provision to mentally ill offenders in prisons

Although it is beyond the scope of the chapter to discuss the provision of mental health services to prisoners, it may be helpful here to outline some of the requirements for services. The service model outlined here has been detailed elsewhere in the literature.[10] The service model recommended for use in prisons consists of six components outlined below, the nature and extent of which will vary depending on the needs arising in each institution.

Intake screening

A two-tier evaluation process is recommended. The first step involves a brief mental health screening for every inmate upon admission. Second, those prisoners identified as being mentally ill are referred to mental health professionals for a more complete assessment. All prisoners should be screened for mental illness soon after admission to a correctional facility (within the first 24 h). The *Jail Screening Assessment Tool (JSAT;* Nicholls *et al*., 2005) was developed to screen people being admitted to gaols for mental illness, as well as self-harm risk and risk of harm to others. The JSAT is administered by psychiatric nurses or other mental health professionals and takes approximately 20 minutes. It is validated for both male and female prisoners.

Ongoing monitoring/screening of prisoners

A process must be in place for ensuring that prisoners are monitored, both formally and informally. This should include self-referrals and referrals by all prison staff.

Comprehensive psychodiagnostic assessment

Comprehensive assessments by psychiatrists or clinical psychologists are required for all prisoners exhibiting symptoms of mental illness. The examination should include consideration of whether the prisoners who are acutely mentally ill should receive treatment in the institution or be transferred to hospital.

Mental health treatment

Once assessed as having a mental illness, the prisoner should be referred to an appropriate treatment program within the correctional facility or correctional system if possible and practical. The size of the gaol and its mandate affects the type of service available. Services should be at the standard available in the community.

Gradual post-release monitoring/supervision and continuity of services

Treatment should continue post-release in the community. The transition back to the community is often difficult, as evidenced by high recidivism rates and ongoing illness. Service needs for mentally ill people leaving prison include initiating psychiatric treatment and psychosocial services with a community mental health agency, locating housing, and finding employment.

Programme evaluation

Given the complexity of mental health services in prisons, it is critical that programmes are evaluated on an ongoing basis (e.g. Elliot, 1997). Such evaluations are as important as the other components in achieving assessment and delivery of mental health services. Also, wherever possible, assessment must be linked with treatment. Ongoing evaluations of the effectiveness of the assessment/treatment decisions should be built into the system. Evaluation informs decision-makers about the outcome of their decisions. Over time, this feedback can lead to improvements in the assessment, referral, and treatment phases of the model. Data on the base rates of mental disorder among women in custody-and the number of female prisoners who fall into the MDO categories—also can prove valuable in planning for future treatment needs.

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11.5

Homicide offenders including mass murder and infanticide

Nicola Swinson and Jennifer Shaw

There is a widespread public perception of the mentally ill as violent.[1,2] Until the early 1980s there was a consensus view that patients with severe mental illness were no more likely to be violent than the general population. Emerging evidence from various countries over the past two decades, however, has established a small, yet significant, association between mental illness and violence.

Large-scale birth cohort studies, such as a 30 year follow-up of an unselected Swedish birth cohort, show a significantly increased risk of violent offences in men and women in the presence of major mental disorder.[3] Community epidemiological studies in New York[4] and in Israel[5] again show an increased risk of violence in psychiatric patients. An important contribution to this field is data from the Epidemiological Catchment Area study, showing that major mental illness increases the rates of violence over a 12-month period from a 2 per cent base rate to 8 per cent, but co-morbid substance abuse increases this rate further to 30 per cent.[6] Co-morbid substance abuse and personality disorder substantially increase the risk of violence, as demonstrated in the MacArthur Risk Assessment Study which showed rates of violence in discharged psychiatric patients of 18 per cent in those with major mental disorder, 31 per cent with major mental disorder and co-morbid substance abuse, and 43 per cent in those with personality disorder and co-morbid substance abuse.[7]

Public fears are often fuelled by media reporting of high-profile cases of homicide by people with mental illness.[8] Despite indications that rates of homicide among the mentally ill are relatively constant across countries,[9] studies of mental disorder in people convicted of homicide show that 8.7 per cent of homicides in New Zealand are 'abnormal',[10] yet evidence from Canada indicates that 35 per cent of perpetrators are mentally unwell.[11] Indeed rates ranging from 8 to 70 per cent have been found, varying with different definitions of mental disorder.[12]

The National Confidential Inquiry into Suicide and Homicide by People with Mental Illness was established at the University of Manchester in 1996. The core work of the Inquiry is to establish rates of mental disorder in homicide and the clinical care received by those in contact with services.

General population homicides

There are 500–600 homicides annually in England and Wales. Perpetrators and victims are predominantly young males, especially when the victim is unknown to the perpetrator. In such 'stranger homicides' perpetrators are less likely to have a lifetime history of mental illness, symptoms of mental illness at the time of the offence, or contact with mental health services.

In the UK the total number of both total homicides and stranger homicides increased between1973 and 2003 but neither category increased in people with mental illness.[13] Similar trends have been noted in work from both the UK,[14] and in other countries.[10]

The commonest method of homicide is with a sharp instrument; shooting is relatively rare, accounting for less than 1 in 10 homicides in the UK.

Around half of all convictions are for murder and just under half for manslaughter. One in 25 receives a verdict of Section 2 manslaughter, diminished responsibility.

Infant homicide

Despite an increasing rate of homicides in the general population, convictions for infanticide and the rate of infant homicide has remained relatively constant, at around 4.5 per 100 000 live births.[15] Infanticide has become a generic term for killing of infants, even though the criminal charge in England applies to a crime for which only a woman can be indicted.

Although the risk of homicide is higher in the first year of their life than at any other time, the rarity of infant homicide in absolute numbers means that there is a lack of high quality, systematic data at a population level which incorporates clinical characteristics.[16]

Data from the National Confidential Inquiry from 1996 to 2001 shows that 1 in 25 of the 2665 homicide perpetrators identified were convicted of infant homicide. Half of these infants were killed by their father and around a third by their mother. A quarter of perpetrators had symptoms of mental illness at the time of the offence and a third had a lifetime history of mental illness. Perpetrators of neonaticide were predominantly young, unmarried mothers experiencing symptoms of dissociation at the time of the homicide.

There were significant differences between male and female perpetrators, with males being more likely to have previous convictions for violent offending. Females were more likely to kill within a month of the birth and they were more likely to have affective disorder and symptoms of mental illness at the time of the offence but few of these women were under the care of mental health services.

Most males received a custodial sentence, whereas three quarters of women received a community sentence or hospital disposal.[16]

Multiple homicides

Multiple homicides, in particular serial homicides, have generated a great deal of public and media interest over recent decades yet this phenomenon is rare in the UK. The rarity of these events means that there is a lack of empirical evidence about the characteristics of perpetrators and victims in the UK, with most evidence emanating from the United States. Even then, however, there is an absence of systematic, robust evidence, with many studies being limited by small sample size.

Most definitions of multiple homicides include three criteria; number of victims, which can vary from 2 to 10 in different definitions,[17] time, and motivation. The temporal relationship distinguishes subcategories: mass murder consisting of a single episode and location, with serial, and spree murders occurring over time in separate locations. The latter two are differentiated by an emotional 'cooling-off' period, which is present in serial homicide. Other authors have discussed motivation, such as sexual gratification and internal psychological gratification, but the lack of robust evidence means that it seems premature to include motivation as part of any such definition.[17]

Mass murder has been classified by victim type such as family annihilators and classroom avengers. Mullen[18] proposed a category of 'autogenic (self-generated) massacre', which encompassed perpetrators indiscriminately killing people in pursuit of a highly personal agenda, arising from their own specific social situation and psychopathology. They were characterized by social isolation, being bullied in childhood and personality traits such as suspiciousness, obsessional behaviour, grandiosity, and persecutory beliefs. He concluded that these murders are essentially murder-suicides, where the intention is to kill as many people as possible before killing themselves. It would now appear, particularly with recent events in Virginia, that this form of multiple homicide is an established form and concerningly appears to becoming more common. Cantor *et al.*[19] propose that media-related modelling is a potential factor in the emergence of this crime, with perpetrators often seeing themselves as lone warriors, themselves modelled on media images, and well informed about previous, similar, massacres.

An exploratory study, incorporating a nested case control study, showed that serial homicide offenders were more likely to be male dominated, compared with single homicide offenders, and were more likely to use strangulation. Moreover, victims of serial murders were significantly more likely to be females who were unknown to the perpetrator and the motivation being sexual.[17] Unfortunately most classification systems of serial murder, including the FBI classification, have been criticized as being inherently flawed due to weak operational definitions and unsubstantiated assumptions regarding behaviour and characteristics.[20]

There is, unfortunately, a lack of robust evidence regarding multiple homicides. There seem to be clear similarities between serial and mass murderers, but also fundamental differences. Mass murderers appear more likely to use firearms as a method, whereas serial murderers tend to kill in a more personal manner, using methods that afford greater physical proximity, such as strangulation, in addition to a greater propensity for female victims. Clinically, some evidence indicates that a substantial proportion of mass murderers have a severe mental illness, often a psychotic illness. On the other hand, it is proposed that serial murderers can be distinguished by lower levels of severe mental illness and the presence of higher degrees of psychopathy.[21]

Female perpetrators of homicide

Around 1 in 10 perpetrators of homicide in England and Wales are female,[13] which is consistent with data from other countries, such as Finland.[22] Stranger homicide by females is rare. In one-quarter of cases the victims are the perpetrators' own children and a current or former partner in over a third.

As with men the commonest method is stabbing, although females are proportionally more likely to use suffocation or poisoning when compared with men.

There are no clear gender differences in the proportion of those with severe mental illness but females are proportionally more likely to have a diagnosis of alcohol or drug dependence than men. Females are less likely to receive a prison sentence and are more commonly placed on a hospital or community rehabilitation order.[13]

Homicide by older people

Homicides perpetrated by the elderly are exceptionally rare. In England and Wales they account for less than 1 in 50 homicides. The male to female ratio in perpetrators over 65 years is the highest of all age groups, at 19:1.[13]

There is a distinct lack of robust evidence regarding homicide in this population. Elderly spouse homicides have been described by Knight[23] as involving a couple perceived to have a close, caring relationship with the homicide of the wife, by the husband, occurring in an abrupt and unexpected manner. Depression is well recognized in elderly homicides, not infrequently with associated delusions of impoverishment and ruin. Perpetrators are often in care giving roles with physical or psychiatric disability in the victim. The homicide is often followed by suicide of the offender.[24]

Those over 65 years are more likely to receive a hospital order or community disposal, than a custodial sentence.[13]

Perpetrators of homicide with mental health service contact

The aim of the National Confidential Inquiry is to collect detailed clinical information on people convicted of homicide, focusing on those with a history of contact with mental health services.

The inquiry collects a national consecutive case series of patient homicides occurring since April 1996. Data collection involves collecting information on all homicides from the Home Office Homicide Index, which includes details of the perpetrator, victim, and method used. Where available, psychiatric reports prepared for the trial are obtained. Antecedent data (of previous offences) is collected from the National Crime Operations Faculty. Details on each case are submitted to mental health services in each individual's district of residence and adjacent districts to identify those with a history of mental health service contact. These individuals become Inquiry cases. Information on trust Inquiry cases is obtained from clinical teams via a comprehensive questionnaire sent to the consultant psychiatrist.

In the UK around 1 in 10 people convicted of homicide have been in recent contact with mental health services. In most of these cases the responsible service is a general adult psychiatry service, rather than a specialist service. The remaining cases are under alcohol and drug services, child and adolescent services, and forensic psychiatry services. Around one in five cases have had lifetime contact with services. This compares with data from other countries, such as Australia, where one in three perpetrators has had contact with psychiatric services.[25]

Of those in contact with mental health services in England and Wales, the most common diagnosis is schizophrenia, although less than half have severe mental illness (schizophrenia or affective disorder). There are high rates of co-morbid alcohol and drug dependence and personality disorder. Only one-third have previous admissions under the Mental Health Act (1983).

A high proportion of these patients have a history of violence, including convictions for violence, which, worryingly, are not documented in the case notes in a number of cases. Similar findings regarding the prevalence of violence were found in an examination of findings from public inquiries into homicides in the UK.[26] A small number of homicides are committed by patients who have previously been on a restriction order because of a violent offence.

Around half of those prescribed medication are non-compliant or disengaged from services at the time of the offence and relatively few are receiving any psychological intervention.[13]

Perpetrators with schizophrenia

There is a well documented increased risk of violence in those with schizophrenia.[27] This has been shown in studies from the UK[28] and in other countries such as New Zealand[29] and Denmark.[30]

Around 1 in 20 perpetrators of homicide have a diagnosis of schizophrenia, a half have been in recent contact with services and one-third have never had any service contact.[13] These findings are broadly consistent with other UK data from remand prisoners,[31] and with data from other countries with rates of schizophrenia ranging from 7 per cent in Finland,[32] 7.5 per cent in Australia[25] to 12.6 per cent in Canada.[11]

Of those with recent contact one-fifth have a secondary diagnosis, commonly personality disorder or substance dependence, and a history of violence is common. Nearly a half have a history of violence when psychotic, around one-quarter are psychotic at the time of the homicide. Victims are most commonly family members; in less than one in six cases the victim is a stranger. Similar rates of stranger homicide by the mentally ill are found in Australia and New Zealand.[10] The majority of these patients have symptoms of mental illness at the time of the homicide and one in four receives a verdict of diminished responsibility. Of those not in contact with services, the vast proportion are psychotic at the time of the offence.[13]

It is of concern that nearly one-third of all perpetrators with schizophrenia receive a prison disposal.

Despite clear evidence of an increased risk associated with schizophrenia it is important to present a balanced view to prevent unnecessary stigmatization. The proportion of violent crime in society which is attributable to schizophrenia is consistently less than 10 per cent.[27] Wallace *et al.*[25] showed an increased risk of serious violent offending in males with schizophrenia of five times that of the general population. However, he also highlights data which indicate that, in any given year, 99.97 per cent of all those with schizophrenia will not be convicted of a serious violent offence, and that the probability of patients with schizophrenia committing homicide is extremely low.

Risk assessment

Nearly one in three Inquiry cases were seen during the week before the homicide, a similar proportion within 1–4 weeks and the remainder between 1–12 months. A substantial proportion had mental state abnormalities at final contact, often distress, depressive symptoms, hostility, or increased use of alcohol or drugs. Despite this immediate risk was judged to be low or absent in 88 per cent cases at the last contact.

There are clear difficulties in predicting risk of serious violence, given the rarity of its' occurrence alongside the high prevalence of risk factors such as substance abuse and a history of violence within the patient population. In an examination of findings from public inquiries into homicides it was shown that only 28 per cent of homicides were judged 'predictable', yet 65 per cent were seen as 'preventable'. 'Preventability' was conferred by 'improved mental health care'.[26]

Use of enhanced CPA to manage risk

In the National Confidential Inquiry sample from 1999 to 2003, nearly three quarters of those with recent contact were not receiving care under the provisions of enhanced Care Programme Approach (CPA), including a substantial proportion of patients at high risk such as those with schizophrenia, personality disorder, a history of detention under Mental Health Act legislation, or a previous history of violence. Furthermore, one-third of those with severe mental illness, a history of violence, and detention under the Mental Health Act were not under enhanced CPA.

Among those who were being cared for under the provisions of enhanced CPA, a significant number were non-compliant with medication or disengaged from services at the time of the offence. It seems, therefore, that even if risk is recognized high-risk patients are not receiving the intensive care, commensurate with their level of risk, in the community.[13]

Preventability

Clinicians identified one case in five in recent contact where the homicide could potentially have been prevented. Factors viewed as increasing the chance of preventing the homicide included a diagnosis of schizophrenia, multiple previous admissions, and detention under the Mental Health Act. Factors which were seen to have made the homicide less likely were better patient compliance; closer contact with patient's family; closer patient supervision; improved staff communication; and better staff training.[13]

Longitudinal trends

When longitudinal data from the National Confidential Inquiry from 1997 to 2003 was examined it was apparent that, despite a rise in the homicide conviction rate in the general population, there has been no consistent change in rates of mental illness symptomatology at the time of the offence, contact with mental health services, lifetime history of mental illness, or specifically schizophrenia. Significant upward trends can be seen in the number of perpetrators with a history of drug and alcohol misuse, in particular the use

of cocaine and crack cocaine. There has been a significant decrease in those receiving a verdict of diminished responsibility but, surprisingly, no change in rates of those receiving a hospital order.[13]

Further information

http://www.medicine.manchester.ac.uk/suicideprevention/nci/ (the website for the National Confidential Inquiry which is regularly updated).

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11.6

Fraud, deception, and thieves

David V. James

Introduction

Dishonesty and deception are mundane and ubiquitous elements of human behaviour. Various forms are also categorized as criminal offences in the codes or statutes of all organized societies. In criminological terms, fraud, deception, and theft are forms of stealing, in other words dishonestly depriving others of goods or services. However, deception and fraudulent misrepresentation play a much wider role in human behaviour and interactions. This chapter will first consider briefly the broader picture, before considering in detail psychiatric aspects of stealing.

Fraud and deception

It is a common tendency to distort memory or reality through the lens of wishful thinking. To deny, to lie to others, and to engage in self-deception is part of the human condition. At one level, this can amount to innocuous forms of self-distraction, such as daydreaming or the childhood world of make-belief. Both blend the borders between fantasy and reality. Deception, which more bluntly put means lying, is on the other hand instrumental in purpose. The essential elements of lying are a conscious awareness of falsity, the intent to deceive and a pre-conceived goal or purpose. In some walks of life, such as advertising or politics, it may be part of the job, at least when the individual thinks that they can get away with it. Deception of others may slide into self-deception, the editing of memory to suit current desires. This may concern a wide range of life's extravagancies, from the fraudulent endeavours of the narcissistic fantasist to the imperatives of those who convince themselves, or wish to convince others, of their own supposed illnesses or infirmities. Particular forms of fraud, deception, and self-deception relevant to the medical context are factitious disorder, malingering, and pathological lying: these are dealt with in Chapter 5.2.9.

Theft

Types

Categorization of offences differs between jurisdictions. However, that for England and Wales forms a basic, illustrative framework. Offences are categorized under the Theft Act 1968 into theft, burglary, vehicle offences, and deception. The Theft Act 1978 further clarifies the area of deception (dishonestly obtaining services from another), dividing it into evading liabilities and debts, and making off without payment. The Fraud Act 2006 provides for a general offence of fraud with three ways in which it can be committed—by false representation, by failing to disclose information, and by abuse of position.

Rates/prevalence

Rates of criminal theft, fraud, and deception can be examined in terms of offences committed per unit of population. In England and Wales, with a population of 53.3 million, the number of reported fraud, theft, and deception offences in 2006 was 2 789 600, which equates to approximately 5.2 per 1 000 total population. These comprised 51 per cent of all crimes reported to the police.[1] However, crime is under-reported to the police and it is notable that the rate of offending as recorded by population survey in England and Wales is generally more than double. International comparisons of crime figures are hampered by differences in classification, measures, and time period.

A motivational classification of theft

Theft and dishonesty may arise from a range of motives. There is no necessity to invoke mental disorder in order to explain thieving. However, a minority of cases are related to serious mental disorder, such as mania, schizophrenia, depression, and organic brain disorder; and other forms of abnormal psychological processes can act as drivers for criminally dishonest behaviour. These go beyond the formal classification of such behaviours in, for instance, the DSM-IV-TR,[2] where these appear only in the specific syndrome of kleptomania and as a component of the definitions of conduct disorder and anti-social personality disorder.

The following classification is not exhaustive and the categories not mutually exclusive. The great majority of thefts will fit into the first two categories, and it is the remainder that are more likely to form part of a presentation to a psychiatrist.

(a) Ordinary theft

Ordinary theft may be planned or impulsive, but is deliberate and motivated by the usefulness of the object or its monetary value.

1 Professional—crime as a career choice.

2 Delinquent—theft as one component of a delinquent or anti-social lifestyle.

3 Survival offences—driven by poverty, desperation, or necessity.

(b) Emotionally driven theft

Emotionally driven theft is the consequence of emotion, rather than as a means of financial gain.

4 Anger or revenge—based upon depriving someone else, rather than personally acquiring.

5 Fear—coerced into committing an offence by threats from a third party.

6 Excitement—this can be as part of a dare or a rite of passage, particularly in adolescents.

(c) Secondary theft

Secondary theft is attributable to the presence of an underlying disorder.

(i) Pecuniary

7 To fund addictive behaviour—e.g. alcohol dependence, drug addiction, pathological gambling.

(ii) Non-pecuniary

8 Depressed stealing:

(a) cry for help (attention-seeking behaviour): stealing in a way that is sure to be detected in order to obtain support and other help.

(b) suicidal gesture: a depressed persons may steal articles of which they have no need, the action serving as a justification for feelings of guilt and a form of suicidal equivalent.

(c) substitution: the stolen object compensates for something else. For example a rejected wife might steal from her husband in order symbolically to establish a degree of control and compensate for the loss of affection.

(d) distraction: e.g. 'absent-mindedness' in a shop as a consequence of distraction by depressive ruminations, or distress in the context of bereavement or divorce.

9 Manic stealing: stealing in the context of manic disinhibition or delusion.

10 Psychotic: the result of delusional drive or command hallucinations.

11 Confusion: related to cognitive deficit, as a consequence of organic brain disorder, or to dissociative states.

12 'Kleptomania': compulsive stealing as an impulse-control disorder.

Management

A psychiatrist will only have a role in the management of theft cases where a particular psychiatric or psychological issue is central. The management will depend upon the nature of the underlying disorder. Treatment for the primary disorder will, in most cases, be supplemented by a psychological approach to helping the patient understand the reasons for their offending behaviour, recognize triggers or danger points, and develop strategies for dealing with these in the future.

Kleptomania constitutes a particular diagnostic entity, and shoplifting is a common behaviour which not uncommonly leads to requests for psychiatric reports from the courts. Both will be considered in more detail below.

Kleptomania

Definition

Kleptomania is an old term, first used by Marc and Esquirol in 1838 to indicate a 'stealing madness'. Kleptomania was designated a psychiatric disorder in DSM-III in 1980, and, in DSM-IIIR in 1987, it was grouped under the category 'impulse control disorder, not elsewhere classified'. The disorder is described further in Chapter 4.13.1 (Impulse Control Disorders) and only aspects most relevant to forensic practice are considered here.

The core characteristic is that objects are stolen despite the fact that they are typically of little value to the individual, who could have afforded to pay for them. Sometimes, they are hoarded and sometimes dispensed with or returned. DSM-IV-TR describes the essential features of the disorder in five diagnostic criteria (312.33):

(a) 'Recurrent failure to resist impulses to steal objects that are not needed for personal use or for their monetary value.

(b) Increasing sense of tension immediately before committing the theft.

(c) Pleasure, gratification, or relief at the time of committing the theft.

(d) The stealing is not committed to express anger or revenge and is not in response to a delusion or a hallucination.

(e) The stealing is not better accounted for by conduct disorder, a manic episode, or anti-social personality disorder'.

In addition, individuals with kleptomania 'experience the impulse to steal as ego-dystonic and are aware that the act is wrong and senseless'.

Diagnostic problems

Criterion A, which concerns the senselessness of the theft, is often considered to be the characteristic which separates kleptomania from ordinary theft. However, some kleptomaniacs may desire the items and be able to use them, even if they are not strictly needed: this may be particularly the case with those who hoard.[3] Concerning criteria B and C, some patients report amnesia surrounding the time of the theft and therefore deny feelings or tension immediately beforehand or relief at the time of committing the theft.[3,4] There is also some suggestion that kleptomaniacs who repeatedly steal over long periods eventually lose the feelings of tension and pleasure in a behaviour which has simply become a habit.

There may be overlap with other disorders.[5] Kleptomania and drug addiction share similar core qualities.[6] The presence of repetitive thoughts and behaviours suggests to some a link with the obsessive-compulsive spectrum.[7] With the high comorbidity with depressive disorders, kleptomania might be categorized within the affective spectrum.[8]

Epidemiology

The prevalence of kleptomania is unknown though it is thought to account for less than 5 per cent of shoplifting.[2] In the US the lifetime prevalence may be 0.6 per cent[3] though some consider that it may be higher because the associated embarrassment and illegality may deter people from reporting it.[9]

A US study of psychiatric inpatients found a lifetime prevalence of 9.3 per cent.[10] While of 107 inpatients with depression,

3.7 per cent had kleptomania[11] and, of 79 inpatients with alcohol dependence, 3.8 per cent reported symptoms consistent with kleptomania.[12]

Kleptomania was generally believed to be much more common in women, but in a summation of four studies of kleptomania 63 per cent were women,[5] suggesting that the preponderance of women is not as great as was once assumed.

Comorbidity

Kleptomania is highly comorbid with depression and anxiety.[13–15] Estimates of lifetime comorbid rates of mood disorders range from 59[9] to 100 per cent,[16] and of anxiety disorders from 60 to 80 per cent.[17] Some of the comorbidity with depression and anxiety may be secondary to the consequences of the behaviour. Compared with people with alcohol dependence and general psychiatric disorders, those with kleptomania scored significantly higher on measures of impulsivity, sensation-seeking, and disinhibition.[14] In one study,[18] 43 per cent of people with kleptomania met the criteria for at least one personality disorder, the most common being paranoid, schizoid, and borderline. However in a meta-analysis none satisfied the diagnostic criteria for anti-social personality disorder.[19]

Aetiology

The aetiology of kleptomania is uncertain. Suggested causes include attempts to relieve feelings of depression through stimulation,[20,21] or to make up for early deprivation.[5,22] The various theories are considered further in Chapter 4.13.1

Treatment

Treatment involves a combined psychological and pharmacological approach. There is little evidence to suggest one particular psychological approach, but a combination of cognitive behaviour therapy and psychosocial interventions is generally adopted. As regards drug treatments, there have been case reports and small series suggesting the efficacy of a range of drugs,[23] in particular selective serotonin re-uptake inhibitors, mood stabilizers, and opioid antagonists. However, there is a need for controlled trials before definitive conclusions can be drawn.

Shoplifting

Shops specialize in making items seem desirable and tempting to the shopper. Most people shoplift at least once at some point in their lives, usually opportunistically in adolescence. But the incidence of shoplifting remains unknown. An early observational study in the UK[24] suggested that 1 to 2 per cent of customers in an English department stores took items without paying, which compared with 1 in 12 in New York City and 1 in 18 in Dublin. The number of people convicted of shoplifting is evidently far smaller. Whereas earlier studies had suggested that shoplifting was more common in women,[25] this is now thought to have been an artefact of case selection, as samples in most studies have been limited to court samples or sub-samples referred to psychiatrists for reports.[26]

There is no unitary phenomenon of shoplifting. It has been suggested that those who shoplift fall into two groups—those who do so out of rational choice and those that suffer from depression.[27] A survey of 1649 shoplifting convictions in Montréal found that only 3.2 per cent were suffering from serious mental disorder, but that affective symptoms were relatively common.[28] A further study of 106 shoplifters[26] reported that depression was the most common psychiatric disorder associated with shoplifting and that the majority of shoplifters were poor and unemployed. The range of possible psychiatric disorders associated with shoplifting is indicated in the classification of theft above, but in the large majority of cases of shoplifting, there is no psychiatric reason for avoiding payment.

Management

A thorough assessment is needed to order to establish how and why the shoplifting occurred and to indicate the presence or absence of underlying mental illness or disorder. Where such illness or disorder is present, it should be treated. Where no such factors are in evidence, psychological therapies may still have a role in offender rehabilitation, usually in a group setting.

Further information

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11.7

Juvenile delinquency and serious antisocial behaviour

Susan Bailey

Introduction

Juvenile crime and delinquency represent a significant social and public health concern. Both rates of mental disorders and offending are high during adolescence. This chapter reviews prevalence rates of mental disorders in young offenders, screening, and assessment of juveniles, principles of interventions with young offenders before describing principles of forensic mental health, policy and practice, how mental disorders in adolescence can impact on offending and antisocial behaviour, how policy is shaping practice in this field and how mental health practitioners may be involved in meeting mental health needs and undertaking medico-legal assessments

Delinquency, conduct problems, and aggression all refer to antisocial behaviours that reflect a failure of the individual to conform his or her behaviour to the expectations of some authority figure, to societal norms, or to respect the rights of other people. The 'behaviours' can range from mild conflicts with authority figures, to major violation of societal norms, to serious violations of the rights of others.[1] The term 'delinquency' implies that the acts could result in conviction, although most do not do so. The term 'juvenile' usually applies to the age range, extending from a lower age set by age of criminal responsibility to an upper age when a young person can be dealt with in courts for adult crimes. These ages vary between, and indeed within, countries and are not the same for all offences.[2,3]

Adolescence as a context

The adolescent population in the UK constitutes half of the child population with around 7.5 million young people in the transitional stage between childhood and adulthood, (age 10–19).[4] Adolescence is a transitional stage of development between childhood and adulthood—a stage of possibility and of promises and worries that attend this possibility. The developmental tasks of adolescence centre on autonomy and connection with others, rebellion and the development of independence, development of identity and distinction from and continuity with others. The physical changes of puberty are generally seen as the starting point of adolescence whilst the end is less clearly delineated. Adolescence ends with attainment of 'full maturity'. A range of social and cultural influences including the legal age of majority, may influence the definition of maturity.[5]

Mortality among adolescents, in contrast to almost all other age groups, did not fall during the second half of the twentieth century, the main causes being accidents and self-harm.[4]. Health needs are greater in this age band than in children in middle childhood (5 to 12 years) or of young adults, and arises out of mainly chronic illness and mental health problems. The main concerns of young people, in relation to health, focus on issues of immediacy that impact on their relations with peers and include problems with skin, weight, appearance, emotions, and sexual health including contraception.

The principal aim of the Youth Justice System (YJS) is to prevent offending by children and young people under 18 years of age. There are 157 Youth Offending Teams in England and Wales. The YJB commissions some 3000 custodial places at any one time for young people under the age of 18 years in 18 Prison Service Young Offenders Institutions, 15 Local Authority Secure Children's Home and 4 private sector Secure Training Centres. In 2005–2006 there were 301,860 recorded offences the 4 highest recorded offences being theft and handling 18.5 per cent, violence against the person 18.1 per cent, motoring offences 16.6 per cent, criminal damage 12.9 per cent. 16 and 17 year old were responsible for 49.6 per cent of offences with males responsible for 80.6 per cent and females 19.4 per cent of all offences resulting in a disposal. Offences by ethnicity were white 85.2 per cent and Black and Ethnic Minority 14.8 per cent. Of the 212,242 disposals 80 per cent received pre-court of first tier disposals with 17 per cent receiving a community sentence and 3 per cent a custodial sentence.[6]

Risk factors for, and pathways to antisocial behaviour are summarized in Tables 11.7.1 and 11.7.2 respectively. There is a significant overlap between the risk factors for offending, poor mental health and substance misuse and the number of assessed risk factors increases as a young person moves further into the Youth Justice System.[7] Many young offenders are not engaged in mainstream education and health services. It is critical that these young people are supported to access the mainstream and specialist services they require while under the supervision of the YOT or in custody. Otherwise, once their sentence ends they can become detached from services and their circumstances are likely to deteriorate, leading to more offending and greater demands on specialist services as they get older.

In the UK, the Children's National Service Framework for Children, Young People and Maternity Services (NSF) set out a vision of a comprehensive child and adolescent mental health service.[8]

Table 11.7.1 Major risk areas in children and adolescents with persistent antisocial behaviour[35]

Broad child-centred factors
Genetic vulnerability
Perinatal risk
Male sex
Cognitive impairment
School underachievement
Hyperactivity/inattention temperament
Family factors
Criminality in parents and siblings
Family discord
Lack of supervision
Lack of effective feeling
Abuse
Scapegoating
Rejection
Neglect
Influential contextual factors
Drug and alcohol abuse
Unemployment
Crime opportunity
Peer group interaction

A young person in contact with the criminal justice system, whether in custody, or in the community, should have the same access to this comprehensive service as any other child or young person within the general population. Treatment options should not be affected by a young offender's status within the criminal justice system. The Change for Children Programme has the aim of improving outcomes for all children in the following 5 areas: being healthy, staying safe; enjoying and achieving; making a positive contribution; and achieving economic well-being, and to narrow the gap in outcomes between those who do well and those who do not. If we do not address the mental health needs of young offenders then they are excluded from the opportunity to participate in improvements in these 5 outcomes and ultimately from the ability to achieve their full potential.

Table 11.7.2 Critical pathway to serious antisocial behaviour[64]

Family features
Parental antisocial personality disorder
Violence witnessed
Abuse, neglect, rejection
Personality features
Callous unemotional interpersonal style
Evolution of violent and sadistic fantasy
People as objects
Morbid identity
Paranoid ideation
Hostile attribution
Situational features
Repeated loss and rejection in relationships
Threats to self-esteem
Crescendo of hopelessness and helplessness
Social disinhibition
Group processes
Changes in mental state over time

There is a high prevalence of mental health problems among young people in custody.[9] YJB research published in 2005 reported the following findings.[10]

- 31 per cent had mental health problems
- 18 per cent had problems with depression
- 10 per cent suffered from anxiety
- 9 per cent reported a history of self-harm in the preceding month
- 9 per cent suffered from post-traumatic stress disorder
- 7 per cent had problems with hyperactivity
- 5 per cent reported psychotic-like symptoms

One in five young offenders were identified as having intellectual disability IQ<70. Additionally, needs were identified across education 48 per cent and social relationships 36 per cent. Needs were unmet because they were not recognized.

Research consistently reveals high levels of psychiatric disorders among detained juveniles, although rates vary widely by study, ranging from more than 50 per cent to 100 per cent.[11–21] The variations between the studies may reflect methodological differences or true variations between countries and samples. Advances in developmental psychopathology and increased understanding of the continuities between child and adult life[27] demonstrating that many childhood disorders once thought to resolve with age cast long shadows over later development.

There are several reasons why high rates of mental disorders may be expected in youth in contact with juvenile justice. First, prevalence rates of psychiatric disorders in community samples were shown to be around 15 per cent.[22] Also, severe delinquency is common in the adolescent population, with about 5 per cent showing an early-onset and persistent pattern of antisocial behaviour.[23] A substantial number of adolescents will show offending behaviour and will have a mental health disorder simply because of coincidental overlap between both conditions. Second, because delinquent and antisocial behaviour reaches high levels among juvenile justice populations, a diagnosis of conduct disorder (CD) will often be made. Because CD shows high comorbidity rates with several other psychiatric disorders,[24] increased levels of many types of disorder may be expected. Third, risk factors for youthful offending overlap substantially with those for several types of non-disruptive child psychiatric disorders, therefore identical risk factors may underlie both antisocial behaviour and emotional or developmental problems. Disorders for which mental health interventions are provided, such as substance use disorders (SUD's), may also lead to judicial involvement. Also, because of the prevalence of complex comorbidity, treatment in a regular mental health care programme may be intricate and often is not possible, thus increasing the likelihood of judicial involvement. In addition, severely disordered persons may be less likely to have the personal capability and have adequate resources to defend themselves and to avoid more drastic legal interventions.

Grisso and Zimring listed three principal reasons for concern regarding mental disorders in youthful offenders: a) the obligation to respond to mental health needs in those in custody, b) assurance of due process in adjudicative proceedings, and c) public safety.[25]

Mental health treatment within the juvenile justice system is often inadequate. It has been reported that only about 20 per cent of incarcerated youth with depressive disorders, 10 per cent with other mental disorders, and less than half with SUD receive intervention.[26,27]. Much more research is needed into the treatment needs of this population.

Risk and protective factors

Understanding is growing of how risk factors combine to both precipitate and maintain antisocial behaviour. Several environmental and individual risk factors including psychiatric pathology in childhood have been identified.[28] Not all risk factors need to be present in a single individual but multiple risk factors greatly increases the risk of a serious and long-term negative development.[29] Positive characteristics or experiences may act protectively. These protective factors may be specific interventions or experienced within the natural contact of development. When protective factors are present, young people may show positive social development despite high risk of antisocial behaviour, or they may abandon their problem behaviour after a difficult phase. Such trajectories are less well investigated than the risks.[30–33] It is also more difficult to implement adequate research designs in this field.[34] It could be assumed that the opposite to the risk value of the variables listed in Table 11.7.1 may promote positive development. However, truly protective effects need to compensate for a given high-risk constellation (moderator approach). The available research suggests a number of factors that may protect from the risks of antisocial behaviour. Table 11.7.3 reports a selection of such personal and social resources that have already been proven or may be promising (for a detailed review see[33]).

Table 11.7.3 Multilevel examples for protective factors against serious antisocial behaviour[35]

Biological/bisocial	- Non-deviant close relatives; no genetic vulnerabilities; high arousal; normal neurological and hormonal functioning.
Pre-and perinatal	- Non-alcoholic mother; no maternal smoking during pregnancy; no birth complications
Child personality	- Easy temperament; inhibition; ego-resiliency; intelligence; verbal skills; planning for the future; self-control; social problem solving skills; victim awareness; secure attachment; feelings of guilt; school and work motivation; special interests or hobbies; resistance to drugs
Cognitions/attitudes	- Non-hostile attributions; non-aggressive response schemes; negative evaluation of aggression; self-efficacy in prosocial behaviour; non-deviant beliefs; realistic self-esteem; sense of coherence.
Family	- No poverty; income stability; harmony; acceptance; good supervision; consistency; positive role models; continuity of caretaking; no disadvantage; availability of social support.
School	- Achievement and bonding; low rate of aggressive students; climate of acceptance; structure, and supervision.
Peer group	- Non-delinquent peers; support from close, prosocial friends.
Community	- Non-deprived, integrated and non-violent neighbourhood; availability of professional help.
Situational	- Target hardening; victim assertivesness; social control.
Legal	- Effective firearm and drug control; effective criminal justice interventions.
Cultural	- Low violence; tradition of moral values; shame and guilt-orientation; low exposure to violence in the media.

Pathways of care and the juvenile justice system

Juvenile justice is a high-volume system, which makes clear logistics and a clear pathway of care necessary. Early identification of mental health needs may result in diversion from custody by using community services rather than adjudication and derive economic benefit by affording non-custodial disposal. Nonetheless a significant number of young persons progress to pre-trial assessment, albeit from the home or a residential care setting.

Preadjudication dispositions should be informed therefore by best available screening and assessment processes. In this context specific tools may be used to derive markers of psychopathology and of ongoing risk to self and others as well as to address medico-legal questions posed by the criminal justice system including assessment on disposition, matters of public protection, treatment for mental disorders, and need for security and likelihood of recidivism.

For those detained in prison, screening must determine if urgent problems (such as suicidal intent or consequences of substance use) require immediate attention; a detailed diagnostic assessment of the young person may take a longer period of time and continue as the youngster moves from one institution to another. Later critical transitions, for which an additional screening may be useful, include re-entry into the community, assessment of readiness for re-entry, mental health planning for integrated continuing care post detention as part of a multiagency re-entry strategy, and, where necessary, community residential programmes monitoring emotion or reactions, especially where the young person is returning to stressful conditions such as a troublesome family.

General principles of assessment

Standard clinical assessment tools used in child and adolescent psychiatry cover many of the areas considered in forensic child and adolescent risk assessments.[36] This is especially important as juvenile justice systems in particular are not always equitable. In choosing between the many scales available it is important to question not just their proven scientific properties but also their feasibility for practitioners to use.

It is important to consider the purpose for which the scale is to be used (see table below). Scales that measure psychopathology may not be good ways of assessing the risk that the psychopathology poses. Measures used to map out types of symptom must have good content validity. An instrument required to pick out one group of symptomatic people from the rest of the community (e.g. mental health screening of young people in custody) needs to have

good criterion validity. A related issue is the extent to which the scale is intended to measure change.

Grid for specifying requirements of a structured scale in a juvenile forensic population

Assessment required (yes / no)

Purpose of assessment	Psychopathology	Need	Risk
Screening of all juveniles coming into contact with an agency			
Detailed assessment e.g. for sentencing, planning treatment			
Measuring change e.g. during treatment or sentence			

Child psychiatry uses multi-axial and developmental concepts of child psychopathology. Specific and general intellectual delays are very common among young people in the juvenile justice system[10] as is co-morbidity of disorders. Broad-band interviews, however, offer only poor coverage of rare conditions such as pervasive developmental disorders.

Needs assessment

Needs assessment may have advantages over more traditional ways of diagnosing disorders, mainly because this method also indicates whether specific conditions need attention and intervention. Especially in delinquent youth characterized by multiple problems, such an approach may carry substantial advantage. A health care need should be distinguished from a general need. One commonly used definition of a health care need is '*the ability to benefit in some way from (health) care*'.[37]

Needs and risk assessment are two separate but intertwined processes essential for clinical management (see Fig. 11.7.1). Assessment of danger to others and the need to address this problem is at the centre of legislative and policy decision-making. The attention of the public and media are focussed on this area. Needs assessment may both inform and be a response to the risk-assessment process.[38] The reciprocal process can be termed 'risk management' when accurate information about the risk assessment, combined with recurrent needs assessment, leads to risk-management procedures. A recurrent needs-assessment and risk-assessment process should identify changes in problem areas, thus leading to monitoring or intervention as part of risk management. Core to this assessment are appropriate mental health screening tools and processes that are available to the young person at any point in the system.[39]

Risk assessment

Risk assessment combines statistical data with clinical information in a way that integrates historical variables, current crucial variables, and the contextual or environmental factors. Structured risk assessment instruments have been developed that aim to increase the validity of clinical prediction. These scales typically contain a number of risk items selected from reviews of research, crime theories, and clinical considerations.[41,42] Items are summed to form a total risk score and may also reveal specific risk patterns (e.g. mainly family or child factors). Such instruments are used for screening, in-depth assessment and related risk management (e.g. for decisions on the child's placement or specific interventions). They can also be applied in differentiated evaluations of intervention programmes. Instruments vary with respect to the age and gender for their clients, problem intensity in the target groups, theoretical and empirical foundations, the number and domains of risk included, scoring procedures, time required for assessment, information sources, institutional contexts of administration and other issues.[43,44] Many instruments have been designed for application in the juvenile justice system.[43,45,46] Most instruments contain factors from various areas of risk (e.g. individual, family, neighbourhood).

Mental disorders and offending

Current concepts focus on a developmental approach to psychopathology in child and adolescent psychiatry and psychology. Physical aggression peaks at around the second year of life and subsequently shows distinct developmental trajectories.[47,48] Attachment enables the mastery of aggression, self-control being developed through the efficient exercise of attritional mechanisms and symbolization. Fonagy has suggested a primary developmental role for early attachment in the development of mentalization (the capacity to understand others' subjective experience). He suggests that impaired mentalization leads to later violence.[49] Threats to self-esteem trigger violence in individuals whose self-appraisal is 'on shaky ground' and are unable to see behind the threats to what is in the mind of the person threatening them. These processes are played out in the complex and toxic co-morbidities seen so much more frequently in child and adolescent than in adult mental health practice.

Oppositional disorders, conduct disorder, and ADHD

Substantially higher rates of physically aggressive behaviour are found in children and adolescents with attention deficit hyperactivity disorder, with those who meet the criteria for ADHD and conduct disorder having substantially greater risks of delinquent acts in adolescence, harmful acts in later adolescence and continued violence and offending into adulthood.[50] Children with hyperactivity, impulsivity, attention deficits and serious conduct problems may also be at risk for developing psychopathy.[51]

Distorted or biased thought processes have over time been implicated in the development of violence. Psychological treatments aimed at reducing violent behaviour in adolescents and young adults traditionally centre on violence as learned behaviour. Patterns of violence and criminal behaviour are seen as embedded in habits of thinking.[52] In juvenile delinquents significant cognitive attributional bias has been shown in aggressive children and youths. They are more likely to perceive neutral acts by others as hostile, and more likely to believe conflicts can be satisfactorily resolved by aggression. In the social context, as the young individual becomes more disliked and rejected by peers, the opportunity for viewing the world this way increases.[53] By their late teens they can hold highly suspicious attitudes and be quick to perceive disrespect from others. In the social context of juvenile incarceration,

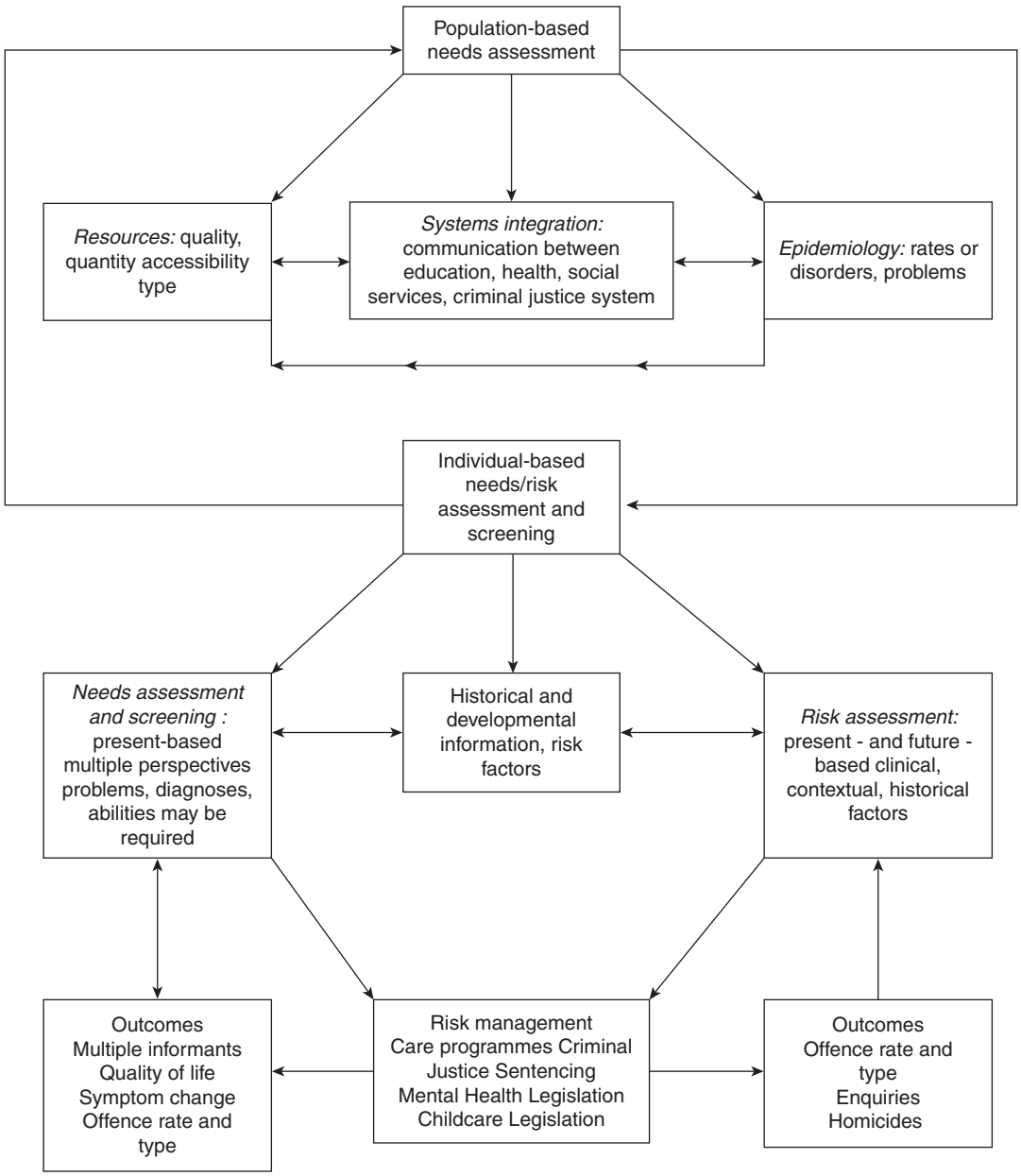

Fig. 11.7.1 Relationship between various screening, need assessment, risk assessment, and management approaches in juvenile justice systems.[40] (Reproduced from Kroll, L. Needs assessment in adolescent offenders. In Adolescent forensic psychiatry (eds. Bailey, S. Dolan, M.), copyright 2004, Hodder Education.)

being 'para'[54] can become in peer group interactions the shared norm.[55]

Depression anxiety and post traumatic stress disorder in childhood and adolescence

As well as the recognized feelings of low mood in depression there is also some evidence of irritability, hostility and anger when depression occurs in adolescence. Irritability in adolescence leads to interpreting annoyances by others as direct threats, increasing the risk of defensive aggression.[56] Nowhere is this more apparent than in juvenile justice populations.[57,58] A self-serving bias with a tendency to attribute good outcomes to the self and bad outcomes to external causes observed in ordinary people, is usually regarded as a mechanism for maintaining self-esteem in the face of threats to the self.

PTSD is related to the conditioning of neurobiological fear responses underlying tendencies to react aggressively to protect the self when exposed to reminders of earlier trauma.[59] In the recent escalating context of both children who have experienced violence in war torn countries and those who live in a context of 'urban war zones' Garbarino[60] sets out an ecological framework to explain the process and conditions that transform the 'developmental challenge' of violence into developmental harm in some children. He set out an accumulation of risk models for understanding how and when children suffer the most adverse consequences of

exposure to community violence and go beyond their limits of resilience, the concept of 'social maps' as products of childhood experiences and of trauma as a psychological wound.

The combination of depression, anxiety and severe PTSD is being increasingly recognized in as being linked to trajectory into adult anti-social personality disorder.[61]

Autism spectrum disorders and learning disability

Autism spectrum disorders are being increasingly recognized in adolescent forensic populations. Their identification is critical to the understanding of violent offending. This is particularly so if an offence or assault is bizarre in nature, the degree or nature of aggression is unaccountable and/or there is a stereotypic pattern of offending. Four reasons have been proposed for offending and aggression in autistic persons:[62,63]

1 Their social naivety may allow them to be led into criminal acts by others;

2 Aggression may arise from a disruption of routines;

3 Antisocial behaviour may stem from a lack of understanding or misinterpretation of social cues;

4 Crimes may reflect obsessions, especially when these involved morbid fascination with violence—there are similarities with the intense and obsessional nature of fantasies described in some adult sadists.[64]

It has been proposed that the paranoia observed in Asperger's syndrome has a different quality from that seen in people with a diagnosis of schizophrenia stemming from a confusion of not understanding the subtleties of social interaction and social rules.[54]

Early onset psychosis

Non-psychotic behavioural disturbance occurs in about half of cases of early-onset schizophrenia and can last between 1 and 7 years. It includes externalizing behaviours, attention-deficit disorder and conduct disorder. This emphasizes the need for mental health assessments repeated over time to include a focus on changes in social functioning (often from an already chaotic baseline level) to a state including perceptual distortion, ideas of reference, and delusional mood.[65]

As in adult life most young people with schizophrenia are non-delinquent and non-violent.[66] Nevertheless, there may be an increased risk of violence to others when they have active symptoms, especially when there is misuse of drugs or alcohol. The risk of violent acts is related to subjective feelings of tension, ideas of violence, delusional symptoms that incorporate named persons known to the individual, persecutory delusions, fear of imminent attack, feelings of sustained anger and fear, passivity experiences reducing the sense of self-control, and command hallucinations. Protective factors include responding to and compliance with physical and psychosocial treatments, good social networks, a valued home environment, no interest in or knowledge of weapons as a means of violence, good insight into the psychiatric illness and any previous violent aggressive behaviour and a fear of their own potential for violence. These features require particular attention but the best predictors of future violent offending in young people with mental disorder are the same as those in the general adolescent population.[67]

Psychopathic personality in young people

A three-factor structure has been proposed,[68] which includes:

- An arrogant, deceitful interpersonal style, involving dishonesty, manipulation, grandiosity and glibness;
- Defective emotional experience, involving lack of remorse, poor empathy, shallow emotions, and a lack of responsibility for one's own actions;
- Behavioural manifestations of impulsiveness, irresponsibility, and sensation-seeking.

Conduct disorder, antisocial personality disorder, and psychopathy are often seen as developmental disorders that span the life course and the terms are sometimes used interchangeably. Conduct disorder and antisocial personality disorder primarily focus on behavioural problems, psychopathy, as described by Hare,[69] emphasizes deficits in affective and interpersonal functioning.

A consensus is likely to be reached only when we have longitudinal studies demonstrating the stability of psychopathic traits over the lifespan and evidence that the same aetiological factors contribute to this disorder at all ages. As there is significant overlap between the behavioural aspects of juvenile psychopathy and ADHD and between the callous-unemotional dimension of psychopathy and autistic-spectrum disorders, future work needs to disentangle these constructs from a phenomenological and aetiological perspective. As yet, there are few treatment outcome studies in juveniles with psychopathic traits, although the limited data suggest that these traits might be a moderator of outcome. Most clinicians view youth psychopathy as a potentially treatable disorder, and there is some evidence that identification of psychopathic traits in young people has a number of benefits, which include:

- Identifying high-risk offenders;
- Reducing misclassifications that have negative ramifications for children and adolescents;
- Improving and optimizing treatment planning for young people with psychopathic traits, who may require more intensive and risk focused therapeutic approaches.

Interventions with juvenile delinquents

A large number of different treatments have been used to reduce antisocial behaviour. These include psychotherapy, pharmacotherapy, school interventions, residential programmes, and social treatments. Kazdin reported over 230 available psychotherapies, the great majority of which had not been systematically studied.[70] This chapter will focus on treatments with a testable scientific basis which have been evaluated in randomized trials and applied to populations of young offenders.[71]

Meta-analyses of treatment approaches to juvenile delinquency have produced reasonably consistent findings.[72–75] Lipsey[73] considered nearly 400 group-comparison studies published since 1950. The main finding was that there was an overall reduction of 10 per cent in re-offending rates in treatment groups as compared to untreated groups. As might be expected, there were of course considerable variations in the results of individual studies. The best results were obtained from cognitive behavioural, skills-orientated, and multi-modal methods. The results from deterrent trials were

particularly poor, though the numbers in these studies were relatively small. Specifically, treatment approaches that were participatory, collaborative and problem-solving were particularly likely to be beneficial. Family and parenting interventions also seem to reduce the risk of subsequent delinquency among older children and adolescents.[76]

McGuire and Priestley[52] identified six principles for effective programmes:

1 Intensity should match the extent of the risk posed by the offender.
2 A focus on active collaboration, which is not too didactic or unstructured.
3 Close integration with the community
4 Emphasis on behavioural or cognitive approaches.
5 Delivered with high quality with training and monitoring of staff.
6 Focus on the proximal causes of offending behaviour (peer groups, promoting current family communication, and enhancing self-management and problem-solving skills) rather than distal causes (early childhood).

The reviews suggest that there are a number of promising targets for treatment programmes, which include antisocial thoughts, antisocial peer associations, promotion of family communication and affection, promotion of family supervision, identification of positive role models, improving problem-solving skills, reducing chemical dependencies, provision of adequate living conditions, and helping the young offender to identify high risk situations for antisocial behaviours. Conversely, the systematic reviews have also suggested a number of approaches that are unlikely to be promising. For instance, improving self-esteem without reducing antisocial cognitions is unlikely to be of value. Similarly, it is unlikely that a focus on emotional symptoms that is not clearly linked to criminal conduct will be of great benefit.

Life experiences associated with treatment ressistance are summarized in table 11.7.4.

Promising interventions for adolescent antisocial behaviour

A rational starting point when considering interventions would be to consider the main causal factors and processes (see above), and design interventions around them. However, in practice many other considerations have shaped interventions, from the desire to punish offending youths, to making use of what is currently available at relatively low cost. For generic interventions for conduct disorder also highly relevant to juvenile delinquency and serious antisocial behaviour (see Chapter 9.2.5).

Table 11.7.4 Life experiences associated with treatment resistance

Early modelling experiences
Early exposure to related phenomena
Enduring antisocial behaviours and aggressive response patterns
Limited judgement skills
Low academic achievement
Clusters of confrontative acts
Personality traits of callousness
Jealousy and revenge
Limited parental/carer supervision
Erratic punishment schedules
Absent, neglecting, or abusive parenting
Parental psychopathology

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Working with young offenders with mental health problems—some practice points for interventions

Interventions with juvenile offenders, regardless of whether they are addressing offending behaviour or mental health problems, should take into account developmental and cognitive factors significant to this age group. Interventions designed for use with adults are usually highly structured and target driven. This style of intervention is often perceived by juvenile offenders as alienating and not relevant to their lives with the result that they are likely to disengage (either actively or passively) from the programme.

The skills required to engage a juvenile offender in a 'therapeutic alliance' are often different to those necessary with adult offenders. Adult offenders are more likely to see the value of participating in an enhanced thinking skills course, possibly as a means to an early release or to improve the quality of relationships within their family unit. Such goals may be perceived as too long range to have any meaning to a juvenile offender, or simply seem irrelevant. Juvenile offender's general experience of relationships with adults, particularly professionals, is of authority figures that give instructions, set limits on behaviour, and at best are givers of information. The typical responses to this are to adopt an aggressive posture or one of passive indifference. To actively engage with juvenile offenders professionals need to listen attentively and show interest in the young person's perspective. This does not mean agreeing with the young person's point of view; however, it is an opportunity to establish the 'middle ground' within which a therapeutic alliance may be fostered.

Adolescent offenders have often experienced unstable lives with disrupted attachments. Thus they often have difficulty in understanding the significance of life events such as trauma and bereavement that an adult will readily understand is likely to impact on emotional well being. A thorough assessment prior to commencing an intervention and drawing on material from multiple sources, particularly parents or professionals who have a detailed knowledge of the young person, is very helpful. Events such as the loss of a parent and the onset of conduct problems are often closely linked temporally, yet young offenders frequently do not see any connection between such events.

A formulation is a brief statement that summarizes the possible links between different aspects of the young person's life, for instance between a bereavement and the onset of behavioural difficulties. Juvenile offenders are often unable to make formulations because of their failure to understand how different elements of their lives are connected. The ability to make formulations can be seen as a developmental milestone and adolescent offenders, for the reasons identified above, often lag behind their own peer group as well as the adult population. Regardless of whether addressing offending behaviours or mental health problems, professionals working with juvenile offenders need to generate

such formulations collaboratively so that they make sense both within the therapeutic framework and also within the young person's life experience.

Establishing therapeutic goals also needs to be collaborative and developmentally appropriate. Adolescence in general is characterized as a time of heightened emotional responses (partly as a result of hormonal changes), a growing but still limited capacity for problem solving and the tendency to seek immediate advantage rather than long-term gain. All of these factors are likely to be enhanced in juvenile offenders in contrast with the rest of their peer group. Goal setting therefore needs to concentrate heavily on the short-term, i.e. within the young person's perceived time frame. Targets and rewards should be low key in order to reduce the likelihood of extreme emotional responses to success or failure. Therapists working with juvenile offenders need to be active in encouraging the generation of alternative solutions in order to extend the young persons range of problem solving skills.

Juvenile offenders' capacity to generate alternative strategies is often limited by their own, limited emotional range. They will typically respond to any adverse event with hostility and anger; events that would typically evoke a response of sadness or fear within adults. Work on emotional recognition with juvenile offenders will assist them in recognizing a wider range of emotions, both in themselves and others. This enhances their range of options when faced with future adverse events.

There is often a tendency to concentrate on behavioural objectives as the most easily recognized or measured outcomes. However, working on goals such as recognizing and managing arousal levels, or shifting cognitions or attributions in specific situations may prove more beneficial in the longer term even if immediate behavioural changes are not achieved.

Goal setting and intervention strategies should be individually tailored and take into account differences in cognitive ability, maturity and insight within this population. To ensure the young person remains actively engaged in the intervention process it is important to frequently check out their perception of how effective the therapy is, and whether the goals and strategies are relevant. Therapists should frequently check the young person's level of understanding to ensure that the communication is two way and repeats elements or themes as necessary to ensure good compliance and comprehension, rather than adhering to a timetable.

Treatment and special crimes

Juvenile homicide

Violent behaviour often involves a loss of sense of personal identity and of personal value. A young person may engage in actions without concern for future consequences or past commitments.

Violence denotes the 'forceful infliction of physical injury'.[77] Aggression involves harmful, threatening or antagonistic behaviour.[78] Longitudinal studies are invaluable in mapping out the range of factors and processes that contribute to the development of aggressive behaviour and in showing how they are causally related.[79] However, in attempting to work with any individual who has committed a violent act, the question to be answered is 'why this individual has behaved in this unique fashion on this occasion'.[73]

Studies show that children and adolescents who murder share a constellation of psychological, cognitive, neuropsychiatric, educational and family system disturbance.[80–82] In the UK, young people who commit grave sadistic crimes including juvenile homicide are liable to periods of lengthy incarceration. Detention itself can provide time for further neuro-developmental, cognitive, and emotional growth. Irrespective of treatment models, the provisions of education, vocational training, consistent role models and continued family contact are of critical importance.

The approach to juvenile delinquency, including juvenile homicide, in the Netherlands is determined by a policy of minimal intervention, with a strongly pedagogical point of view. The emphasis lies on education and treatment rather than on punishment. Cases recorded by the police as murder or manslaughter in the first instance may not ultimately be presented to court as such. Moreover, the pedagogical nature of the punishments is recognizable in the fact that treatment is ordered in most cases, in the form of placement in a juvenile institution, sometimes in combination with imprisonment, rather than a straightforward youth detention or imprisonment. There are no indications of special policies for prosecuting or handling the cases of legal minors suspected of murder or manslaughter. The limited prevalence of this phenomenon is undoubtedly partly due to the fact that every case is judged on its own merits. Incidentally, this is also noted in the USA where a considerably higher number of youths are sentenced for murder and manslaughter.[83]

Youths who have been prosecuted for murder or manslaughter vary only slightly or not al all from other juvenile delinquents on points such as age, gender and ethnic background, and only to a limited extent on risk factors. Murder and manslaughter are committed alone comparatively more often, and on average the perpetrators start their criminal activities at a later age and are much less likely to have previous convictions than other minors taken into judicial youth institutions. At the same time, it is clear that while the group of youths involved in murder and manslaughter may be small, it is anything but homogeneous. There is great variety in terms of motives, victims, modus operandi etc. In simple terms each case stands on its own.

The majority of young persons who have killed initially dissociate themselves from the reality of their act, but gradually experience a progression of reactions and feelings akin to a grief reaction. The young person whilst facing a still adversarial, and public pretrial and trial process has to move safely through the process of disbelief, denial, loss, grief and anger/blame. Post traumatic stress disorder arising from the participation in the sadistic act (either directly or observing the actions of co-defendants) has to be treated, as does trauma arising from their own past personal emotional, physical and/or sexual abuse.

A combination of verbal and non-verbal therapies are effective but qualities such as previous frequent and severe aggression, low intelligence and a poor capacity for insight weigh against a safe outcome.[84] In understanding the role of violence and sadism in a young person's life one has to understand the depth of their sensitivity and reaction to perceived threat and their past maladaptive behaviours aimed at allowing them to feel in control of their lives. In coming to terms with their internal rage, addressing victim empathy saying sorry and reattribution of blame, expression of anger and distress within sessions is expected and is often sexualized in both form and content. This can spill outside sessions when the young person and carers can become collusively dismissive and rejecting of therapists,[85] emphazing the importance of intensive

work to prepare the young person for transition from long term incarceration and re-entry into the community together with extended aftercare.

Sexually abusive behaviour

Sexually inappropriate behaviour in children and adolescents constitutes a substantial health and social problem.[86] Most, but not all, abusers are male, often come from disadvantaged backgrounds with a history of victimization, and sexual and physical abuse[87] and show high rates of psychopathology.[88] Of particular concern are a significant subgroup with mild intellectual disability whose treatment programmes have to be tailored to their level of development and cognitive ability. Young abusers come within the Criminal Justice System but also should be considered in their own right within the child protection framework. Most adult sexual abusers of children started their abuse when adolescents and yet neither ICD10 nor DSMIV has a diagnostic category for paedophilia in those under 16.

A structured carefully planned multi-agency approach is required when working with sexually aggressive younger children and sexually abusive adolescents. The three stages to assessment of juvenile sexual offenders are:

1 Clarification and rapport building

2 Mapping the abuse: the fantasies, strategies and behaviours; and

3 The future, placement treatment and personal change.

The treatment process occurs in the context of:

- The crisis of disclosure;
- Family assessment;
- Therapeutic work in a protective context for the victim; and
- Reconstruction and reunification of the family.

The 'family' in this context may include foster carers, or long term residential carers.

Treatment outcome

The earliest possible interventions with young over-sexualized children, before their patterns of sexually aggressive behaviours become entrenched, are likely to be most effective. However, there is a dearth of longitudinal follow-up studies looking at treatment outcomes with this younger group of children.

Outcomes may be measured by looking at recidivism, treatment outcome or other measures. At present, there are no longitudinal outcome studies of children and adolescents with sexually abusive behaviour which have measured other outcomes such as adult adjustment, attitudinal change or parenting.

Recidivism as a sole outcome measures for treatment is unlikely to be reliable since persistent sexual behaviour problems in children under the age of criminal responsibility will not appear in crime statistics and conviction statistics for sex offenders of all ages are notoriously unreliable for a variety of reasons including failure to report victimization experiences, failure to proceed with charges and a high rate of trial failures.

Other factors appear to be highly relevant to treatment outcomes with juvenile sexual abusers such as good interprofessional communication and a systemic context for treatment to occur.

New approaches to CBT with sexually abusing youths have recently been described within the context of relapse prevention and a more complex CBT intervention, Mode Deactivation Therapy (MDT), has been suggested for disturbed, sexually abusive young people with reactive conduct disorders or personality disorders[89] CBT group work with sexually abusing children and young people is widely practised in the UK and the principles of this work are described by Print and O'Callaghan.[90]

Other treatment approaches will take into account the living context of the young person and the need for his or her carers to be provided with support and explanation of the treatment process in order to maximize positive results. For instance, when children and young people who sexually abuse are still living at home or in contact with their parents, family work is usually needed. An approach to group work with parents of children with sexually abusive behaviour has been described.[91] In the case of children and young people who are living in the care system, concurrent work for the professionals and carers looking after the sexually abusing child or young person has been strongly advocated.

There are a significant number of mid-adolescent, recidivist, delinquent, sexually abusive youths who are too dangerous to other children and young people to be treated (with any treatment modality) alongside other young people. Many of these young people have been through the court system or are currently facing charges. For these reasons, treatment of the sexually abusive young person needs to be undertaken within a close supervized, intensive, community-based foster placement with specially trained foster carers who are experienced in dealing with young offenders, risk and dangerousness. This type of approach is known by various names such as Multidimensional Treatment Foster Care[92] or forensic foster care.[93] Early results from small-scale studies with this type of intervention are reasonably encouraging.

Outcomes claimed for these approaches include significantly fewer subsequent criminal referrals and more incarcerated boys returning to live with relatives, compared with those who received group home care alone.[92] In a seven-year study of forensic foster care at the Treatment for Appropriate Social Control (TASC), Yokely and Boettner[93] describe a social responsibility model to teach recidivist youths 'pro-social skills and values that compete with antisocial behaviour'.

Dynamic psychotherapy aims to work at an unconscious level with the sexually abusive young person to explore and understand the reasons for his persistent behaviour. However, evidence-based treatment outcome studies have not yet been undertaken for dynamic therapy with juvenile sexual abusers. A clinical description of long-term dynamic therapy[94] with these children emphasizes the need to establish a systemic child protection context for the safe delivery of such treatment.

In summary, the components of effective treatment interventions with children and young people who sexually abuse will include the following:

- A well planned, systemic, child protection orientated, treatment context.
- Treatment should be one of a number of positive interventions into the life of the young person and his or her family.
- All interventions should be part of an agreed inter-agency care plan for the young person.

- Offence-specific interventions, such as CBT, aimed at straightening out the distorted cognitions and self-justifications of sexually abusing young people should be the core of any intervention programme for this client group.
- Treatment programmes not focussed solely on the victimization of the young person.
- Interventions should occur at all possible levels including individual work with young person, family work (where relevant), support for foster carers or for professional care staff and consultation to the professional network.

Firesetting/arson

Arson can have a devastating impact on the victim and the wider society. Juvenile arsonists are not a homogenous group, with a wide range of familial,[95] social,[96] developmental interpersonal,[97] clinical and 'legal' needs. Kolko and Kazdin[98] highlighted the importance of attraction to fire, heightened arousal, impulsivity and limited social competence. As with other forms of serious antisocial behaviours, no single standard treatment approach will be appropriate for all individuals.[99] In addition to the general assessment of antisocial behaviour the specific domains to be considered include:

- history of fireplay;
- history of hoax telephone calls;
- social context of firesetting (whether alone or with peers);
- where the fires were set;
- previous threats/targets;
- type of fire, single/multiple seats of fire setting;
- motivation (anger resolution, boredom, rejection, cry for help, thrill seeking, fire fighting, crime concealment, no motivation, curiosity, and peer pressure).

For recidivistic firesetters therapy may include:

- Psychotherapy to increase the understanding of the behaviour, including antecedents defining the problem behaviour, and establishing the behavioural reinforcers;
- Skills training—to promote adaptive coping mechanisms;
- Understanding environmental factors to manage or self trigger solutions;
- Counselling to reduce psychological distress;
- Behavioural techniques to extinguish the behaviour;
- Education to promote understanding of cause and effect; and
- Supervision for the staff caring for the adolescent.

Early modelling experiences and early exposure to related phenomena militate against a good outcome.

The role of specialist child and adolescent mental health services in medico–legal assessment

In the case of T&V v United Kingdom[100] it was stated that a child's age, level of maturity and intellectual and emotional capacities must be taken into account when they are charged with a criminal offence and that appropriate steps should be taken in order to promote their ability to understand and participate in the court proceedings. A responsibility therefore falls on the defence lawyer to be aware of the possibility that a young person may not be able to participate effectively in the trial process, particularly if they are under 14 years old or have learning problems, or a history of absence from school.[101] In 1985, the Office of the High Commissioner for Human Rights, in reference to the age of criminal responsibility stated that there is a close relationship between the notion of responsibility for delinquent or criminal behaviour and other social rights and responsibilities.

All young defendants, regardless of the offences they charged with, should be tried in youth courts with permission for adult sanctions for older youths if certain conditions are met. This should enable a mode of trial for young defendants to be subject to safeguards that can enhance understanding and participation. Assessment of cognitive and emotional capacities should occur before any decisions on venue and mode of trial take place.

Capacity

One fundamental distinction in the criminal law is between conditions that negate criminal liability and those that might mitigate the punishment deserved under particular circumstances. Very young children and the profoundly mentally ill may lack the minimum capacity necessary to justify punishment. Those exhibiting less profound impairments of the same kind may qualify for a lesser level of deserved punishment even though they may meet the minimum conditions for some punishment. Immaturity, like mental disorder, can serve both as an excuse and as mitigation in the determination of just punishment. Capacity is sometimes thought of as a generic skill that a person either has or lacks. However, that is not so. To begin with, it is multifaceted, with four key elements. These are as follows:

1 The capacity to understand information relevant to the specific decision at issue (understanding).
2 The capacity to appreciate one's situation as the defendant is confronted with a specific legal decision (appreciation).
3 The capacity to think rationally about alternative course of action (reasoning).
4 The capacity to express a choice among alternatives.[102]

The second key point is that capacity is a feature that is both situation-specific and open to influence.

Any evaluation of competence should include assessment of possibly relevant psychopathology, emotional understanding as well cognitive level, the child's experiences and appreciation of situations comparable to the one relevant to the crime and to the trial, and any particular features that may be pertinent in this individual and this set of circumstances.[103] The general principles to be used in the assessment are broadly comparable to those employed in any clinical evaluation. However, particular attention needs to be paid to developmental background, emotional and cognitive maturity, trauma, exposure and substance misuse. The likely appropriate sources for obtaining clinical data relevant to assessment of a juvenile's competence to stand trial will include a variety of historical records, a range of interviews and other observations and in some cases, specialized tests. Records of the child's school functioning,

past clinical assessment, treatment history and previous legal involvements need to be obtained. In coming to an overall formulation, there should be a particular focus on how both developmental and psychopathological features may be relevant to the forensic issues that have to be addressed.

The main focus is on the youth's ability to understand and cope with the legal process. This comes from three sources, direct questioning of the defendant, inferences from the functioning in other areas and direct observation of the defendant's behaviour and interaction with others. It is useful to enquire about the youth's expectations about what the consequences of the court involvement might prove to be. Because of the course of juvenile proceedings can vary so widely, with consequences ranging from the extremely aversive to extremely beneficial, rational understanding will necessarily involve a high degree of uncertainty. Potentially relevant problems include: inattention, depression, disorganization of thought processes that interfered with the ability to consider alternatives; hopelessness, such that the decision is felt not to matter, delusion or other fixed beliefs that distort understanding of options (or their likely outcomes), maturity of judgement and the developmental challenges of adolescence.

In providing information to the court, written reports have the advantage of a standard format that helps the consultant to be sure that s/he has considered all the relevant questions; it also provides a familiar structure for readers. In essence, for the sake of consistency and clarity, competence reports need to cover the following areas:

1 Identifying information and referral questions.

2 The description of the structure of the evaluation including sources and a notation of the confidentiality expectations.

3 The provision of clinical and forensic data.

Grisso[104] suggested that psychiatric assessment of competence in young people should include assessment of:

- understanding of the charges and the potential consequences
- understanding of the trial process
- capacity to communicate with their defence lawyer
- general ability to participate in the courtroom proceedings.

In court, a child's ability to give an account of events can be impaired by a number of factors, including poor physical health on the day of the trial, overwhelming anxiety or anger about giving evidence, or intimidation by the physical surroundings of the court. From a psychological perspective, however, the basic evidential capacity of the child defendant will depend on two main components:

- The child's mental state—this needs to be stable, therefore any disturbance that might interfere with the child's perception of the world and the ability to understand it will impair evidential capacity;
- The child's cognitive ability—a concept that includes a large number of facets, such as memory, understanding and the ability to communicate. The last includes both verbal (speech) and non-verbal means, as well as the ability both to comprehend and to express thought. Any psychological assessment therefore has to be across a range of domains.

Discrepancies are particularly likely in the areas of educational achievement, adaptive skills and social and emotional development. A child's ability often is gauged on educational achievement and given as being equivalent to that of a certain age—e.g. a 15-year-old child might have the everyday living skills of a 7-year-old. However, a child who might be unable to cope with monetary change or public transport might well have the emotional and social experiences of an older child and the drives of an adolescent.

When discussing developmental psychology and child development, it is important to bear in mind that none of these processes operates in a vacuum. The child's experience of parenting (important in relation to physical and emotional development), the provision of appropriate role models (moral development and self-control depend heavily on appropriate modelling and social learning) and the learning environment (whether it fostered or hindered intellectual development) all have a vital role. For instance, during adolescence, as young people take on a wider and more social perspective and become integrated within a peer group, they will nevertheless tend to adopt social values and norms (i.e. ideas about 'right and wrong') that are very similar to those of their parents. Hence, despite any demonstrations of teenage rebellion (often short-lived), the majority of adolescents will tend to adopt parental mores, either law-abiding or delinquent.

It should be emphasized that clear-cut ages do not apply to the completion of physical, intellectual, emotional, and social development. For most young people, given appropriate parenting, normal biological development and a structured, emotionally supportive and stimulating environment, the bulk of the aforementioned processes should be achieved by late teenage years and a considerable degree of intellectual maturation may have occurred by the age of 14 years.

When delivering forensic mental health services for children and adolescents it is important that the services are developed in such a way that their needs are met and that the services build on established concepts of service design in line with a strategic framework. Doing so will require long term planning that actively addresses the requirements of an adequate size and composition of an appropriately trained, supervized and managed workforce. Such services should be developed with an awareness of the scope of existing services and recognition of current demands, analysing gaps current services.

Adolescent girls

Longitudinal data demonstrates that girlhood aggression contributes to a cascading set of negative outcomes as young women move into adolescence and adulthood.

Young girls who engage in disruptive behaviour and fight are at risk for:

- Being rejected by peers
- Feeling alienated
- Feeling unsupported in their relationships with peers and adults
- Struggling academically
- Affiliating with other peers prone to deviant behaviour
- Becoming involved in more serious antisocial behaviours
- Choosing antisocial romantic partners
- Initiating and receiving partner violence

- Becoming adolescent mothers
- Having children with more health problems
- Being less sensitive and responsive as parents

Some are sufficiently antisocial and even violent, they are incarcerated, if they are also mothers they may lose custody of their children and opportunities for stable employment and relationships are much diminished.

Given low base rates of girls engaging in physical aggression and violence, identifying girls at risk is a critically important step for prevention and intervention programmes.

High-risk groups of girls to target include those girls who are temperamentally overactive as toddlers and pre-schoolers (fewer than boys) but even more important to target for early identification and intervention. Those who have early pubertal development (girls report engaging high levels of bullying as they enter puberty). They may be likely targets as well as perpetrators and sexually abused girls—especially those abused by their biological fathers over a long period of time.

From the available literature interventions to reduce rates of aggression, relational aggression and violence in female children and adolescents should address the following:

1 Pre-natally delivery of programmes for high-risk expectant mothers, (especially young mothers and those themselves aggressive or disruptive as children).

2 Augment the parenting skills of at risk young mothers—the evidence show children of young mothers with histories of girlhood aggression may themselves be more prone to infection and injuries.
   (a) Provide additional parenting skills around key issues of hygiene, child proofing of homes, good nutrition, meal planning and household management.
   (b) Help young mothers to respond optimally to the perceived challenging behaviour of their infants and toddlers.

3 Middle childhood, girls episodes of physical aggression are often preceded by relational aggression. Interventions to help these girls may include how to:
   - Relate to others
   - Manage strong emotions
   - Understand own aggressive feelings and recognize when own aggression is adaptive in the immediate situation.
   - Understand how 'girl talk' ignites hurtful indirect social relational aggression.
   - Understand how relational aggression lead to physical violence for some girls towards peers, adults and partners

Conclusions

The major challenge of altering the trajectories of persistent young offenders has to be met in the context of satisfying public demands for retribution, together with welfare and civil liberties considerations. In England and Wales for example we lock up more than 3 000 juveniles at anyone time (age of criminal responsibility set at 10).

Treatment of delinquents in institutional settings has to meet the sometimes contradictory need to control young people, to remove their liberty and to maintain good order in the institution, at the same time as offering education and training to foster future prosocial participation in society and meeting their welfare needs. At least in England and Wales, the legislative overhaul of Youth Justice[105] has mandated practitioners to bridge the gap between residential and community treatments and to involve families using Youth Offending Teams (YOT's) to meet this complex mix of needs, but the public demand to remove antisocial youths from the street has led to the implementation of Antisocial Behaviour Orders including children with learning disabilities.

Over the last 30 years there has been a gradual shift in opinion regarding effectiveness of intervention with delinquents, from the 'nothing works' approach to a 'what works' approach. The pressure from politicians and public will remain, for a quick fix solution to problems that span cultures, countries and generations.

Provision of appropriately designed programmes can significantly reduce recidivism amongst persistent offenders. The mode and style of delivery is important; high quality staff and staff training are required. Community based programmes seem better than institution based programmes. In prison settings, the strongest effects are obtained when programmes are integrated into the institutional regimes.

What are the key levers for change?

- Developing an integrated comprehensive screening and assessment tool for mental health, substance misuse and physical health
- Better identification of mental health needs by courts with effective court diversion.
- Improving access to child and adolescent mental health services for 16 to 17 year olds across the board.
- Enhancing the role of health workers in YOT's.
- Integrating work of substance misuse workers in custody and YOT's
- Enhancing intensive resettlement and aftercare provision—RAP Schemes.
- Reviewing the demand and need of nationally commissioned adolescent psychiatric secure inpatient beds (88 by 2008).
- And ensuring all working with young offenders understand and are trained in normal and abnormal child and adolescent development together with awareness of the nature of mental health problems in this stage of the life course and how this impacts on all aspects of a young person's life.

Our knowledge of true prevalence rates of mental disorders in a young offending population is developing further. Child and adolescent mental health practitioners have the skills to set the understanding of delinquency in a developmental context and treat those young offenders with mental disorders. Knowledge is advancing rapidly by an established international research network in Europe and the USA.

Further information

Moffitt, T.E. and Scott, S. Conduct Disorders of Childhood and Adolescent. In *Rutter's Child and Adolescent Psychiatry* (5th edn) (eds. M. Rutter, D. Bishop, D. Pine, S. Scott, J. Stevenson, e. Taylor and A. thapar), Chapter 35, pp. 543–54.

Gives a comprehensive review of Conduct disorder classification, prevalence, subtypes, associated co-occurring dsorders and complicating conditions, tackling risk indicators, diagnostic challenges

and evidence based interventions as such it offers a good context in which to set the current chapter.

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11.8

Child molesters and other sex offenders

Stephen Hucker

Introduction

In most Western societies sexual offenders are more reviled than almost any other type of offender. On both sides of the Atlantic this is reflected in the sanctions that specifically address this group such as Sexually Violent Predator laws in the United States, Dangerous and Long-Term Offender legislation in Canada, and Sex Offender Orders in the UK. Related approaches include the introduction of sex offender registries and the widespread requirement that children at risk from sexual predators be reported by professionals and others.

The general psychiatrist and the sex offender

Though constituting a relatively small proportion of all reported offences, sex offending affects large numbers of people in the general population. Though there are methodological difficulties associated with much of the research in this area, the World Health Organization has reviewed estimates of childhood sexual abuse from 39 countries and found that the prevalence of non-contact, contact, and intercourse in female children was about 6, 11, and 4 per cent, respectively; the corresponding figure for males was about 2 per cent in all categories.[1] However, these must represent minimal figures as it is known that many victims do not report their experience and individual sex offenders, when guaranteed confidentiality, will admit to many more offences than they were charged with or convicted of.[2] Various forms of sexual offending against adults are also underreported but it has been estimated that about 13 per cent of women and 3 per cent of men have been raped at some time during their lifetime.[3]

The long-term effects of such victimization has been extensively studied and it is clear that people with a history of child sexual abuse, for example, experience a wide range of long-term psychological consequences.[4]

Although prone to find reasons to delegate the assessment and management of sex offenders to specialized forensic services, the general psychiatrist will find it impossible to avoid them entirely. Minor varieties, such as 'flashers' (exhibitionists) or 'peeping Toms' (voyeurs), may be viewed by the courts as less serious, and the opinion of the generalist will still be appreciated, especially when specialist resources are scarce and an appointment for a forensic assessment could be long-delayed. It is important, therefore, for the general psychiatrist to have some understanding of this area in order to make appropriate decisions and recommendations.

Definitions of sexual offending

Put simply, a sexual offender is an individual whose sexual behaviour contravenes the law in a particular jurisdiction. The types of activities that may be proscribed vary considerably. Western countries are generally more tolerant though most societies provide sanctions for sexual activity involving children below the age of consent, non-consensual sexual acts, sexual relations with close family, and sexual interference with animals or corpses. There are also typically legal and other interventions where a person fears sexual harassment or assault, and where there has been abuse, or likelihood of abuse, in certain professional relationships. Typically, also, there is regulation of pornography or obscene material.

Relationship between sexual deviancy and sexual offending

There is some overlap of sexual offences with a medical diagnosis of a paraphilia. However, this is not a complete concurrence. Thus, not all paedophiles have molested a child and not all child molesters are paedophiles; many, perhaps most, men who sexually assault adult women are not sexually deviant or paraphilic at all. The psychiatric categories of paraphilia and their characteristics are described elsewhere in this volume (Chapter 4.11.3 by Fedoroff).

Types of sexual offender

The vast majority of sexual offenders are male though it is recognized that women may also commit similar crimes.[5] Male sexual offenders can be broadly divided into: child molesters, rapists, and non-contact sex offenders.

Child molesters

Typologies of this subgroup in part refer to the degree of paraphilic attraction (sexual deviancy). Thus, there has been a common categorization into 'fixated' and 'situational' or 'regressed' types.[6] With the 'fixated' type there is a permanent attraction to children typically dating from adolescence thus conforming to definitions of paedophilia in DSM-IV and ICD-10. Those attracted to males are more likely to repeat their offences with recidivism at least

twice as high as with those attracted to girls. The former tend to victimize boys aged 11–15 years old, whereas the latter molest girls of 8 to 10.

'Fixated' paedophiles tend to commit premeditated offences that often involve considerable planning. Manipulation and 'grooming' behaviour is used as a means of luring, even abducting, children into sexual activity, and they may gain the trust of the parents or other carers. They may appear to have an excellent rapport with the child victims and treat them kindly but their motive is primarily for the child to meet their own need for affection rather than the reverse. It is for this reason that 'needy' children are often selected as victims. Such offenders will typically profess their 'love' for children and convince themselves that their behaviour is not harmful. Other rationalizations, such as that were educating the child or introducing the child to sexual love in a caring way, are common.

'Regressed' or 'situational' child molesters are, according to this typology, attracted primarily to adult females and may be in a marital-type relationship at the time of their offence. They will often report feelings of personal inadequacy or low self-esteem and their offences are more typically spontaneous and occur in the context of a stressful life circumstance.

The 'regressed' child molester, in contrast to the 'fixated' type, is less inclined to 'groom' victims and their caregivers. Victims of this type may be older than those involved with the 'fixated' molester. Molestations however may begin before, and continue past, puberty.

Rapists

Once again, the typology expounded by Prentky, Knight, and colleagues can be useful.[6] In this scheme, offenders are differentiated through their apparent motivation by anger, power, or sadism. Most rapists act alone but individuals involved in 'gang' rape may represent several different types.

'Anger-motivated' rapists act out deviant fantasies of retaliation towards the victim, using violence as a means of expressing generalized anger, typically towards women though sometimes towards people in general. The intention of the attack is to humiliate and debase the victim who will typically have been selected randomly.

The 'power-motivated' rapist type has been further subdivided into the 'power-reassurance' and 'power-assertive' types. The former is plagued by doubts and insecurities about their own masculinity and sexual adequacy, often uses minimal force and may apologize to the victim or even seek a relationship afterwards. However, these victims may have been stalked and the attacks on them premeditated. The 'power-assertive' subtype is motivated by the desire to dominate women and has no doubts about his masculinity but, like the previous subtype, typically does not use gratuitous violence to subdue the victim.

'Sadistic' rapists, are the least common but perhaps most worrisome type, who derive sexual pleasure from inflicting hurt and suffering on their victim. Sometimes, however, this is difficult to differentiate from other types where pain, suffering and humiliation are the consequences, but not the primary motivation, for the attack.

Other types of sexual assault that do not involve penetration, and which represent related behaviours, include toucheurism and frotteurism. These involve, respectively, touching or grabbing strangers, typically females, in a way that provides him with sexual gratification. The former, in particular, may pass unnoticed by the victim, as attackers will typically touch or grab sexual areas such as breasts, buttocks, or crotch, in crowds and similar situations where the incident may be discounted as 'accidental'.

Non-contact sexual offences

This group includes 'peeping Toms', 'flashers', and indecent phone callers with corresponding psychiatric diagnoses of the paraphilias voyeurism, exhibitionism, and telephone scatalogia, respectively.

Peepers have a penchant to observe an unsuspecting female stranger undressing, or couples in the act of copulation. They may masturbate at the scene or later in private while recalling what they saw. Most voyeurs, like most sex offenders who are paraphilic, are aware of their deviant impulses while still adolescent but the behaviour may become chronic.

Exhibitionists derive sexual excitement from exposing their genitals to unsuspecting female strangers. The desired reaction is one of 'shock and awe' and indifference is a useful response, if the victim has the presence of mind. The perpetrator may masturbate at the time or later in private. Though some cases are particularly intractable, it is unusual to see an exhibitionist still active much past the age of about 40 years old.

The unsuspecting victims of obscene telephone callers are greeted with a barrage of sexually explicit commentary over the telephone. When this is a habitual practice, accompanied by or followed by masturbation, it is distinguishable from an isolated incident committed as a prank.

In all three of the above non-contact sexual offences, there is typically no desire to have further contact with the victim and, indeed, such a prospect often fills the offender with anxiety. However, a small minority of these offenders may later commit a more serious sexual offence, even sexually motivated homicide.[7] Atypical features, such as a desire to have personal contact with the victim, or the repeated selection of child victims, may be an early warning sign of such potential and warrants further, expert assessment.

In recent years, paralleling the advance of technology, offenders have been apprehended for using the Internet as a means of obtaining or distributing pornography (especially child pornography) or as a means of contacting children for sexual purposes. Possession of child pornography is an indicator of sexual interest in children and it is associated with self-reported sexual interest in children and with laboratory measurements of changes in penile volume or circumference.[8]

Assessment of sex offenders

In the context of court remanded cases, the usual pre-trial issues of fitness (competence) to stand trial and criminal responsibility will need to be addressed. Only a small proportion of all sex offenders demonstrate symptoms of psychotic mental disorder, particularly schizophrenia and depressive disorders. However, where those disorders are present it is necessary to explore how the symptoms, e.g. delusions or hallucinations, bear specifically on the sexual offending. It may be found that other psychiatric disorders are of greater importance and that a secondary diagnosis of mental retardation or personality disorder, or more particularly, a paraphilia, are more relevant with respect to the offending behaviour. More commonly the court will be interested in sentencing considerations, specifically the risk the offender presents to others, and whether and what medical treatment and/or other professional

interventions might be ordered or recommended by the court in order to reduce that risk.

Interview

The interviewer must assume that the subject will be at best guarded, or frankly hostile, in their response to the assessment process and it may be difficult to achieve the level of affinity more commonly experienced with non-psychotic general psychiatric patients. A non-judgemental approach is therefore preferable, regardless of the examiner's personal emotional reaction to the offender's behaviour. Few will have presented to the psychiatrist or psychologist voluntarily. Moreover, there is a distinct tendency among sex offenders to prevaricate or frankly lie. It is therefore essential for the examiner to have detailed information about the act or acts that are alleged to have occurred. It has already been noted that denials, rationalizations, distortions, and minimization are the norm with sex offenders. It is not, however, the assessor's function to judge the issue, in particular when guilt has not been determined by the court.

It is obviously unhelpful, in terms of gaining rapport or obtaining additional information, to accuse the subject of dishonesty. Rather, a sympathetic approach suggesting that sometimes people have difficulty accepting unpleasant aspects of themselves, may be more helpful. Another approach is to invite the subject to explore why the victim made the accusations if they are untrue and whether they can accept, if not the whole, then some part of the allegations against them.

It is also important to remember that, while the individual may have been accused of one type of deviant sexual behaviour, other types may have occurred and been undetected. Paraphilic disorders tend not to occur as it were in pure culture but rather in association with other paraphilias, typically at least two or three. Thus, for example, an exhibitionist may have been reported for exposing specifically to children rather than adults and this will suggest an additional diagnosis of paedophilia. Or, an individual who has been convicted of rubbing himself against women in public places may also have made obscene phone calls and harbour fantasies of rape.

In the case of child molesters it is important to consider how he gained access to his victims. Exploration of the methods of 'grooming' is important in understanding ways to assist the offender to avoid risky situations in the future.

In terms of the overall assessment, identification of psychopathologies outside the domain of sexual deviation is important. The presence of psychosis will have an implication for the type of treatment to be recommended, even if it is not common among sex offenders. It seems likely that some such sex offenders' behaviour is dismissed as a function of the psychosis and underlying paraphilic disorder may easily be discounted or not considered at all. More commonly it will be personality disorders or traits, alcohol or substance abuse, mild-to-moderate depression, and anxiety disorders, rather than major mental illness that will be noted. Attention to these will be an important part of any subsequent treatment or management strategy.

Psychometric testing

Psychometric testing may contribute additional information an overall assessment, in particular where the subject is not forthcoming in a personal interview. There are a number of general personality assessment instruments available. These include the well known and widely used *Minnessota Multiphasic Personality Inventory* (*MMPI*), the *Millon Clinical Multiaxial Inventory* (*MCMI III*), and the *Personality Assessment Inventory* (*PAI*). All have been extensively used in offender populations including sex offenders, and common profiles have been identified. Although none can specifically identify a sex offender, information concerning impulsivity, denial, judgement, and general psychopathology may be very useful.

There are also a number of psychological tests that have been specifically designed for the assessment of sex offenders. These include the *Multiphasic Sex Inventory II* (*MSI-II*) and the revised version of the *Clarke Sex History Questionnaire for Males* (*SHQ-R*). The *MSI-II* is designed to measure the sexual characteristics of an adult male (though there is a female version too) alleged to have committed a sexual offence or sexual misconduct, including those who deny the allegations. Though standardized in the United States on a large sample of sex offenders it is more widely used. It consists of 560 true/false questions and the completed questionnaire must be sent to the developers for computerized scoring and interpretation. The SHQ-R consists of 508 questions and the completed questionnaire may again be sent away for scoring and an interpretive report returned.

Laboratory testing

As sex offenders are prone to lie and distort their self-report of deviant interest and behaviours a more objective method of assessment has long been pursued. One of the earliest to be developed was the use of the penile plethysmograph (PPG, or phallometry) to measure changes in response to erotic stimulation. This method involves measuring changes in the size of the penis while presenting the subject with carefully selected images, both still and moving, of both sexes and different age groups, and audiotaped descriptions of various sexual activities. There are certainly problems with PPG testing, including the standardization of stimulus materials used, and some offenders are either able to learn to suppress their physiological responses or masturbate before the testing in order to render themselves unresponsive.[8] Nonetheless, the PPG, more commonly using a circumferential device, or the volumetric method, is extensively used in assessments of sex offenders (for critical review, see Ref.[10]).

Particularly in the United States, the PPG has come under attack as, in addition to standardization and reliability issues, it uses pictures of children whose consent or that of their parents was never obtained. The computer-generated images have been developed to attempt to obviate this concern. More recently the use of virtual reality of computer-generated images has also been used experimentally. Other, less intrusive methods of assessment are also being adopted, including the Abel Screen[11] which measures time spent viewing non-nude images.

Mention must also be made of polygraphy with sex offenders as it has been used extensively in many parts of the United States and to some extent in the United Kingdom. The subject is asked questions relating to their sexual interests and activities while their pulse, respiration, and skin conductance are measured. However, research in the area is generally weak and, despite its widespread use and perception of usefulness, particularly for monitoring sex offenders in the community, the method is controversial (for review, see Ref.[8]).

Assessment of risk in sex offenders

The courts will often wish to have a professional opinion regarding the risk an offender presents to re-offend, and it is important to have an understanding of the factors that will contribute to this (see Table 11.8.1).

Risk has been divided into:

1 Static risk, i.e. involving those factors which cannot change, such as the offender's age, sex, or number of previous criminal convictions and

2 Dynamic risk, i.e. involving those which potentially could change, either as a result of treatment or some other intervention, or simply by the passage of time. This can be further subdivided into relatively stable, though nonetheless potentially changeable, factors such as sexual preferences or negative attitudes, and acute factors, such as access to victims, reversion to substance use, and active mental illness.

These are important in estimating an offender's risk to re-offend. Static factors have received the most scientific study and it is chiefly that these have been incorporated into various actuarial or statistical instruments that have been developed in the past several years, based on follow-up studies of samples of sex offenders. Among these instruments the Rapid Risk Assessment of Sex Offender Recidivism (RRASOR), the STATIC-99,[12] and Sex Offender Risk Appraisal Guide (SORAG),[13] are the most widely used. An assessor intending to use any of these instruments needs to be thoroughly familiar with the literature on the topic and to have participated in training workshops that are given at conferences from time to time. Useful though these tools can be, too heavy reliance upon them is no substitute for a full understanding of how these were constructed and their limitations.[14]

Table 11.8.1 Predictors of sexual offence recidivism

Type of risk factor	Predictor
Static	Prior sex offence Prior non-sexual offences Prior non-contact sex offences Prior treatment dropout Any boy victims Any unrelated victims Any stranger victims Early age of onset Young age of offender Minimal cohabitation history Childhood behaviour problems Separation from parents as a child Antisocial personality disorder Prior violation of conditional release
Stable dynamic	Sexual preferences, children Sexual preferences rape/violence Sexually entitled attitudes Pro-child molester attitudes Pro-rape attitudes Lack of adult love partner Emotional loneliness Lifestyle impulsivity Ineffective problem-solving skills Callous and unemotional attitudes Aggressive, hostile, and suspicious Negative social influences
Acute dynamic	Access to potential victims Substance abuse Sexual preoccupation Emotional collapse Collapse of social support system Rejection of supervision Acute mental illness

(Reproduced from Webster, C.D. and Huxler, S.J. (2007) Violence risk: assessment and management. Copyright 2007, John Wiley & Sons, Inc.)

Treatment issues

It is rare for paraphilic individuals to present for treatment in order to prevent themselves from becoming a sex offender. Though some paraphilias are not usually associated with criminal charges (e.g. transvestitic fetishism) others, such as paedophilia, are more likely to present through the courts or probation and parole services *after* an offence has been committed. It is important to realize that sexual preferences are highly resistant if not impossible to change. The most that can be expected with sex offenders who have deviant sexual preferences (paraphilias) is to help them learn to control their behaviour and to recognize that their propensity will always remain in the background, much as alcoholics are advised to consider themselves always vulnerable to relapse.

Psychological treatments

Various psychological therapies have been attempted with sex offenders. Psychodynamically based individual and group treatments have been the most commonly used. It has become clearer more recently, however, that cognitive-based therapies (CBT) are the preferable approach to take, although techniques involving classical behaviour therapy, e.g. covert sensitization, are also sometimes used for specific purposes, such as creating aversion to deviant arousing images and replacement with non-deviant ones. Cognitive behaviour therapy itself involves helping the subject to develop strategies to alter their thought processes in order to avert their deviant behaviour, to improve their social skills, and to remedy their distorted beliefs and attitudes.

There is little or no evidence for the efficacy of psychological treatments prior to the introduction of CBT. Based on meta-analysis of 43 published studies, it has been shown that treatment programmes using this approach are associated with a reduction in overall recidivism rate from about 17 to 10 per cent.[15] Nonetheless, there is still considerable controversy over the effectiveness of formal sex offender treatment programmes.[16]

Medications

Various medications have been used to treat sex offenders. Based on empirical observations of animals, which become less sexually active following neutering, hormonal treatments that reduce testosterone levels have been extensively employed. All require careful discussion with the potential patient concerning side effects and it is important to obtain written, informed consent.

Oestrogens proved to be problematic because of the serious risk of thrombo-embolic complications and a safer alternative was found in Cyproterone acetate (Androcur), an anti-androgen, which is available in Europe, including the UK. Because this drug has not

been made available in North America, Medroxyprogesterone acetate (Provera) was introduced. Both however can still be responsible for minor side effects such as weight gain, tiredness, and gynaecomastia (with Cyperoterone, especially) but also more serious problems including thrombo-embolism and increase in blood sugar. More recently, leuteinizing hormone-releasing hormone (LHRH) agonists such as Leuprolide acetate (Lupron), Goserelin (Zoladex), and others, have been found useful as they produce almost total suppression of testosterone production such as would be seen following surgical castration. They tend, however, to be used mainly in very high-risk offenders or those who have failed with other drugs.[17] All of these hormone-affecting substances, though especially the LHRH agonists, have a tendency to leach calcium from the bones[18] and it is necessary to monitor carefully for this side effect and to administer antidotes including calcium supplements, vitamin D, and possibly biphosphonates.

The main problem with the hormonal treatments is their lack of acceptance by those who might potentially benefit. An alternative in the form of serotonin-reuptake inhibitors has therefore been better received though double blind trials are still lacking. They depress libido in about 50–60 per cent of cases though higher doses, such as are used with obsessive–compulsive disorder, are often necessary. There appears to be little basis on which to chose one SSRI over another, other than patient tolerance of side effects.[19]

Ethical problems

Several ethical issues have been mentioned above in passing. It is, however, worth emphasizing in conclusion that a disinterested professional demeanour is important when assessing sex offenders. No matter what his or her own private views, it is not the place of the clinician to decide on guilt or innocence or in any other way to pass judgement on the offender or alleged offender. Moreover, alienating the offender will present a further impediment to gaining information and to providing treatment when indicated and necessary.

It is important, at the assessment stage, to identify for the subject the nature of the evaluation, the role of the assessor, and the person or agency for whom they are acting, for example a Child Protection Service, a defence lawyer, or Crown prosecutor. This may determine the degree of cooperation but to not explain this fully and simply to present oneself as a 'doctor' in a helping role when the intention of the evaluation is solely to provide a risk assessment as opposed to treatment, for example would be unethical.

Certain assessment procedures, such as penile plethysmography and polygraphy (when used in a clinical setting) are particularly contentious. Though both may provide useful information, written and fully informed consent should always be obtained beforehand. PPG has been particularly criticized for the use of child images as sexual stimuli when the consent of neither the child nor its parents have been obtained, though the development of computer-generated images may avoid that particular objection.

Finally, when drugs that have been developed and marketed for other purposes are used to suppress sexual drive, the patient needs to be fully informed of the potential benefits as well as the risks involved but should not be denied complementary or alternative treatments should they decide not to expose themselves to the potential side effects.

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11.9

Arson (fire-raising)

Herschel Prins

How great a matter a little fire kindleth!

New Testament, authorised version (Letter of James, 3:5)

The title of this chapter merits brief comment. Why is arson (fire-raising) a special problem, and why is there a dual title? Arson is a special problem because not only is it regarded as a very serious form of criminal behaviour, but because its detection can be very difficult. It is an offence that can be committed at 'one removed' by an offender, and it may sometimes involve unintended victims. Forensic psychiatrists will meet a number of arsonists in the course of their work but, increasingly, general psychiatrists are likely to come across them from time to time, for two reasons. *First*, the High Court in England and Wales has suggested that psychiatric reports are advisable in all cases where the motivation for the offence is unclear. *Second*, as will become clear from the writer's later comments, there has been a worrying increase in arson committed by young adults and children. Thus it is probable that child and adolescent psychiatrists are likely to come across cases more frequently. However, Soothill indicates a word of caution. He suggests that over-reliance upon psychiatric involvement may tend to 'medicalize' socially problematic behaviour.[1] The dual title that heads this chapter indicates the legal term used in the United Kingdom to describe acts of unlawful fire-raising. However, the term is not in universal use; readers will find that in other jurisdictions (notably in the North Americas) the terms are fire-raising, fire-setting, incendiarism, and in certain specific instances, pyromania and pathological fire-raising.[2] The term fire-raising is used in this chapter since the alternative terminologies can be subsumed under it.[3,4]

Brief historical context

The phenomenon of fire, its uses and misuses, has figured extensively in myth, legend, and literature. For example, Prometheus is said to have stolen fire from the Gods and the myth became the mainspring for much psychoanalytic theorizing about fire-raising behaviour.[3] There are numerous early historical references to incendiary mixtures and devices, including sketches for mortars by Leonardo da Vinci.[5] In the mid-nineteenth century, the medical profession became interested in the explanation of fire-raising behaviours; and subsequently, adherents of psychoanalysis proposed various complex and somewhat doubtful explanations for such conduct. In particular, they linked fire-raising behaviour to sexual disturbance of one kind or another. Although sexual problems do appear in the backgrounds of *some* recidivist fire-raisers, the importance of the links has, in the present writer's view, been somewhat overstated.[6] Having said this, it should perhaps be noted that the phenomenon of fire is not infrequently linked linguistically to aggression and sexuality. For example, we speak or write of 'white hot rage', 'heated arguments', 'inflamed passions', to have the 'hots' for a sexual partner. Language is the conveyor of cultural values and attitudes and can be a powerful force in influencing our modes of thinking and expression about the phenomenon of fire in its many manifestations.

The size of the problem

During the past two decades concern about the increase in fire-raising has been expressed worldwide. Table 11.9.1 gives the number of offenders convicted of arson in *England and Wales* for the years 1999–2003.

These figures only provide a partial picture, since it is often very difficult to establish whether a fire has been started deliberately (fires of doubtful origin or, if strongly suspected, malicious ignition). Happily, recent advances in forensic science have brought about improvements in the detection rate. A more reliable picture of the real size of the problem can be obtained from the United Kingdom *Fire Statistics*. The latest figures indicate a continuing worrying number of deliberate fires—some 91 200 in 2004 and, in particular, the number of attacks on vehicles and schools. 'Arson in vehicles . . . accounts for 60% of all deliberately set fires at 55,000 per year'. In 2004, in the United Kingdom, there were 840 school fires, slightly down on 896 in the previous year. Fatalities dropped from 117 in 2003 to 88 in 2004. Overall, the number of deliberate fires decreased by 21 per cent in 2004,[8] an encouraging trend. The cost of fires in

Table 11.9.1 Arson, number of offenders found guilty of arson in England and Wales for the period 1999–2003

1999	2000	2001	2002	2003
2475	2470	2644	2427	2501

(Extracted from Home Office, (2004). Criminal statistics, England and Wales, 2003, Cm 6361. TSO © Crown Copyright[7].)

purely monetary terms is considerable; for example, figures from the Association of British Insurers (ABI) indicate 'that the cost of commercial fire claims in 2005 was 791 million pounds'—a record.[9]

Legal aspects

Legal definitions of arson vary from country to country. In England and Wales, prior to 1971, arson was an offence at common law. Currently, it is dealt with under the Criminal Damage Act, 1971. Similar provisions apply in Northern Ireland. In Scotland, it is dealt with under various common law offences. Section 1 of the 1971 Act states:

1 A person who without lawful excuse destroys or damages any property belonging to another intending to destroy or damage such property or being reckless as to whether any such property would be destroyed or damaged shall be guilty of an offence.

2 A person who without lawful excuse destroys or damages any property, whether belonging to himself or another:

(a) intending to destroy or damage any property or being reckless as to whether any such property would be destroyed or damaged; and

(b) intending by the destruction or damage to endanger the life of another or being reckless as to whether the life of another would be thereby endangered; shall be guilty of an offence.

3 An offence committed under this section by destroying or damaging property by fire *shall be charged as arson* (emphasis added).

NOTE: Recklessness has recently been clarified by the High Court as follows: 'A person also acts recklessly within the meaning of Section 1 of the 1971 Act with respect to (i) a circumstance where he is aware of a risk that exists or will exist; (ii) a result when he is aware of a risk that will occur; and, it is in the circumstances known to him, unreasonable to take the risk'.[10]

The seriousness with which arson and endangering life is recorded is reflected in Section 4 of the Act where both are punishable by maximum penalties of life imprisonment.

Classification, motivation, and management

Classification

An early, large-scale attempt to classify fire-raisers was undertaken by Lewis and Yarnell.[11] For an account of this and other earlier studies see Prins.[12] Faulk proposed two useful broad groupings. *Group I* consisted of those cases in which the fire served as a means to an end (for example, revenge, fraud, or a plea for help); *Group II* consisted of those cases where the fire itself was the phenomenon of interest.[13] Some years ago the present author, together with two psychiatrist colleagues, examined the files of a group of 113 imprisoned arsonists being considered for parole.[14] From this small (and admittedly highly selective) sample a rudimentary classification was devised. (See Table 11.9.2). This has been used by others as starting points for their own and perhaps more sophisticated classifications. The present writer has more recently modified slightly this earlier classification.[3] Despite modification it can be seen to still have certain weaknesses since it collates the behavioural characteristics of fire-raisers, various types of fire-setters and their motivations.[15]

Table 11.9.2 Suggested classification of the motives of arsonists (fire-raisers)

(a) Arson committed for financial reward (insurance fraud, etc)
(b) Arson committed to conceal another crime (for example, burglary or homicide)
(c) Arson committed for political purposes (terrorist and associated activities)
(d) Self-immolation as a political gesture. (Not arson as such, but included here for completeness, see Prins[12])
(e) Arson committed for mixed motives (for example, during the phase of minor depression, as a cry for help, or as a result of abuse of alcohol or other drugs)
(f) Arson due to the presence of formal mental disorder (for example, severe affective disorder, schizophrenic illnesses, organic mental disorder, mental impairment (learning disability))
(g) Arson due to motives of revenge—against (i) an individual or individuals; (ii) against society or others more generally
(h) Arson committed as an attention-seeking act (but excluding motives set out under (e) above) and arson committed as a means of deriving sexual satisfaction and/or excitement (for example, some forms of pyromania)
(i) Vandalistic arson (by young adults and children)

Rix broadened our original classification to include attempts to gain rehousing, carelessness, 'anti-depressant' (to relieve depressed feelings), and 'proxy' (in which the offender had acted on behalf of another who had borne a grudge).[16] Barker, an experienced forensic psychiatrist, in a wide-ranging and meticulous study of the psychiatric aspects of fire-raising, suggested that future classifications need to be more sharply focussed, emphasizing that arson should be seen 'merely as a symptom' to be viewed in the context of the whole person, not only to delineate different 'syndromes' of arsonists but also to identify individual points of therapeutic intervention and future dangerousness.[15]

Recent work by Canter and Fritzon has carried this focus forward. They suggest four themes to arson. Two related to *expressive* acts; (a) those that are realized within the arsonist's own feelings, being analogous to suicide, and (b) those that are acted on objects, like the burning of symbolic buildings. The two others relate to *instrumental* acts; (c) those that are for personal indulgence, similar to personal revenge; and (d) those that have an object focus such as hiding evidence from a crime (emphasis added).[17] More recently Canter and Fritzon's work has been replicated successfully by Almond *et al.*[18] and Hakkanen *et al.*[19]

(a) Some general characteristics of fire-raisers

It is a reasonable generalization to state that fire-raisers appear to be mostly young adult males who have exhibited behavioural difficulties from an early age (see Kennedy *et al.*[20] for a systematic review of the literature on this aspect and Repo and Virkkunen[21]). A significant proportion of these youthful fire-raisers have problems of alcohol abuse and intelligence levels lower than average.[21,22] Females who commit repeated acts of fire-raising and show some degree of mental disorder and self-mutilating behaviours are more likely to be awarded a mental health disposal by the courts than their male counterparts (see Coid *et al.*[23]and Noblett and Nelson[24]).

Motivation

To conform to the requested word limit for this chapter, categories (a), (b), (c), and (d) in Table 11.9.2 are not considered here. Detailed discussion of these and illustrative case vignettes may be found in Prins.[12]

(a) Fire-raising committed for mixed and unclear reasons

These are cases in which it is difficult to ascribe a single specific motive and which cause significant problems in assessment. They are likely to include the presence of a degree of mild (reactive) depression which may lead the fire-raiser to direct anger at a spouse or partner; thus revenge may also play a part (see discussion below). This group may also include cases in which the fire-raising may be a disguised plea for help, or a reaction to sudden separation or bereavement; in a proportion of these cases alcohol appears to play a part.

(b) Fire-raising due to serious mental disorder

Functional psychoses, notably the schizophrenias, may play a part in some acts of fire-raising. Such offenders will most likely be detained in secure hospitals or units. Manic depressive psychosis features occasionally, a classic case being that of Jonathan Martin, the nineteenth century arsonist who set fire to York Minster.[12]

(c) Fire-raising associated with 'organic' disorders

Occasionally, brain tumours, injury, epilepsy, dementia or metabolic disturbance may play a part. For example, although the epilepsies are not commonly associated with fire-raising (or other serious crimes for that matter) one should always be on the lookout for the case in which the crime has been committed when the person appeared not to be in a state of clear consciousness or when onlookers were present. Examples of organic states and their relationship to fire-raising are provided elsewhere.[3,25–27] The relationship between learning disabilities and fire-raising is discussed by Prins[12] and Clare *et al.*[28]

(d) Fire-raising motivated by revenge

Those incidents motivated by revenge are potentially the most dangerous. Such offenders are like the monster in Mary Shelley's *Frankenstein* who said 'I am malicious because I am miserable'. These are the fire-raisers who have serious problems with their feelings of anger and frustration caused by real or imagined wrongs. In considering the links between motives of revenge, it is important to stress the hazards of trying to place motivations for fire-raising in discrete categories; the vengeful fire-raiser may show clear signs of identifiable mental illness (for example, delusional jealousy), may be learning disabled and/or physically impaired, or may not be diagnosable as 'ill' in any formal psychiatric sense.

(e) Pyromania

The diagnostic criteria for pyromania are set out in DSM-V(Rev) TSM (1994) on page 615.[29] The condition and its diagnosis may be said to be one of exclusion. It is dealt with in detail by McElroy in Chapter (4.13.1) of this volume. Perhaps its manifestation of excitement for those who show the disorder is best exemplified by the poet Walt Whitman in his *Poems of Joy* (1860).

> I hear the alarm at dead of night,
>
> I hear the bells—shouts!
>
> I pass the crowd—I run!
>
> The sight of flames maddens me with pleasure.

(f) Sexually motivated fire-raising

The possible connection between fire-raising and sexuality has already been referred to. The lack of frequent association should not blind clinicians and others to its possible existence in certain cases, or its similarity to sex offending. Fras puts it well—as follows: 'In its comparative, stereotyped sequence of mounting pressure . . . it resembles the sexual perversions, as it may parallel them in its imperviousness to treatment'.[30] It is not without significance that imprisoned fire-raisers appear to have more than their fair share of psychosexual difficulties and partnership problems. In Hurly and Monahan's Grendon Prison study, a large proportion of their sample reported difficulties in social relationships with women.[31]

Suggestions for management

The word 'Suggestions' is used to indicate that what follows is not intended to be prescriptive; it must be emphasized that no single form of management is likely to be effective. At the assessment stage it is vital to treat every case as singular. Any attempt at assessing the future risk of fire-setting must view the behaviour on the basis of all the facts (for example, full details of the index offence and antecedent history). One can then begin to take the rounded and long view as advocated by Scott in his seminal paper on dangerousness. Assessors of whatever discipline will gain much from absorbing Scott's balanced and insightful views.[32] Pointers to successful assessment and management may be summarized as follows:

- Distinguish the fraudulent fire-raiser. But note that the fire-setter who appears to be engaged upon a fraudulent insurance claim to 'rescue' a failing business may be suffering from an underlying depressive illness. The history-taking in such cases needs to be painstaking and searching. Do not 'run' with what appears to be the obvious explanation.
- Distinguish the politically motivated. But, note that some politically motivated fire-raisers may also have serious mental health problems. In an age when fear of terrorist attacks abounds and the remedies appear to be of the 'knee-jerk' variety, a cool head in the assessment process is essential.
- Distinguish the vandalistic and the differences between young people who set fires out of boredom or for 'kicks' from child fire-setters who are more likely to have seriously dysfunctional social backgrounds.[33,34]
- Distinguish those who are driven to set fires by clear evidence of mental disorder, notably functional psychosis, severe anti-social personality disorder (psychopathy), organic disorder, and learning disability.
- Distinguish those who appear to exhibit pyromania as defined in DSM-IV TSM(Rev).
- Distinguish those rarer cases in which sexual disorder (and in particular sexual dysfunction) may have played a significant role.
- Distinguish the vengeful. It is important to remember that feelings of vengefulness may persist over long periods of time and such fire-raisers may be adept at concealing their vengeful feelings. These fire-raisers have some features in common with the delusionally jealous (Othello-type syndrome).

Successful assessment and management (which ideally should be a 'seamless' process) needs to rest upon a multi-faceted and multi-team approach. An excellent example of such a multi-disciplinary approach may be found in Clare *et al.*[28] They describe their management of a case that necessitated an understanding of both physical and learning disability combined with a capacity to work intensively using eclectic behavioural techniques over a prolonged period of time. Despite minor setbacks, the offender-patient, who had been subject at one time to containment in a high security hospital, remained free of his long-standing fire-raising behaviour at 4-year follow-up. It would also be unwise to believe that psychoanalytically based psychotherapy had no place in the management of psychotic and seriously personality disordered fire-raisers. Cox described some very productive work with such patients in Broadmoor.[35] Social skills training of one kind or another has a very important part to play. Many fire-raisers (particularly the vengeful and those with a pyromania diagnosis) are socially inept and believe themselves to be misunderstood by society. Techniques aimed at improving their self-regard, self-image, and social competence can help to minimize recidivism.

Conclusion

Not only is arson (fire-raising) a very worrying offence for the reasons given, but it has shown an increase in recent years. Moreover, the 'profile' of those convicted of arson has shifted over the years with an increased proportion of female offenders. And, in a very important recent study, Soothill *et al.* showed at 20 year follow-up that the proportion of those reconvicted for arson had more than doubled. The authors conclude with the sobering observation that 'the situation in relation to arson has deteriorated significantly over the past 40 years'.[36] The causes of fire-raising are complex and attempts at classification have not been entirely successful. Viewing fire-raising as a 'symptom' appears to offer the best hope for more successful diagnosis and management. The brief survey in this chapter has merely touched upon the topic. The suggestions for further reading and the references should assist those who wish to pursue the topic in further depth.

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Further information

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11.10

Stalking

Paul E. Mullen

What is stalking?

Stalking is now used to describe a problem behaviour characterized by repeatedly inflicting unwanted intrusions and/or communications on another in a manner which creates fear and/or significant distress.[1] The intrusions can involve, following, loitering nearby, maintaining surveillance, and making approaches. The communication can be via telephone (including SMS), letter, electronic mail, graffiti and notes attached, for example, to the victims' car. Stalking can be associated with a range of harassments which though not part of the core behaviours are all too frequent. These include, ordering goods and services on the victim's behalf (late night pizza's being a favourite) damaging property, spreading malicious rumours, vexatious complaints, threats, 'cyber terrorism', and assault.

There are two basic patterns to stalking.[2] The first involves repeated incursions predominantly in the form of approaches and following perpetrated most often by a stranger and lasting only a day or so. The second is characterized by a range of both communications and intrusions, is usually perpetrated by an ex-intimate or acquaintance, and lasts for weeks, months, or even years. The first type can be intense and distressing at the time but uncommonly culminates in a physical attack and though upsetting, rarely inflicts long-term psychological or social damage. The second type is associated not infrequently with psychological and social damage to the victim and will involve physical assaults in up to a third of victims.

The epidemiology of stalking

Estimates of the prevalence of stalking, as with any other phenomenon, will vary according to definition, sampling, method of enquiry, and the willingness of subjects to respond and respond frankly.[2–7] Reported lifetime rates of victimization for women are between 8 per cent and 22 per cent and for men between 2 per cent and 8 per cent. Most victims are female (70–80 per cent), most stalkers are male (80–85 per cent), with 20–25 per cent involving same gender stalking, typically male on male.

Cyberstalking

Cyberstalking has attracted considerable interest but few systematic studies. Even the definitions employed of cyberstalking vary widely.[8–10] As befits an online phenomenon much of the information about it is to be found on the Internet rather than in the more traditional sources of academic knowledge.

Sheridan and colleagues[11] in an important study concluded that cyberstalking was usually one more invasive technique for pursuing stalking rather than a distinct type of activity.

Cyberstalking can include the use of the Internet and SMS facilities to:

1 *Send repeated unwanted messages.*

2 *Order goods and services on the victim's behalf.*

3 *Publicizing private information of a potentially damaging or embarrassing nature.* Including circulating e-mails, placing information on the web containing personal details, and occasionally explicit sexual images.

4 *Spreading false information.* A wide range of misinformation can be spread via the Internet with the authors of these calumnies able, should they wish and have the necessary skills, to hide their identity.

5 *Information gathering online about a victim* can cover a wide range of material from addresses, employment histories, to financial details. There are even services for tracing people available online which can be utilized by stalkers whose victim has eluded them.

6 *Identity theft* goes beyond simply pretending to be the victim for the purposes of ordering goods or initiating contacts to an attempt to assume not just the name but the actual property and attributes of the victim.

7 *Encouraging others to harass the victim.* This can cover activities such as placing communication purporting to be from the victim on web likely to attract unwanted communications or attentions. The most egregious example involved a rejected stalker who posted personal advertisements in his ex-partner's name and giving her address which suggested she enjoyed being raped and solicited such attentions. Apparently six men actually came to her house in response to these provocations.[12]

Impact on victims

Stalking is both an act of violence in itself which causes psychological distress and social disruption, and is a harbinger of assault. Being stalked can produce a corrosive state of fear, arousal, and

helplessness. As with domestic violence for most victims it is not the blows which are the most destructive but living in a chronic state of intimidation and the expectation of imminent intrusion. In the study of Pathé and Mullen[13] the majority reported disruptive levels of anxiety with intrusive recollections of the stalking, sleep disturbance, lowered mood, with 25 per cent admitting considering suicide to escape the situation. A community study found increased rates of psychiatric morbidity and post-traumatic symptomatology amongst those stalked for more than 2 weeks but not amongst those who had experienced the briefer periods of harassment.[14] Dressing and colleagues[7,15] also document significant psychological and social disruption in response to being stalked with 56 per cent reporting agitation, 44 per cent increased anxiety, 41 per cent sleep problems, and 28 per cent increased depression.

Stalkers: classifications and typologies

Stalking, like most forms of complex human behaviour, can be the outcome of a wide range of psychological, social, and cultural influences. Some stalk in hope, some in anger, some in lust, some in ignorance, and many in mixtures of the above. In an attempt to advance the understanding of stalkers a range of typologies and classifications have been advanced.[16]

Classifying stalkers by the nature of their prior relationship with the victim has the advantages of simplicity and utility. The classification advanced by Mohandie and colleagues[17] represent the best supported by empirical evidence of such approaches to date. They divide stalkers into those with and without a prior relationship. Those with a prior relationship are subdivided into ex-intimates (ex-partners both long term and more casual) and acquaintances (including friends, colleagues, and professional contacts). Those without a prior relationship are subdivided into firstly 'public strangers' who were encountered through the media or in their public roles, and secondly into 'private strangers' encountered by chance in the interactions of everyday life. This classification's greatest utility is in predicting the risks of assault, with those with a prior intimate relationship constituting the highest risk group and those targeting public strangers the lowest. In their view the pursuers of public strangers are the most likely to be psychotic with those pursuing ex-intimates being relatively impervious to therapy but responsive to criminal sanctions.

The typology first developed by Mullen and colleagues[1,18] depends primarily of the context in which the stalking emerges and the motivations which initiated and sustained the behaviour. Its appeal has been primarily to clinicians managing stalkers and their victims.[19] There are five main types:

1 *The rejected* whose stalking begins in the context of the breakdown of a close relationship. The stalking is initially motivated either by the desire for reconciliation or to express the rage at rejection, with a mixture of both being quite common. The stalking is often sustained by the pursuit of the ex-partner becoming a substitute for the lost relationship with the satisfactions from intrusion and control replacing those of intimacy.

2 *The intimacy-seeker* who is pursuing love. The stalking begins in the context of a life bereft of intimacy and is motivated by the hope, or firm expectation, of obtaining a loving relationship with a stranger or casual acquaintance on whom they have fixed their amorous attentions. The pursuit is sustained in the face of indifference or outright rejection because better a love based on fantasy or delusion than no love at all.

3 *The incompetent suitor* who is pursuing a sexual encounter or friendship. This usually begins in the context of loneliness and is motivated by a desire to start some form of relationship with someone who has attracted their interest. This group often pursues intensely with multiple intrusions but rarely persists for more than a day or so, presumably because multiple rebuffs bring few rewards.

4 *The resentful*, whose stalking starts in the context of a grievance at being unjustly treated or humiliated. The initial motivation is revenge but this gives way to the satisfactions obtained from the sense of power over someone who has previously been experienced as an oppressor, or the representative of oppressors.

5 *The predatory*, which begins in the context of the desire to act out violent or sexual fantasies often of a sadistic or paedophilic nature. The initial motivation is to gain information about the movements of a potential victim (usually a stranger but occasionally an acquaintance). The stalking continues because of the satisfactions accruing not just from voyeurism but from the excitement and sense of power which comes from rehearsing the planned attack in fantasy whilst watching the future victim.

Each of the stalker types, hopefully with the exception of the predatory, has correlates in normal behaviour. When relationships break down one partner is often confused or distressed by the separation and seeks to understand, to reconcile, or to express anger. The incompetent suitor is kin to the awkward adolescent male and the socially inept adult who fails to traverse effectively the social minefields of courting or simply making acquaintance. The intimacy seeker is the adolescent crush and the enthusiastic fan writ large. Even the resentful is not far removed from some seekers after justice and those asserting their rights. In theory the boundary between persistent approaches as part of socially acceptable behaviour and the crime of stalking are difficult to pin down. In practice the distinction is rarely a problem. Stalkers are those who repeatedly force themselves on another person in a manner which creates obvious distress. It is the total disregard, or blindness to, the disturbance and often fear that their behaviour creates which distinguishes the stalker from their more normal counterparts. Sometimes the stalkers are so caught up in their own world they are oblivious to their effect on others. Sometimes they are blinded by delusion. Sometimes self-righteousness makes them indifferent. But sometimes they delight in the effect they produce in their victim.

Psychopathology of stalkers

Stalkers are rarely, if ever, drawn from the psychologically adequate or socially able of the world. The estimates of the proportion of stalkers whose behaviour is directly related to mental disorders varies according to where the researchers derived their sample. For example, Zona and his group[20] whose sample contained many who pursued Hollywood celebrities had a significant number with erotomanias and morbid infatuations.

In broad terms psychotic disorders are relatively frequent in the intimacy seeking group. In the resentful type it is the paranoid disorders which unsurprisingly predominate, though most are not

associated with frank delusion. The rejected often have problems around dependency, rigidity, control, and self-esteem with substance abuse and depressive states on occasion complicating the picture, but psychotic states are uncommon. The incompetent suitors are socially disabled sometimes by shyness, sometimes by narcissism, sometimes by intellectual limitations, sometimes by culture, sometimes by disorders such as Asperger's syndrome, rarely by psychosis, but always by interpersonal insensitivity or indifference. The predatory are sexually perverse and not infrequently have marked psychopathic traits, but again are rarely psychotic.

Attempts have been made to conceptualize stalking as a manifestation of obsessive–compulsive disorder. Stalkers are certainly often obsessive in the everyday sense of that word in their pursuit of the victim. They rarely however regard their behaviour as unjustified let alone irrational, and few see their persistence as senseless. They may resist the urges to stalk on occasion but for the most part devote themselves wholeheartedly to the pursuit. Anxiety is more likely to be generated by the fear of failure, or of consequences, than by not acting on their impulses to stalk. They may well spend many hours thinking about the object of their unwanted attentions, and in the resentful reliving the experiences of actual or supposed injustice, so in that sense they are ruminators. Personality traits of rigidity, rumination, and the overvaluing of order are not infrequently so marked in rejected and resentful stalkers as to justify a label of an obsessional personality. In short, the behaviour often has an obsessive quality but the state of mind rarely conforms to that found in obsessive–compulsive disorders.

Attachment theory has unsurprisingly been evoked to explain stalking. That stalkers as a group don't do interpersonal relationships very well is obvious. Evidence exists that insecure attachment styles predominate amongst rejected stalkers, the intimacy seekers may have the type of secure attachment style only sustainable by delusion, and the incompetent and resentful favour the dismissive style. This is useful in assessment and management but what connection it may have with any theory of early development is speculative and here as elsewhere more likely to be productive of mythologizing than good clinical practice.

The stalking of health professionals

Health care professionals have a heightened vulnerability to being stalked by their patients and clients.[21–24] The risk stems largely from resentful and disappointed patients but in part from lonely and disordered people who misconstrue sympathy and attention for romantic interest. While some stalking behaviours constitute little more than minor irritations, they may also ruin a clinician's career.

Sandberg *et al.*[25] studied an inpatient psychiatric service reporting 53 per cent of clinical staff had been stalked by patients. Galeazzi and colleagues[26] found 11 per cent of the mental health professionals in an Italian service had been stalked for lengthy periods by patients. Purcell and colleagues[14] surveyed a randomly selected sample of 1750 psychologists (73 per cent female). The lifetime prevalence of stalking by clients was 19.5 per cent with 8 per cent being stalked in the preceding 12 months. Most victims were working in direct client care (95 per cent) and experienced rather than new entrants to the profession. Stalkers fell predominantly into either the intimacy seeking (19 per cent) or resentful (42 per cent) types. Over 30 per cent of psychologists in this study were subjected to vexatious complaints by their stalker. The impact of complaints to professional boards, health ombudsmen, and other agencies of accountability can be devastating.

Too often in the past therapists who fell victim to stalking by patients had to bear the additional burden of implied or overt criticism from colleagues to the effect that, had they more adroitly managed the therapeutic encounter and the resultant transference, they would not now find themselves in this predicament. There should be no sympathy with blaming the victim, even if it comes in the guise of technical advice or supervision. Being stalked is a risk inherent in the therapeutic process. Our colleagues should be accorded support and help, if for no other reason than we do not know when it may be our turn to face the pursuit of the vengeful or lustful patient.

Risk assessment and risk management

Stalking came to prominence because it was regarded as a risk factor for violence. Subsequently it became clear that the damage inflicted on those who are stalked could also encompass significant social and psychological damage.[23]

Assessing and managing the stalker requires a primary focus on the risks they present to the victim. Nevertheless the risk that stalkers incur from their own behaviour also needs to be considered (Table 11.10.1). The conflict between the stalker's desires and the victim's interests are obvious, but they are at one in being at risk of damage from the stalking situation. There can be a tragic symmetry between the victim forced to live an increasingly restricted life in a state of constant fear, and the stalkers devoting all their time and resources to a damaging and ultimately self-defeating pursuit. The victim's and the perpetrator's lives can be laid waste. This is not to argue for equivalence between victim and perpetrator but merely to note they share the chance of disaster. A perspective which encompasses the risks to stalkers and victims has the advantage for health professionals of reducing the ethical dilemma when treating stalkers around whose interests one is serving, the patient's or their victim's. We help both to the extent that we contribute to stopping the stalking, or reducing its damaging consequences.

The empirical basis for evaluating risk in the stalking situation

Risk assessment in stalking situations is currently hampered by a paucity of either retrospective or prospective studies of representative samples. Clinicians do not, however, have the luxury of deferring action until such evidence emerges. They must, for the present, depend on integrating knowledge from stalking research, borrowing from the systematic studies of risk in other areas, and drawing on clinical experience.[27]

The risk of continued or recurrent stalking

The duration of stalking is longest for rejected stalkers pursuing ex-intimates and intimacy seekers, with the incompetents usually pursuing only briefly. Women, here as elsewhere, are more persistent than men.[6,13] Once stalking has continued for more than 2 weeks the chances are high that it will continue for months.

The risks of psychological and/or social damage

Female victims of stalking report a greater psychological impact than male victims.[5,13] Clinically, the distress and disruption to

Table 11.10.1 The stalker's clinical risk factors and future hazards specific to stalking situation

Risk factor	Management possibilities include
Clinical	
1. Mental state e.g. depression, delusional preoccupations	Active treatment usually involving pharmacotherapy
2. Substance abuse	Referral to a specialist substance abuse service where possible or to self-help groups like AA
3. Anger	Anger management remains a problematic area. Ideally those with anger control problems should be receiving special help independently of the broader management of their stalking
4. Attitudes towards, and beliefs about, the victim which sustain stalking	Appropriate legal interventions; CBT and focussed psychotherapies aimed at such areas as; abandoning love, accepting loss, confronting misperceptions
5. The conviction that they are right to engage in stalking	Enhancing victim empathy. Confronting false attributions using CBT
6. The refusal to engage in any therapy, or conform to legally imposed restrictions on access to the victim	Ultimately confronting the stalker with consequences (e.g. through breaching parole, referring back to court, etc.); employing motivational interviewing strategies to assist the stalker to appreciate the need for intervention
7. Social incompetence	Social skills training, therapies aimed at enhancing self-efficacy
8. Paraphilia	Sex offender program incorporating CBT +/− pharmacotherapy as indicated
Future hazards	
1. Likely future contact with the victim	Every effort should be made to enforce a total ban on direct contact or direct communications
2. Lack of a feasible set of plans for avoiding a recrudescence of stalking	Ensure structured plan around avoiding provocations and using protections re: stalking; CBT to assist the stalker to overcome the compulsion to stalk
3. That the underlying precipitants remain unresolved	Focussed psychotherapy aimed at the areas identified in the formulation; social skills training for the inept; assistance abandoning the relationship; the treatment of paraphilias using CBT +/− pharmacotherapy as indicated
4. Continuing instability of residence and/or employment	Assistance obtaining housing; career counselling; and active employment rehabilitation as indicated and appropriate
5. Continuing social isolation	Use of clubs, day centres, recreational counselling, domestic pets
6. Likely low level of compliance with legal restraints on contact with victim	Ensure knowledge of consequences of breaches and never collude—implicitly or explicitly—with avoiding those consequences
7. Likely low level of cooperation with any treatment programme	Use of compulsory community treatment orders either imposed by court or as part of mental health legislation

victims is usually most obvious in ex-intimates pursued by their rejected partners, perhaps because of the higher levels of violence and intimidation combined with the complexity, as well as the intensity, of feelings stirred up in this situation.[13] Psychological distress was higher amongst victims who were subjected to prolonged and repeated following and the experience of property theft or destruction.[2,13,28,29] The relationship between psychological impact and the experience of physical violence is less clear, despite its intuitive appeal.[29,30]

The risks of violence

(a) Prior relationship

Victims who have shared a prior intimate relationship with their stalker are at a high risk of physical violence.[1,4,13,20,31–35] Purcell and colleagues[6] for example reported in a random community sample that ex-intimates were the most likely to be attacked (56 per cent), followed by estranged relatives or previous friends (36 per cent), then casual acquaintances (16 per cent), work-related contacts (9 per cent), and finally strangers (8 per cent). Such findings should not be interpreted, however, as suggesting that victims of stalkers who are not ex-intimates are in little danger of physical violence. A chance of between 8 per cent and 36 per cent of being assaulted is no small risk.

(b) Threats

Between 30 per cent and 60 per cent of stalking victims are threatened.[1,20,31] In a community-based study 44 per cent of those threatened were subsequently assaulted and 73 per cent of victims assaulted by their stalker had previously been explicitly threatened.[6] In short threats predict violence and should be taken seriously.

(c) Mental disorder

Research has generally concluded that psychotic stalkers are less likely to be physically violent than their non-psychotic counterparts but the relationship to personality disorder remains unclear.[1,34,36,37]

(d) Substance abuse

Substance abuse is associated with violence in the stalking situation.[1,38–40]

(e) Prior offending and antisocial behaviour

The empirical data on the association between past criminal or violent behaviour and stalking violence is inconsistent, however the balance of the evidence favours such a relationship.[1,34,39,41]

(f) Demographic variables

The gender of stalkers has repeatedly been shown to have no impact on the prevalence of either threats or assault.[4–6,13,42]

(g) The nature of the stalking

Violence is predicted by escalating intrusiveness and intensity of the stalking behaviour. The strongest association is to physical intrusions into the victims house or place of work.[36]

The assessment process

Initial assessments of stalkers often occur in the context of pre-sentence or parole board evaluations. Victims may be encountered in a wider range of contexts, many seeking help from general rather than forensic mental health professionals. Stalkers usually lack insight into their behaviour and tend to deny, minimize, and rationalize their actions. Victims often minimize the experience of stalking and over-emphasize their own responsibility for the harassment, which should be of no surprise to anybody experienced in working with victims in other contexts. Conversely, the problem of false claims of stalking victimization cannot be entirely ignored.[43] This makes it essential to assess collateral information from such sources as witness statements, victim impact reports, judges' sentencing remarks, and professional to professional contacts, confidentiality allowing. Attempts to contact the victim when assessing the stalker, or the stalker when assessing the victim are, in our opinion, best avoided. However skilfully managed, such contacts tend to be experienced by the victim as the professional acting as an agent of the stalker, and by the stalker as support for their beliefs that this is a misunderstanding within a mutual relationship rather than a unilateral imposition of unwanted attentions.[27]

Management

The management of stalkers remains very much the province of forensic mental health professionals and even amongst them it is a specialist area. Basic approaches to identifying potentially remediable risks and their management however is presented in. Victims of stalking are however likely to be seen by a wide range of mental health professionals.

Reducing the impact on victims

Stalking victims will often present with significant problems with anxiety symptoms and depressed mood. The symptom complex of PTSD will be present to a greater or lesser extent in most victims of prolonged and intense stalking. Like many victim groups there may be a reluctance to disclose the details or even the existence of the traumatic experiences. As noted earlier self-blame is not infrequently part of the picture.

Given the high profile of stalking there are a number of disturbed people who claim they are being stalked as a way to express their distress, claim attention, or give form to their persecutory delusions.[24,43] This group, particularly if delusional, are often obvious given the flamboyant, implausible, and exuberant accounts of victimization. Care should be taken in dismissing claims of being stalked, however, as there are some very unpleasant stalkers out there some of whom stalk leaving few, if any, objective signs. False victims require help and treatment not rejection, but they require quite different treatment from actual victims.

Stalking victims need first and foremost good psychiatric care. Manage the stress symptoms, treat the depression, ameliorate the distress, and provide adequate support. Individual treatment is best initially but the use of groups for long-term support and treatment is worth considering.[44] What follows is a brief account of stalking specific interventions.

1 Informing others. When you are stalked it is essential to inform those you live with, work with, and are friends with. This performs three functions:
   (a) It allows you a 'reality check' on your fears that stalking is occurring.
   (b) It enables others to support you and equally important avoid inadvertently assisting the stalker.
   (c) It prevents those around you being put at risk by ignorance provoking the stalker thereby also falling victim.

3 Avoid contact and/or confrontation. All contacts or direct communications with a stalker risks reinforcing their behaviour. Confrontation and worse still violence, legitimizes, and encourages their violence. Once stalking is established it is usually too late to simply sort things out by having a meeting.

4 Documentation. The best protection for stalking victims lies in the criminal law. The police are more likely to respond appropriately if you can demonstrate that the behaviour is occurring and make it relatively easy for them to pursue the allegations. Keep the letters (dated), record the phone calls and retain the tapes, keep a diary noting approaches and where possible witnesses to those events, record verbatim any threats and obtain witnesses or photographs of any property damage. Not only will this be invaluable for a later prosecution it is part of the victim taking control back over their lives.

5 Restraining, non-molestation, apprehended violence and other such orders. Sometimes these civil orders do provide an effective means of reducing or stopping stalking. Police often advise their use, hopefully not just to avoid work. Some jurisdictions insist on obtaining such orders prior to considering criminal prosecution. Reservations have to be expressed, however, not only about their effectiveness (totally ineffective for intimacy seekers, relatively ineffective for the rejected and intermittently effectively for the resentful) but also about the level of insecurity and distress consequent on their breach and facing the frequent indifference of the police to such breaches.

6 Increased security. Simple cheap security measures such as good locks, movement triggered lighting outside the house, and securing the mail box may provide a degree of reassurance and a modicum of security.

Conclusions

Not a few psychiatrists, at least of my generation, have difficulties with the notion of problem behaviour like stalking being a proper subject for mental health concern. Psychiatry has traditionally been wary of concerning itself directly with criminal and antisocial behaviours.[45,46] The approach taken in this chapter was, in contrast, to define a pattern of behaviour destructive to the interests of perpetrator and victim and then to examine its origins, effects, and potential therapeutic management. That this is an enterprise with risks for the ethical integrity of psychiatry is undoubted. But recognizing that psychiatry can have a role in assessing and managing problem behaviours, without first performing obfuscating transformations into supposed mental disorders such as paraphilias

and impulse control disorders, allows a more clear sighted and effective approach to areas of human activity where our intervention can benefit both the actor and the wider community.

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11.11

Querulous behaviour: vexatious litigation, abnormally persistent complaining and petitioning

Paul E. Mullen

> Querulantenwahn (Ger.) A form of so called paranoia in which there exists in a patient an insuppressible and fanatic craving for going to law in order to get redress for some wrong which he believes done to him. Individuals who fall victim to this disorder are always strongly predisposed . . .. extremely egotistical . . . know everything better . . . differs from other forms of paranoia in so far as the wrong may not be quite imaginary . . . the more he fails the more he becomes convinced that enormous wrong is being done to him . . . neglects his family and his business . . . going down the road to ruin.[1]

The above quote neatly summarizes classical psychiatry's view of querulous, or litigious, insanity as a form of paranoia. A problematic form, however, in that the querulousness was usually based on a genuine grievance and was often regarded as developing on the basis of predispositions rooted in the sufferer's personality.[2–4] As to treatment Krafft Ebing[3] notes the 'necessary and beneficent (effects of the) appointment of a guardian and commitment to an asylum' but regretted that this 'takes place unfortunately only after they have used up their property, insulted the courts, and disturbed public order' (p. 395).

Psychiatries interest in the querulous (from the Latin to mutter and to mumble) waned rapidly in the latter half of the twentieth century. The diagnosis was appealed to less and less and the literature largely fell silent.[5] In part the disappearance of querulousness, and even the querulous patient, from the realms of psychiatry paralleled the decline of paranoia as a diagnostic entity. In part it reflected psychiatries increasing reluctance to play the role of social regulator. Probably most importantly the emerging culture of individual rights made pathologizing complainants potentially disastrous as it could deprive them of access to the major social mechanisms for obtaining justice.[6] Psychiatry lost interest in the querulant, however, at the very time that the 'culture of complaint' drew more and more vulnerable people into the systems of complaint management. Agencies of accountability, which range from Ombudsmen's offices, via registration boards, to complaints departments, are now almost all faced with the problems created by a small group of people pursuing grievances with a persistence and insistence out of all proportion to the substantive nature of their claim. It is estimated that 20–30 per cent of the resources of these agencies are being consumed by less than 1 per cent of unusually persistent complainants. In the civil and family courts the number of interminable cases being pursued, often by unrepresented litigants, escalates year by year. Last, but not the least, querulants pursuing what they regard as their rights through repeated petitions and intrusive approaches to politicians and heads of state distract protection services from more substantial threats.[7–9]

Clinical features

The querulant pursue their vision of justice through litigation in the court, through petitions to the powerful, and finally through the various agencies of accountability. In practice all three avenues are often explored. In the nineteenth and early twentieth century it was the civil courts in which these dramas were usually played out. Today the main burden falls on the complaints organizations.

It is not easy to distinguish the querulant from the difficult complainant or even from social reformers and victims of gross injustice. A simple typology may assist:

1 *Normal complainants* are aggrieved seeking compensation, reparation, or just an apology. They will accept conciliation and reasonable solutions, though they may become persistent and insistent if provoked by inefficiency or injustice.

2 *Difficult complainants* also seek compensation and reparation but often want in addition retribution. They tend from the outset to anger, to seeing themselves as the victim of others intentional malevolence, and to resist all solutions but their own. Eventually, however, they will settle for the best deal they can obtain.

3 *Altruistic reformers* who pursue goals of social progress via the courts, petitions, and complaints. They sacrifice their personal interests in pursuit of better outcomes for others. Though they may have a political agenda which is sectarian (e.g. antigenetically

modified foods, fathers rights) they do not have idiosyncratic and personalized objectives.

4 *Fraudsters* who knowingly pursue false or grossly exaggerated claims.

5 *The mentally ill* whose claims are driven by delusional preoccupations frequently bizarre in nature which reflect underlying disorders often of a schizophrenic type.

6 *The querulous* who seek personal vindication in addition to compensation, reparation, and retribution. They are on a quest for justice which becomes totally preoccupying. Unlike reformers, and most of the difficult, there is an obvious discrepancy between the provoking event and the importance attached to it by the querulous. They appear to seek not resolution but continuation of the conflict. They lay waste to their social and economic functioning.

The querulous are usually males who first become embroiled in complaining and claiming in their fourth or fifth decade. Premorbidly they were often able to function reasonably well. They rarely have criminal records or prior psychiatric contact, and substance abuse is not prominent. Many had relationships but by the time they reach psychiatrists they have usually alienated their family and friends. Querulants are often disappointed people who feel their qualities have been ignored and left unrewarded. Their pursuit of justice offers an opportunity to vindicate their lives and obtain the public recognition so long denied. Their personalities tend to have the traits of self-absorption, suspiciousness, and obsessionality combined with an enviable capacity for persistence.

Clinically they typically present as energized, garrulous individuals eager to convince you of the merits of their case. There is an enthusiasm which can seem almost manic but unlike the manic they are totally focussed and almost impossible to distract from their narrative of injustice. They may come with bags overflowing with documents testifying to their misplaced scholarship. If challenged they usually become patronizing as they pedantically refute all objections, to their complete satisfaction. Alternatively they may become menacing and overtly threatening.

Communications from querulants were noted by Lester and colleagues[10] to be often characterized by:

- Multiple methods of emphasis including, underlining, highlighting (often in multiple colours), and capitalization
- The generous use of inverted comas, exclamation marks, and question marks
- Numerous foot and marginal notes
- The use of attachments, often extensively annotated, some potentially pertinent (e.g. letters received, copies of legislation), others of less obvious relevance (e.g. Magna Carta, UN Declaration of Human Rights)
- And many many pages.

The content of communications may also be unusual sometimes containing:

- Legal, medical, and other terms used frequently but often incorrectly
- Repeated rhetorical questions
- A curious combination of rambling repetitiveness with pedantry (more difficult to describe than recognize)
- Veiled threats to harm themselves or others if their wishes are not granted or
- Exaggerated politeness and attempts to ingratiate.

Clinical assessment

The querulous can only be adequately assessed by considering the development over time of their behaviour as well as their state of mind. In an interview they may present as merely overenthusiastic and over hopeful pursuing their legitimate rights with at worst a degree of fanaticism. It is the unfolding of their story which reveals the damage they are suffering, and they have inflicted on those around them.

Case history (representing an anonamized conflation of several cases)

A man in his late forties made a complaint to the local bank manager over the manner in which mortgage documents had been prepared. There were grounds for legitimate concern as irregularities had occurred, though of a minor nature and of a kind which might have been expected to be to his advantage. His initial complaint to the bank was rejected. He appealed unsuccessfully to the banking ombudsman. He stopped paying the mortgage and initiated civil action. Over the next 5 years he pursued his complaints with the human rights commissioner, the securities exchange commissioner, consumer rights organizations, via further civil litigation, and petitions to Parliament and the Queen. The foreclosure on the mortgage intensified the complaints and litigation. Finally he made a series of bomb threats leading to his prosecution and referral. When assessed he was righteously indignant, believing he had been right to take extreme action to bring attention to an injustice which had destroyed him and his family and threatened the very economic fabric of the nation. He firmly believed he was owed millions in punitive damages, and that when he inevitably prevailed this would bring down the transnational banking corporation which owned his particular branch office. He regarded himself as a whistle blower who would be publicly recognized as one of the major social reformers of his generation. The changes over time in the grievance, the agents, his state of mind, social situation, beliefs, and aims are presented schematically in Table 11.11.1

Traditionally psychiatry has attempted to distinguish between deluded querulants, who are in the business of mental health services, and the non-deluded, who are not. Unfortunately for this approach the querulous present a formidable phenomenological challenge. They advance their ideas plausibly making apparently rational connections between the underlying grievance, which is almost always based on some actual injustice, and their current claims and complaints. Unlike many deluded patients, their beliefs do not usually seem to arise either on the basis of some difficult to understand interpretation of an event, or from an idiosyncratic insight into reality. The querulous offer a detailed and apparently logical account of the emergence of their grievances and the progress of their quest for justice. Reasonable that is if taken in cross section but not when considered over time when there emerge gross discrepancies between the supposed initiating cause and subsequent behaviour. The persuasive presentation can obscure the essential absurdity of the quest and distract attention from the chaos they have created for themselves and those around them. The temptation is to normalize the clinical presentation but this is

Table 11.11.1 A case of querulous behaviour: the changes over a 5-year period in the various domains illustrating the descent from the reasonable if over hopeful and oversensitive to the unrealistic and unrealizable

Grievance	*Agents*
Errors in mortgage documents	Bank's accountant
Potential financial loss	Plus manager
Actual financial loss	Plus senior management
Victim of major fraud and theft	Plus banking ombudsman
System wide corruption	Plus lawyers and judges
A campaign of financial corruption threatening the nations economic stability	Plus wife
	Plus various public agencies
	Plus police
	Plus prime minister
	Plus secret services
State of mind	*Social situation*
Rigid discontented man obsessional traits but articulate and ambitious	Moderately successful small businessman, married, two children, but experiencing financial pressures and marital problems
Increasingly fixated on grievance	Business begins to fail as all his attention moves to grievance
Pursuit of justice subordinates all other concerns	Marriage breaks down
Increasingly convinced he is being persecuted and spied upon	Alienates few friends he had
He is a man of destiny fighting forces of national and international corruption	Bankruptcy
	Living alone
	Destitute
Beliefs	*Aims*
That order and due process are the bedrock of civilization	Compensation for malfeasance (the sum rapidly escalating over the years)
Bad things happen not by chance but because of carelessness or malevolence	Reparation—a return of his house mortgage free
That he had never received the recognition and rewards he deserved	Retribution—punitive damages, sackings, and criminal prosecutions
He was destined for greatness	Vindication by public recognition as a whistle blower who had reformed the banking system

(Reproduced from P. E. Mullen and G. Lester, Vexatious litigants and unusually persistent complainants and petitioners: from querulous paranoia to querulous behaviour, *Behavioural Sciences and the Law*, **24**, 333–49, copyright 2006, John Wiley & Sons, Inc.)

to ignore both the peculiarity of their behaviour and beliefs, as well as the devastation they have wrought on their own lives. Sometimes the querulous are obviously deluded, sometimes they appear to inhibit that borderline which is captured in such terms as overvalued ideas and delusion like ideas. Debates over the phenomenological niceties should not, however, distract from recognizing the pathological nature of such querulousness.

The querulous are sometimes regarded as obsessional. The level of preoccupation, the ruminative quality of their thinking, and the pedantic attention to the minutiae of their case, all suggest obsession. Certainly most, if not all, querulants have obsessional personality traits. But the querulant does not regard their core beliefs and the behaviour as absurd or absurdly insistent. Quite the reverse they know they are right and are totally identified with their ideas. The querulous therefore may be regarded as obsessive or fixated but not as having an obsessional disorder.[6]

Management

Our courts and agencies of accountability are designed to deliver conciliation, arbitration, reparation, and compensation, but rarely retribution, except in the exceptional case of punitive damages, and never personal vindication. The querulous seek above all personal vindication and retribution so from the outset are doomed to fail.

One view is that it is the failures of the courts and complaints organization that drive claimants to become querulous. Charles Dickens articulates this in his great novel Bleak House arguing the courts 'give to monied might the means abundantly of wearying out the right. . . (which) so overthrows the brain and breaks the heart to leave its worn out lunatics in every madhouse'. Lester and colleagues[10] in their study of unusually persistent complainants failed to document any significant differences between the manner in which the complaints had been dealt with in those whom became querulous and those who did not. This suggests a role in the pathogenesis for vulnerability not just reacting to provocations.

The impact of querulousness can be reduced by improved recognition and improved management practices in courts and agencies of accountability.[6] Psychiatrists currently only tend to be involved after the situation has reached the stage of the querulant either becoming seriously depressed or being charged with threats, violence, or contempt.

The literature on the therapeutic management of the querulous is both small and predominantly discouraging.[11] Ungvari,[12] however, reported successful treatment using pimozide. Our own experience is that relatively low doses of atypical antipsychotics are helpful though the response is slow in coming often taking months before there is obvious improvement. The first problem is attaining some semblance of a therapeutic alliance with the patient. This requires avoiding being caught up in discussions of the rights and wrongs of their quest. The focus should be on the price they and their family are paying for the pursuit.[6] Interestingly some of those who come on orders from the court which mandate treatment will accept medication and other therapeutic interventions as they wish to make clear they abide by the law. Paradoxically they can

be ultra compliant patients. A number have continued voluntarily in treatment after the end of the order though they never acknowledge either that they were in error or in need of treatment because of their querulousness. What changes is the involvement in the querulous ideas, the degree of preoccupations, and the behaviour, but the core belief that they were right never wavers. Querulous behaviour appears to be sustained by a range of cognitive distortions including:

- Those who do not fully support their cause are enemies.
- Any lack of progress is the product of malevolent interference from someone.
- Any compromise is humiliating defeat.
- The grievance is the defining moment of their lives.
- That because they are in the right the outcomes they seek must be not only possible but necessary.

These distortions are open to challenge and amelioration if not completely overcoming. In theory the cognitive therapy approaches advocated for the delusions should also be of value.[13,14] The problem with the therapeutic management of querulous behaviour is that we have no trial of treatment or even much beyond case reports. This reflects widespread prejudice that the querulous are not the business of mental health and even if they are they are untreatable. Hopefully if this neglect is overcome and querulous behaviour is once more recognized as a legitimate concern for mental health professionals then systematic studies of therapy will follow. For the present the querulous destroy the fabric of their lives as well as creating distress and occasionally damage to those around them.

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11.12

Domestic violence

Gillian C. Mezey

Introduction

Over the last decade, the issue of domestic violence has been transformed from a position of 'selective inattention' to becoming a high-priority social and public health issue.[1] Although it is now recognized that experiences of domestic violence are associated with adverse mental as well as physical health outcomes for the victim, this has not always been the case. Early psychiatric writings tended to attribute responsibility for violent relationships, to the masochistic traits of women who are drawn to and then fail to separate, from abusive and violent partners. During the 1980s and 1990s, however, the perception of victims of domestic violence, or 'battered women' began to change, towards an understanding that the responsibility for domestic abuse lies with the wife beater, rather than the wife beater's wife.

This change came about as a result of several factors; first, effective lobbying by the feminist movement which put the issue of domestic violence firmly on the political agenda; second, the influence of a number of researchers who began to conceptualize the psychological and behavioural problems seen in victims as the consequence, rather than the cause of, domestic violence, for example through the identification of a specific 'Battered Woman Syndrome',[2] and finally the introduction of Posttraumatic Stress Disorder as a distinct psychiatric diagnosis in 1980 (APA, 1980).

Definition

Domestic violence is currently defined in the United Kingdom as 'Any incident of threatening behaviour, violence or abuse (psychological, physical, sexual, financial or emotional) between adults who are, or have been in an intimate relationship'.[3] Most cases of domestic violence involve the abuse of a woman by her male partner, however, domestic violence may also involve other family members, same sex partners, and the abuse of men by women partners.

Epidemiology

The estimated prevalence of domestic violence varies, according to the definition being used and the population surveyed. Based on an analysis of 48 population-based studies from around the world, the prevalence of domestic violence is between 10 and 69 per cent over a lifetime and between 3 and 52 per cent in the past year.[4] In the United Kingdom, around 16 million incidents of domestic violence are recorded annually, with a lifetime prevalence of 21 per cent lifetime and 4 per cent in the past year for women and 10 per cent lifetime and 2 per cent in the past year, for men.[5] Women who report domestic abuse consistently report experiencing more incidents and more injuries and being more fearful of their partner, than male victims.[5]

Aetiological factors

Domestic violence arises out of a complex interplay of personal, situational, and socio-cultural factors. Poverty, alcohol use, low academic achievement, being single, separated or divorced, and witnessing or experiencing violence as a child are the most important individual risk factors for domestic violence.[4] Poverty operates at an individual and societal level, in that it places increased stress on the individual and the family system and also acts as a marker for a number of other social conditions (e.g. low education, overcrowding) that combine to increase the risk of domestic violence. Alcohol use by the perpetrator also significantly increases the risk of domestic violence.[4,5] Women appear to be at particularly high risk of domestic violence during pregnancy and the immediate post-partum period.[6] Experiences of domestic abuse in childhood and adolescence are associated with an increased risk of perpetration, for men[4] and re-victimization, for women[7] in adult relationships. Inequality and a power imbalance within the relationship[8] and in the wider society,[9] also increases the risk of domestic violence. Higher rates are found in societies where men have economic and decision-making power in the household, where women do not have easy access to divorce, and where there is a high level of public acceptance of men's right to discipline their wives.[4,9]

Mental health effects of domestic violence

Domestic violence is associated with a range of adverse physical and psychological health and social outcomes.[1,4,5] In extreme cases, the violence results in the victim's death. Between 40 and 70 per cent women victims of homicide are killed by a current or former spouse or partner, compared with between 4 per cent and 8 per cent male victims of homicide.[4]

Women who are abused by their partners over many years, often find it extremely difficult to leave their partner and, even if they do leave, many women end up returning to the family home. There are a number of psychological and social reasons why many women find it difficult to separate emotionally, as well as physically, from their violent partners. Learned helplessness,[2] the progressive erosion of confidence and self esteem cased by the abuse, feelings of guilt, shame and isolation, make it difficult for victims to assert themselves, seek help, or even contemplate the possibility of an existence separate from their partner. For some women the abuse appears, paradoxically, to strengthen their emotional ties to the perpetrator, so-called 'traumatic bonding'.[10] Women who try to leave abusive partners, experience significant economic and social hardship, including lack of accommodation, inadequate financial support, and difficulties in caring for the children, as well as a disruption caused to their family and social support networks. These difficulties, as well as the fear of being tracked down by their partner, are often so daunting, that many women end up returning to their homes, choosing an existence that is familiar to them, in preference to precarious survival elsewhere. It is clear that physical separation does not always end the violence; many separations result in an escalation of threatening and violent behaviour, including stalking[11] and the risk of domestic homicide is greatest around the time, or shortly after, separation.

Domestic abuse is associated with increased rates of depression, suicidality, Posttraumatic Stress Disorder, alcohol and substance misuse, and dependence in victims of abuse, compared with the general population.[12] The more severe and chronic the abuse, the greater the impact on the victim's mental health and symptoms of depression and hypervigilance may persist, even after separation.[2]

Where present, symptoms of mental illness must be treated appropriately. However, offering victims a non-judgemental sympathetic response, providing information about options available to them, about the risks associated with staying, or going and providing information about community resources, including refugees and counselling facilities are likely to be more important for the victims than any specific 'treatment'.[13]

Risk assessment

In the absence of direct questioning by a health professional, which is conducted in a sensitive and non-judgemental way, women are unlikely to spontaneously disclose experiences of domestic abuse in the context of a health consultation.[5,13] Unless domestic abuse is identified, then the risk cannot be properly assessed or communicated to the victim. Screening for domestic abuse in health settings appears to be acceptable to women and is also effective in terms of increasing rates of identification.[13]

The frequency, severity, and chronicity of the abuse must be taken into account in assessing and managing risk. Browne's study,[14] comparing battered women who had killed their partner with battered women who had not killed, identified the following risk factors for domestic homicide: frequency of violent assaults; presence and severity of injuries; alcohol intoxication or substance misuse in the perpetrator, threats to kill, sexual violence, suicidal ideation (in the victim), and access to a weapon. Campbell[15] also identified the following risk factors in a study of 220 cases in which women had been killed by violent partners: unemployment (male), choking, abuse during pregnancy, threats or harm to the child, the presence of a stepchild in the home, and separation. Morbid jealousy in the perpetrator and stalking behaviours are also more common in lethal, compared with non-lethal cases of domestic violence. Risk assessment must be carried out in all cases of domestic violence and communicated to the victim, to allow her to make informed and safe choices.

Confidentiality and domestic violence

The primary consideration for the health professional should be the safety of the victim and any affected children. In general, the victims consent should always be sought prior to sharing information with others. A breach of confidentiality, without the victim's knowledge, or consent, could increase the risk she faces and may discourage further disclosures to health professionals. The legal framework varies in different countries but in the United Kingdom, health professionals should be guided by the Data Protection Act (1998) and by principles on information sharing and good practice, as set out within GMC[16] and Royal College of Psychiatrists[17] guidance. The Crime and Disorder Act (1998) allows for information to be passed on, in the absence of consent, in cases where the courts request information about a specific case or if the health professional judges there to be a significant risk of harm to the woman, her children, or to someone else if that information is not passed on. The health professional should always inform the woman if they intend to breach confidentiality, they should properly record their reasons for doing so and they should only pass on the minimum information required to achieve their objective. Further specific guidance on sharing information in the context of domestic violence has recently been provided by the Home Office.[18]

Perpetrators of domestic violence

Studies of men who abuse their partners are difficult to interpret, largely because of the difficulties in identifying and recruiting men who are representative of the population as a whole. There is little evidence that domestic abuse is primarily attributable to underlying mental illness in the perpetrator. However, personality profiles of perpetrators indicate high rates of personality disorder, as well as alcohol and substance misuse and jealousy. There are two main personality profiles described in a perpetrator of domestic violence. The borderline/emotionally dependent type tends to confine their violence to within the family, they tend to be extremely insecure, jealous, and dependent on their partner and the violence is often precipitated by actual or threatened separation. The antisocial/narcissistic offender, is violent both within the family and outside and their violence is often associated with alcohol and drug misuse and high rates of criminality.[19,20]

Group treatment programmes, aimed at changing the behaviour of men who batter, have had a degree of success in reducing violence.[21,22] However the evidence is limited and results are hard to interpret, given the fact that many men, arguably those who are at highest risk and who most need behaviour changing, are least likely to seek help or to remain in treatment. Treatment appears to be most effective when men are mandated through the Courts. Elements of such programmes include: encouraging men to take personal responsibility for their violent behaviour; increasing their awareness of the dynamics involved in the use of violence in relationships; challenging attitudes and beliefs around the use of

violence in relationships between men and women; and developing skills for relating non-violently to others. Perpetrators of domestic abuse are encouraged to accept responsibility for the violence and to consider the consequences of their violence and the gains and losses it entails. Some perpetrators may need to learn particular skills to manage situations where they may previously have resorted to violence. It is important that any treatment programme of perpetrators monitors the effectiveness of the programme through maintaining constant contact with female partners in order to confirm whether participation is having the desired effect and, more importantly, is not endangering them further.

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11.13

The impact of criminal victimization

Gillian C. Mezey and Ian Robbins

Epidemiology

The prevalence of crime depends on the methodological approach that is adopted, the questions being asked and the population being surveyed. Crime figures are also affected by the willingness, or unwillingness of individuals to declare themselves as victims. This is particularly the case with 'sensitive' crimes, such as domestic and sexual violence. Not surprisingly, the self completion phases of the British Crime Surveys have been more effective in identifying such experiences than standard survey methodology. In addition, not all violence is necessarily recognized as a crime, and similarly not all crimes are necessarily defined as such, by their victims. Occasionally, violence may occur by mutual consent.

In the UK the British Crime Survey is regarded as the most reliable and comprehensive data source on criminal victimization, providing information about the extent of crime, as well as trends in the frequency and patterns of crime and changes in public attitudes to crime over the years. The British Crime Survey for 2005–2006 found there were approximately 10.9 million crimes against adults living in private households.[1] Not all individuals in the population are at equal risk. Most crime differentially targets and damages individuals who are poor, disempowered, and marginalized within society. The risk of victimization is highest for divorced single or separated individuals between the ages of 16–24. Men are at greater risk of experiencing violent crime than women, except in the categories of domestic violence and sexual assault, where women are more at risk.[2] Women are more vulnerable to domestic violence and younger people are more at risk of crime than elderly people. Alcohol is also involved in a significant number of violent offending both offenders and victims having been found to be inebriated or to have recently consumed alcohol at the time of the offence.[3] Severe mental illness also appears to be a risk factor for crime victimization. In her study of 936 patients with severe mental illness living in the community, Teplin[4] found that over one quarter of them had experienced violent crime in the past year, a rate more than 11 times that of the general population, even after controlling for socio-demographic variables.

Criminal victimization can have profound psychological and emotional effects, with the impact of violent (including sexual) victimization being greater than property or non violent offending. The experience of crime and the perception of crime as possible or probable, also has an impact on the individual's fear of crime, and on their lifestyle. Women and the elderly are most fearful of crime, even though, in reality, young men are at greater risk. This may be because of the greater perceived adverse consequences of victimization and greater vulnerability in women and elderly people.[5]

General effects of victimization

The experience of victimization can leave the individual feeling 'diminished, pushed down, exploited and invaded'.[6] Victims are often describe feeling stigmatized and isolated and unable to communicate their distress or feelings of vulnerability. Although friends and relatives may initially be supportive, such support may begin to fall away if the victim fails to recover within a reasonable period of time.

Social support and gender are important predictors of psychiatric problems, including PTSD, in crime victims.[7] Andrews *et al.*[8] found that, women were more likely to receive negative responses from family and friends following violent crime and also to have higher rates of PTSD at 6 months follow up. The benefits of positive social support and impact of negative social responses were greater for women victims than men. Negative support was predictive of PTSD in both men and women, although this effect was more pronounced in women.

Immediate and short term effects of crime

High rates of dissociative symptoms are reported by victims of violent and sexual assault in the form of numbing, reduced awareness, derealization, and depersonalization, at the time of the crime.[9,10] Dissociation may have immediate survival value, in terms of reducing the victim's sense of immediate threat and minimizing the pain. However, it may also interfere with the individual's longer-term recovery. Peri-traumatic dissociation at the time of the offence has been found to predict post-traumatic stress disorder development in women victims of violent and sexual assault.

Dissociative symptoms are part of the diagnostic criteria for Acute Stress Disorder which is a risk factor for the development of Posttraumatic Stress Disorder. Brewin *et al.*[11] found that a diagnosis of Acute Stress Disorder at one month post trauma, predicted 83 per cent of post-traumatic stress disorder cases at 6 months follow-up. Acute Stress Disorder may be experienced during or

immediately after a trauma and should resolve within four weeks of the conclusion of the traumatic event.

Long term psychological effects

Most crime victims are able to resume normal functioning and health, following a transient state of disequilibrium, without the need for medical or psychological intervention. However, some victims go on to develop chronic and persistent psychological or psychiatric problems.[12] This may include increased rates of depression, anxiety and substance misuse. Recovery following criminal victimization is largely dependent on how the victim processes and makes sense of what has happened, whether the act can be accommodated into an existing frame of reference or whether the experience is so overwhelming and outside ordinary everyday experience as to render them incapable of reaching some kind of resolution.

Amongst crime victims, victims of violent crime have higher rates of psychological disturbance than victims of property crime who, in turn, have higher rates of disturbance that non victims.[13,14,15] Perception of life threat, physical injury and completed rape are associated with particularly high rates of PTSD.[14,16,17]

Many of the psychological responses exhibited by victims and witnesses of crime fit within a post-traumatic stress disorder framework. In a study of 391 women victims, 27 per cent of all crime victims developed post-traumatic stress disorder.[18] Although most victims of crime show substantial improvement up to 9 months after the offence, very little spontaneous recovery occurs thereafter[19,20] and for some victims the effects are profound and long lasting.[18]

Physical health effects of crime

In general, people who have experienced crime have a poorer perception of their physical health and physical functioning and experience more chronic medical conditions than non victims. Physical and sexual assault are associated with increased cigarette consumption, alcohol and other drug abuse, self neglect, risky sexual behaviour, and eating disorders.[21] Shepherd and Farrington[22] have suggested that a young man from a deprived urban area may suffer 60 years of incapacity as a result of injury, reduced quality of life, and self esteem. Increased crime rates are found in poorer areas, which means that the negative impact of crime on physical health may be difficult to disentangle from the negative impact of poverty and deprivation on physical health.

Responses following specific criminal acts

(a) Murder

The act of murder has profound effects, not just on the individual victim, but also on the friends, family members and acquaintances who are left behind and who are sometimes referred to as the 'secondary victims.' The act of murder is shocking in its finality and irrevocability, and the responses of survivors are both qualitatively and quantitatively different from the normal grieving process.[23,24] Rock[25] has suggested that it is not just the death itself, but the manner of death and its social meaning that is so devastating for those who are left behind. Unlike a 'natural' death, survivors are unprepared for their loss, there can be no anticipatory mourning, no reconciliation, and no proper leave taking. Many survivors describe feelings of stigmatization, isolation, shame, and betrayal, but feel unable to communicate their distress or to connect emotionally with fellow beings. They often feel marginalized by the criminal justice system, with little access to information and they are burdened with having to cope with the inevitable, legalistic bureaucracy and the practical demands of life during a period of acute distress and emotional turmoil. In cases where the perpetrator and the victim are members of the same family, the survivors may experience particularly intense feelings of guilt and conflicting emotions.

The effects of violent traumatic bereavement on the secondary victims include physical health problems, cognitive impairment, and psychological effects, including posttraumatic stress disorder, depression, phobic avoidance, and impaired work and social functioning.[26,27] Female gender and losing a child predict worse psychiatric outcome.[27] Survivors of homicide, tend to manifest both trauma symptoms and symptoms of grief, phenomena, with either predominating or appearing intermittently.[28] This has lead to a proposed new diagnostic category of 'traumatic grief', which contains two core components; trauma and of loss.[29]

(b) Rape and sexual assault

The definition of rape varies across countries and between states within countries. In the UK, prior to 1994, the definition of rape was restricted to penile penetration of the vagina, with other forms of non consensual penetrative sex being defined as indecent assault. However, in 1994, the definition of rape was extended to include non consensual anal intercourse, thereby recognizing male rape victims for the first time. The 2003 Sexual Offences Amendment Act further broadened the definition of rape to include penetration of the mouth as well as penetration of the vagina or anus by the penis. It also introduced three new measures on the issue of consent: first, that a person can only consent to sexual relations if they have the freedom and capacity to make that choice, second, that all the circumstances at the time of the offence must be considered in determining whether the defendant is reasonable in believing the complainant consented and third, that individuals will be considered most unlikely to have agreed to sexual activity if they were subject to threats or fear of serious harm, if they were unconscious, drugged or abducted, or if they were unable to communicate because of a physical disability.

In the majority of cases of rape, the perpetrator is known to the victim and in many cases, the rapist is the current or former husband or partner.[30] In spite of the seriousness of the offence, the British Crime Survey[30] found that only 60 per cent women who had been subjected to rape or serious sexual assault had told anyone about it and only around one in seven cases had been reported to the police. Reasons women gave for not reporting include: fear of reprisals, fear of public identification, fear of appearing in Court and having to give evidence and lack of confidence in the legal system.[31] There is some evidence suggesting that women who proceed with prosecution following rape do worse, in terms of social adjustment and self esteem at one year follow up than women who decide not to proceed.[32] Whether this is because Court proceedings delay or slow down the process of psychological recovery following rape, or because the legal process and particularly the experience of being cross examined in Court, represents a form of 'secondary traumatization', is not entirely clear. If women do proceed with the Criminal Justice process, however,

preparing them for the experience, providing them with appropriate information beforehand and giving them the opportunity to exercise choice, can help to offset the potentially de-stabilizing and distressing impact of criminal proceedings. Ultimately, the attitude taken by the police and the way the victim feels they have been treated appears to be more important in determining their psychological adjustment and satisfaction with the process, than the actual verdict.[32]

About one-third of women who report rape develop long term psychological and social problems. These effects tend to be more severe and chronic than following non sexual violence.[33,34] Rape trauma syndrome was first described in the 1970's[35] and was subsequently superseded by Posttraumatic Stress Disorder. Posttraumatic stress symptoms are generally present in the days and weeks following the assault, but then spontaneously resolve in the majority of cases. For some victims, however, the condition may become chronic and persist for many years, if left untreated.[36] Higher rates of depression, suicidal ideation, generalized and phobic anxiety, alcohol and drug dependence and sexual dysfunction as well as physical health problems are also found in rape victims compared with non rape victims.[37–41]. Women who have been raped often describe problems in relationships, with excessive dependence, inability to trust and loss of confidence and self esteem. Similar responses have been described with male victims of sexual assault.[42]

The characteristics of sexual assault that predict long term mental health problems are: being the victim of a completed rape, being injured and the perception of a threat to life.[14] Other predictors of long term disturbance include; prior psychological and social problems previous victimization, particularly childhood abuse past psychiatric illness, drug or alcohol misuse and lack of a supportive network.

Psychiatric treatment may be required for individuals who develop serious psychological problems following a sexual assault. It is probably inappropriate to embark on psychiatric treatment too early, because of the natural tendency for symptoms to resolve spontaneously in the weeks and months following the assault. Unless symptoms have resolved by 6 months, however, they are unlikely to resolve spontaneously thereafter without some form of psychological intervention or psychiatric treatment (see Chapter 4.6.2)

There is no evidence that counselling is effective in alleviating short term distress or in preventing the development of long term psychiatric disability in rape victims. Indeed recent studies have suggested that counselling may even be harmful, if carried out by inadequately trained and supervized individuals.[43] Many of the key organizations working with victims e.g. Victim Support, emphasize their role as supporters and befrienders and they provide both practical assistance, such as accompanying victims to identification parades, helping with paperwork and compensation claims, as well as offering emotional support following the assault and through any subsequent criminal proceedings. Rape victims are most likely to benefit from services that are co-ordinated, integrated and streamlined.[43] An example of this in the UK has been the development of Sexual Assault Referral Centres (SARCs), which provide a 'one stop shop' of medical, counselling, legal and forensic services for victims of sexual assault. Evaluation of these centres and in particular the benefits for victims and the Criminal Justice System, is ongoing.

(c) Burglary and robbery

The effects of burglary are generally less severe and long lasting than following violent and 'contact' crimes although some victims may develop chronic mental health problems, including Posttraumatic Stress Disorder.[18] Repeat victimization is especially common in the case of burglary and second or subsequent burglaries are more likely to have a greater long term impact. Repeated experiences of burglary may lead to victims of burglary taking additional security precautions or even moving house in order to restore a sense of safety and control over their lives.[44] Individuals living in areas of poverty and deprivation and single parent families are most vulnerable to burglary. Robbery, unlike burglary involves not only direct contact between the victim and the perpetrator, but also implies a degree of life threat and is therefore more likely to precipitate post-traumatic psychiatric illness, including post-traumatic stress disorder.[45]

(d) Workplace violence

Violent assaults in the workplace have increased in frequency and severity in recent years.[46–48] Budd *et al.* found that working at night was the only significant factor which predicted its occurrence. There was a clear relationship between workplace violence and increased job stress, reduced job satisfaction, increased likelihood of looking for a new job, as well as bringing weapons to work Kopel & Friedman[49] found that police officers witnessing incidents of violence reported intrusive thoughts and images, and used avoidance to deal with the intrusive phenomena. Whilst recognizing that avoidance is a feature of post-traumatic stress disorder, they also suggest that denial and avoidance are part of the culture in male-dominated occupations such as law enforcement agencies. Miller-Burke *et al.*[50] found that most employees who had experienced robberies had multiple adverse consequences. Psychological functioning, physical wellbeing, social and occupational functioning were all impaired. Having been involved in more than one incident was associated with more severe outcomes.

In the United States Hewitt and Levin[51] point to the high rates of occupational assaults among health-care workers. Whilst fatal workplace assaults are more likely to involve males in the course of robberies, women were more likely to be involved in non-fatal workplace assaults, with health-care workers being most affected. The rate for health and social care workers was 10 times that of private non-health care industries. Williams[52] found that 26 per cent of nurses reported physical assault while at work. This rate of assault is similar to that found by O'Connell and Bury[53] who carried out a survey of all general practitioners in the Eastern Health Board of Ireland. They had a 98 per cent response rate which revealed that 21 per cent of general practitioners had experienced violence or aggression although in only 7 per cent of the incidents reported did it result in injury. Not surprisingly there is a strong inverse relationship between workplace assaults and job satisfaction.

(e) Child and adolescent victims of crime

Children and young people are especially vulnerable to crime and victimization, in particular by people they know and are dependent on, although stranger violence is a small but important problem.[54] The 1992 British Crime Survey[55,56] looked at the victimization of young people away from the home and reported that about one-third of 12 to 15 year olds had been assaulted in the last 6 to 8 months. This would compare with a rate of around 1 per cent in

adults. Most of these assaults happened at or near school and were committed by a sole perpetrator who was known to the victim. The survey also identified high rates of fear of crime amongst adolescents, with girls being more fearful than boys. A range of mental health problems have been identified following victimization within the child and adolescent population.[57]

Hate crimes

The defining characteristics of a hate crime is that the individual victim is targetted because of bias or prejudice, based on their actual or perceived social grouping or ethnicity, sexual orientation, religion or political orientation. Herek *et al.*[58] suggest that a significant proportion of lesbians and gay men who had been assaulted believed that their sexual orientation had been a motivating factor. One-quarter of men and one-fifth of women said that they had experienced victimization because of their sexual orientation. When compared to other crime victims, they had significantly more symptoms of depression, anxiety and PTSD as well as more crime related fears and a lower sense of mastery. Rose & Mechanic[59] reported that 73 per cent of lesbians and gay men had experienced at least one homophobic attack. Victims of homophobic violence had more PTSD symptoms than did victims of other homophobic crimes or non victims although there was no significant difference in rates of depression. They also found that homophobic sexual assaults were more likely to involve known assailants, and multiple perpetrators and more likely to be repeated.

Hate crimes tend to be under-reported to the police, the psychological effects tend to be long lasting, and have a negative impact not only on the individual victim, but also on the community. It has been argued that hate crimes require policy directed to addressing the causes rather than simply dealing with the needs of individual victims. Black and ethnic minority individuals appear to be particularly vulnerable to criminal victimization and in a proportion of these cases the offence is considered to be 'racially motivated'. [60,61]. Around one in six of all incidents of criminal victimization against Asians and African-Caribbeans are considered to be racially motivated[61] and, regardless of the type of offence, ethnic minority victims tend to report higher levels of worry about crime then white victims.[60] Fear of crime appears to be particularly high amongst Bangladeshi and Pakistani individuals, who are also most likely to describe their victimization as racially motivated. It has been suggested that victims who perceive their victimization to be racially motivated, experience more serious and persistent psychological effects that individuals who did not consider racism as a motivation.

(g) Terrorist crimes

A number of studies have looked at the impact of terrorist attacks which, whilst relatively infrequent, in comparison to other crimes, have a considerable political and social impact. Whalley and Brewin[62] reviewed the mental health effects of terrorist attacks on the direct victims, as well as on the general (non affected) population. In the general population, symptoms of distress and stress are high in the first few days following an incident, but tend to resolve spontaneously so that, although PTSD is found in between 11 per cent-13 per cent of the general population, in the first six weeks following a single terrorist attack, this falls to below 3 per cent after 8 weeks. Factors such as previous adversity, pre-existing mental health issues and membership of minority groups may increase vulnerability to the impact of terrorism. PTSD is the most common psychiatric disorder in direct victims of terrorist attacks, followed by depression although other problems such as traumatic grief, panic disorder, phobias, generalized anxiety and substance misuse have also been reported.[63] Most studies have reported that between 30–40 per cent of those closest to the site of the attack, will develop a psychiatric disorder within 2 years. However, the majority of direct victims have no contact with mental health professionals. In the aftermath of the September 11th attacks in New York only around one-quarter to one-third of those suffering from PTSD were in receipt of treatment. Whalley and Brewin[64] suggest that a focused outreach approach such as a 'screen and treat' programme may be needed to identify those with significant impairment and help them to access evidence based treatments.

The extent of physical injury during a terrorist attack is the best predicator of post-traumatic stress disorder rates both in the short term[65] and many years after the event,[66] although it does not predict the development of a depressive illness. Studies which have looked at the impact of shootings tend, by their very nature to be small scale, but have found significant levels of distress and high rates of post-traumatic stress disorder and other psychiatric disorders.[67] Being held hostage has also been related to high levels of distress both in victims and their families.[68] Where captivity has occurred, there may be strong attachment and paradoxical gratitude towards the captors with positive emotions including compassion and identification with the terrorist's values, often described as the Stockholm syndrome.

Support services and treatment interventions

There are so few culturally accepted rituals of support for victims of crime, that it often becomes the task of the therapist to normalize the process. In the United Kingdom victim support schemes offer practical assistance, for example accompanying the victim to identification parades and court hearings, completing Criminal Injuries Compensation Board forms, as well as providing support and reassurance following crime. Referrals to the schemes are generally made by the police, but are occasionally accepted from involved professionals or from the victims themselves. However, victims of serious crimes, such as sexual assault or physical violence, and the families of murder victims may develop psychiatric illness, which requires referral to mental health services for specialist treatment.

Treatment approaches are drawn from a variety of paradigms including cognitive behavioural, psychodynamic, psychosocial, and pharmacological treatments, and are often trauma focused in general rather than being specific to problems associated with criminal victimization. Ochberg (6) categorizes them into two main approaches, the first focuses on previous personality and attributes mental health problems and difficulties in adjustment following crime to pre-existing unresolved issues and weaknesses, rather than to the traumatic events. The second approach focuses more on the events themselves, the individual strengths and coping styles of the victim and setting realistic achievable goals.

In the UK the 1998 Crime and Disorder Act placed an obligation on the NHS to work in partnership with the Police and Local Authorities in dealing with the consequences of crime. Many people who present to the NHS for treatment following violent crime

do not present to the Police or criminal justice agencies. As many of the services for crime victims are organized by or accessed through the criminal justice system these people are likely to miss out on services. There is a clear need for a direct relationship between the NHS and criminal justice agencies in the way in which treatment is provided and its relationship to the criminal justice processes but as yet, other than in a small number of specific projects it does not happen.

Often immediately following victimization help is offered and family and friends rally round. This may not be the time when most people need formal psychiatric help. The majority of people post assault experience sympotms of PTSD and other psychiatric problems but also the majority improve spontaneously even in the case of serious crimes such as rape. The question comes as to when to intervene. The National Institute for Clinical Excellence (NICE, 2006) recommend a position of watchful waiting and if in the first month(s) symptoms are not improving or indeed may be increasing then treatment is indicated otherwise waiting and monitoring to identify who will not spontaneously improve is the approach of choice.

Previous work had already shown that timing of appropriate services is important. Shepherd *et al.*[62] have shown that while accident and assault victims have similar levels of depression and anxiety in the immediate aftermath three months later the assault victims have higher levels of symptoms. In the immediate aftermath of criminal victimization it has been suggested that having the opportunity to talk may reduce later symptoms Where there have been specific RCTs of immediate intervention they have not shown themselves to be particularly effective. Rose, Brewin, Andrews and Kirk[63] compared an education intervention with education plus psychological debriefing or an assessment only condition in a randomized controlled trial. They found that while all groups improved over time there were no significant differences between groups. This is hardly surprising when considering the result of Rose, Bisson & Wessely[64] who carried out a systematic review of RCTs examining the impact of debriefing. They found 11 RCTs of which six showed no effect at all, three had a positive effect and two had a negative outcome.

This has led to a belief that there is no point in any immediate interventions but this ignores the contribution made by organizations such as Victim Support or Rape Crisis centres. They do not offer formal therapy but rather give information, offer the opportunity to ventilate emotions, practical help and assistance in making compensation claims or participating in the criminal justice system. Shepherd and Bisson[65] who make the case for more integration between health services and other agencies describe the two models of Victim Support interventions which are initiated within the NHS and address the needs of victims which would otherwise be overlooked.

In terms of formal psychological therapies and in the longer term Cognitive Behavioural Therapy has been used with great effectiveness. Ehlers & Clark[66] reviewed the use of CBT in early interventions. They concluded that CBT was superior to supportive counselling but that brief CBT in the first month showed no superiority over repeated assessments. Where longer programmes ie 16 session were offered in the first 4 months they found that CBT was superior to supportive counselling, repeated assessment or no intervention at all.

There have been a number of other empirical studies demonstrating the effectiveness of structured cognitive behavioural approaches to treating established PTSD, which are discussed further in Chapter 4.6.2

Conclusion

The impact of crime on the individual victim is profound but is frequently underestimated by mental health professionals. Wide-ranging personal, social, and economic consequences could be prevented if a range of appropriate interventions were available. Most post traumatic stress treatment programmes in the United Kingdom have developed in response to specific disasters, which may not be relevant to or as effective with crime victims. In order to provide appropriate treatment to crime victims, mental health professionals need to recognize the importance of active interagency liaison with the police, the courts and with voluntary organizations such as victim support schemes. Crime victims tend to be relatively invisible and disempowered; they are less likely to be supported by active campaigning groups than survivors of major disasters and, because of associated feelings of shame and stigmatization, they may be reluctant to claim their entitlement to proper care and treatment. The fact that their plight is often used as a political football is likely to reinforce feelings of helplessness and insecurity. Given its prevalence, crime represents both an ordinary and an extraordinary event; it is likely to affect everyone at some point in their lives and the fact that most crime victims recover from the experience should not deprive those who need it, to proper care.

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11.14

Assessing and managing the risks of violence towards others

Paul E. Mullen and James R. P. Ogloff

'Prediction is very difficult, especially about the future'

Niels Bohr (1885–1962)

Introduction

Assessing and managing the risk of our patients being violent towards others now occupies a prominent position in virtually all forms of mental health practice, but it remains a contentious area. At the highest level researchers, psychometricians, and statisticians argue about almost every aspect, even whether anything useful can be said about individual outcomes rather than group indicators. At the next level an industry flourishes of selling training, and risk assessment tinstruments, to those who then appear as experts in a wide range of mental health and criminal justice contexts. On the ground, almost everyone in mental health is drawn into filling out purpose-designed forms and complying with protocols, most of little or no demonstrated validity. This chapter is intended to make clinicians aware of both the possibilities and limitations of existing approaches to the assessments of risk. Given that there is no reason for mental health professionals to evaluate risk without gaining information to manage it, this chapter will also address the management of risk for aggression and violence.

Constructing risk

A critical analysis will be attempted of how risk has come to be constructed in our society and how this is impacting on mental health and criminal justice. When an approach is adopted which attempts to reveal the foundations and historical evolution of a widely accepted social construct, like risk, there is a danger that it will appear overly sceptical or even mocking. It is important to emphasize at the outset that:

1 The assessment of the probability of patients behaving in ways damaging to others and the management of that risk is a legitimate clinical activity.

2 That attributions of levels of risk to a patient occurs in a social and cultural context and is inescapably a construct.

The discourses around the dangerousness of the mentally ill have gradually been replaced by those of risk. This change is usually presented as a product of the progress of knowledge and the improved conceptualization of that knowledge.[1–4] The language of danger transmuted into the language of risk also emphasizes the probabilistic nature of risk assessment. We are not now and probably never will be in a position to be able to determine with certainty who will or will not engage in a violent act. Relying on a range of empirically supported risk factors, though, we can makes a reasoned determination of the extent to which those we are assessing share factors that have been found in others to relate to an increased level of risk. Risk embodies the interaction of a range of factors, which are not necessarily dangerous in themselves, such as age, gender, marital status, ethnicity, employment status and, of course, mental disorder.[5] Risk factors can be any variables which are statistically associated with a future violent episode or event. There is no assumption of causality linking the predictor to the predicted.

Risk assessment came relatively late to the mental health field. Not until 'harm to others' or 'undue risk' became criteria for involuntary hospitalization and forensic detention did 'dangerousness' of patients assume the spotlight. The focus on risk first surfaced in Western Societies in the 1970s in the context of concerns about damage being inflicted on individuals by the actions, or inactions, of corporate and governmental agencies. These concerns fed the emergence of widely based environmental movements as well as an escalating number of class actions and individually driven litigations. Under the banner of risk a new blaming system emerged of which Douglas[6] writes 'we are . . . almost ready to treat every death as chargeable to someone's account, every accident as caused by someone's criminal negligence, every sickness a threatened prosecution. Whose fault? is the first question . . . then what damages? what compensation? what restitution . . .' (pp. 15–16). One response to this culture of blame has been the emergence of what O'Malley[7] refers to as a new prudentialism in which individuals, professionals and corporations, increasingly held responsibility for the impact of their actions on others, resort to risk management strategies in which risk is assessed, managed, insured against, and where possible removed.

Psychiatrists and psychologists are among those who have become caught up in the 'culture of blame'. Any damaging or distressing occurrence which is experienced by, or caused by, someone who is, or has been, a patient of the mental health services, is transformed into a preventable tragedy for which professionals are

to be held responsible. Rose[8] suggests the new imperatives of risk assessment and risk management operate to establish mechanisms to control mental health professionals which through standards, audits and enquiries not only regulate professionals but hold them personally responsible for unwanted outcomes. Douglas (6) argues 'probability analysis arrives at politics in the form of a word 'risk' . . . the word gets its connection with probability squeezed out of it and put to the same primitive political uses as any term for 'danger" (p. 48). Risk assessment and risk management are concepts which have the potential to shift blame towards clinicians who have failed to follow procedure and away from managers who fulfilled their responsibilities by ensuring correct protocols were in place, irrespective of the possibility of the realistic application of such protocols. The language of risk can also shift the focus from politicians who determine resources and establish systems of care to those who fail to identify and manage risk in the individual case. Perhaps most importantly the increasing centrality of risk assessment potentially creates a vision of the mentally disordered as primarily embodiments of varying degrees of risk and the mental health services as agents in controlling and obviating the supposed danger to the community.

Assessing dangerousness used to be the almost exclusive province of the forensic mental health professional.[9,10] It was a marginal activity based on arcane knowledge and assumed wisdom that only experience could provide.[11,12] Risk assessment and management in contrast have become central to current mental health practice in almost all its guises. It has become among the most important activities defining professional competence. Understanding the cultural, legal, and political roots of the increasing hegemony exerted by the rhetoric of risk over psychiatric practice may demystify, but does not free the professional from the imperatives of operating effectively in this new environment. It seems so obvious, as to be self evident, that mental health professionals are expected to consider the probability that their patient will act in a destructive manner and to act to prevent such harms. But the self evident is often the ideological unconsidered. It is not obvious that a mental health professional's primary responsibility is to the wider community rather than their patient. It is not obvious that it is possible to effectively predict such risks as they apply in the individual case. It is not at all obvious how we should act in the face of a prediction of risk, and it is certainly not obvious that such concerns should be a major determinant of our approach to patients.

Words are rarely innocent. Risk is not the same as probability, for risk implies a degree of danger. Even those deemed to be at 'low risk' still appear to present some degree of danger. No one, it seems, is considered risk free. Risk management is not the same as harm minimization, for it promises a prevention of unwanted outcomes. Psychiatry deals with disorders which have both substantial morbidities and mortalities. Good management may reduce but cannot, in our present state of knowledge, prevent all such morbidity and mortality. Furthermore, reducing morbidity and mortality long term may only be possible at the price of accepting an increased probability of mortality in the short term. Suicide in prison, for example, can be prevented by isolating and observing vulnerable inmates in transparent plastic bubbles, bereft of features from which suspension is possible, or by the simple expedient of chaining them hand and foot to a bed (both strategies are in use today). If the only good is preventing self harm such draconian measures acquire currency irrespective of the psychological damage and abuse of basic human dignity involved. Moreover, as the aim has become removing all risk of suicide in prison, more and more vulnerable prisoners are being subjected to such restrictions and in many jurisdictions 'witch hunts' now regularly follow every death across jurisdictions. We cannot force the genie of risk back into the lamp. Mental health professionals will continue to be made publicly accountable. Professional self regulation is being replaced by statutory regulation and the ravages of civil litigation.[13] As Rose[8] notes we will be forced to play a central role 'in the strategy of reducing risk and minimizing harm under threat of sanction and within the disciplines imposed by a plethora of practices of blame' (p. 18).

Contemporary approaches to risk assessment

It is no longer possible for mental health professionals to distance themselves from the process of risk assessment. Throughout the 1970s and 1980s mental health professionals were almost of one voice in proclaiming both their inability to predict dangerousness and the basic pacifivity of the mentally disordered.[14] Despite this public stance dangerousness criteria came to dominate civil commitment, with courts simply ignoring arguments that the prediction of dangerousness was beyond the ken of psychiatrists and psychologists.[15] In addition liability based on failures to predict dangerousness was established in landmark cases including those arising out of Poddar's killing of Tarasoff[16] and Hinkley's attempt to assassinate President Reagan.[17] Last, but not least, the criminal courts increasingly suborned mental health professionals into predicting dangerousness in the pursuit of such sentences as preventive detention and death.[15,18] In the US and the UK the emergence of the language of risk at the end of the 1980's was in part a recognition of the centrality assumed by predicting and managing the potential for violence perceived by the public to reside in the mentally disordered. In the UK the public inquiry into the killing of Jonathan Zito by the psychiatrically disordered Christopher Clunis set a pattern for future homicide enquires.[19] Failures of risk assessment and management by individual practitioners, together with inadequacies of communication and service provision, were identified as major contributors to 'avoidable' killings.[19] Such developments set risk assessment at the very centre of the mental health agenda.

Given the challenges of risk assessment, what is the current state of knowledge and can it be of assistance to clinicians? The limited research available on 'dangerousness prediction' conducted in the 1970s showed that psychiatrists and psychologists had unacceptably low levels of accuracy in predicting which patients would go on to be violent in the future.[20] It was found, perhaps not surprisingly, that psychiatrists, psychologists, and release decision-makers tended to make conservative decisions that suggested that people were at risk for dangerousness or violence when, in fact, they were not. Similar findings have been obtained over time. For example, Belfrage[21] found that clinicians found 90 per cent of a group of 640 offenders sentenced to psychiatric treatment in Sweden to be at 'risk of severe criminality;' when, in fact, only 50 per cent went on to commit any kind of crime.

Reasons given for the errors made in risk prediction include clinicians' confusion and lack of knowledge of valid risk markers

and risk factors. In addition, as with other areas of decision-making, even if clinicians do have a reasonable understanding of risk factors, it is difficult to systematically consider them and to put together a risk appraisal in a systematic way. Advances in risk assessment have included the identification of an expanded range of predictor variables relevant to violence. Most important among these are those variables that are subject to change (i.e., they can change over time and they can be influenced by treatment or other intervention). Generally speaking, risk assessment variables can be classed as 'static' (i.e., those that cannot be changed) and 'dynamic' (i.e., those that can change over time). Actuarial risk schemes, which will be discussed later in this chapter, are based upon variables that were measured from the past. These historic variables generally could not change over time. For example, if one began being violent as a young person, that fact will not change over time. Dynamic variables, in contrast, are subject to change over time, sometimes rapidly. These variables include such things as state of mind, situational factors, attitudes, plans, support, etc. Effective risk assessment must take into account both static and dynamic variables; however, risk management generally requires an understanding of the dynamic risk variables. Contemporary approaches to risk assessment and management take into account both static and dynamic variables, thus considering an individual's past, present, and future risk factors that might affect the likelihood of him or her becoming violent.

There has been considerable progress since Monahan[20] first reported that psychologists and psychiatrists were essentially unable to predict risk to any acceptable extent. Current research shows that risk assessment approaches provide a level of accuracy that now far exceeds chance.[22]

The limits on mental health professionals' engagement in risk assessment

There are two legitimate perspectives on risk assessment:-

1 The clinician whose work involves considering patients' levels of risk as part of a process whose purpose is primarily to improve the management of patients.

2 The forensic evaluator for whom risk assessment is a tool to improve the reliability of opinions provided to courts and tribunals charged with making decisions about an individual.

Forensic mental health professionals in the US and Canada tend to see their role primarily as evaluator. It is not uncommon for forensic psychiatrists and psychologists to primarily conduct assessments for the courts as their means of employment. This reflects the generally accepted separation of the assessment and treatment roles. In stark contrast in the UK and Northern Europe the clinician perspective dominates even for forensic specialists. Relatively few forensic clinicians would operate as court evaluators alone, though some like their US colleagues would try and separate the roles.

This chapter is written for mental health professionals in a wide range of contexts, not for forensic specialists. As a result the emphasis on this chapter will be on the clinical perspective. North American readers should, however, keep in mind that this perspective is not shared by many specialists in the forensic field there who would consider it not just problematic but ethically questionable to mix the assessment and treatment roles.

Boundaries need to be drawn around when, where and for what purpose, mental health professionals can ethically engage in assessing the probability of an individual committing violent or criminal acts. There are somewhat different constraints operating on clinicians than for those carrying out evaluations for the purpose of preparing reports for decision-making bodies like courts and tribunals.

The ethical and practical constraints on a clinician assessing risk entirely in service of effective treatment include:

1 Ensuring the assessment serves the interests of the patient in terms of improving management and protecting them from acts which will damage their interests. The seriously mentally ill when they become violent all too often target those who support and care for them thus destroying the relationships critical to their own social survival. In addition, criminal and violent acts of the sort usually associated with the mental disordered rarely, if ever, brings anything but increased problems for the patient. Thus, reducing the likelihood that the patient will engage in criminal and violent acts serves not only the public interest but also the patient's interests.

2 Mental health variables (which include psychological variables and personality traits) are a prominent feature of the individual's clinical picture and are also of potential relevance to the probability of future damaging behaviours.

3 Avoiding providing greater emphasis to risk than is necessary given the totality of the patients needs and vulnerabilities.

4 The assessment should wherever possible connect potential risk to those factors whose amelioration will reduce that risk. Ultimately a health professional can only justify engagement in risk evaluation if they lead to better outcomes for the evaluated as well as the community. Risk assessment finds its ultimate justification in risk management.

5 Avoiding using risk and risk assessment to disqualify patients from access to the treatments they require. Increased risk requires increased therapeutic enthusiasm, not rejection.[23]

6 That any concerns raised by a risk assessment are shared with the patient and the proposed management strategies explained. Even unwelcome restrictions are resented less if the reason for their imposition is explained.

7 Retaining an awareness of the limitations of the predictive power of risk assessments and the need to ensure proportionality between the risk actually apprehended and any imposed remedy.

8 Ensuring a level of professional competence adequate to the task.

9 Making use, where possible, of the skills and knowledge of the multidisciplinary team in the assessment.

Those obligations on those engaging in risk assessment as part of an evaluation for a decision-making body are even more onerous and should include:-

1 The patient consents to the examination in the knowledge of the nature of the assessment, the purposes to which it may be proffered, and the limitations on confidentiality that may apply.

2 A reasonable body of empirical evidence exists to guide the risk assessment including, where possible, empirically validated structured risk assessment measures.

3 The risk assessment is conducted in consideration of the legal parameters governing the decision-making body (e.g., criteria to be considered for change of orders or release for forensic psychiatric patients) while realizing that the legal questions to be answered never parallel the clinical or evaluative results mental health professionals can reasonably provide (e.g., there is no clinical parallel to legal criteria such as 'undue risk').

4 The assessment is based on a careful analysis of the relevant characteristics of the particular individual which in all but exceptional circumstances have been obtained in part by a direct examination of the individual.

5 The risks are expressed in terms of probabilities (not attributions of dangerousness) with clear admissions of the fallibility and potential variability in the prediction. The problem should be acknowledged of employing risk factors derived from studies on populations from different cultures and contexts. After all nobody would use risk data from Los Angeles to calculate the car insurance premium for a driver in Dublin or Oslo without considerable caution.

6 Account is taken not just of the probability of damaging behaviour but the nature and severity of such conduct. Proportionality needs to be maintained between what is predicted and the response. It is all too easy to employ methods which establish increased risks of a wide range of unwanted behaviours only to find them used to justify draconian and punitive responses which would only be acceptable in the face of the imminence of serious violence.

7 The confidence and certainty with which any prediction is formulated to take account of the implications for the person being assessed. Risk predictions may be offered in terms of probabilities but they will almost always be used to justify all or nothing decisions.

8 That personal and professional integrity is strictly maintained. This is no simple matter when the evaluator is either in the pay of the patient, or of those whose interests are not necessarily those of the patient.

The potential conflicts generated when acting for a patient as both a professional evaluator and a clinician are so considerable that some experts argue that such a dual role is inherently unethical. They argue in preparing reports for decision-making bodies the interaction ceases to be that of health professional and patient, and becomes entirely that of expert, or 'forensisist', and evaluated. The expert's obligation is then not to the evaluated but to their own professional competence and the rules governing the process the report will serve (e.g., criminal court, family court, mental health tribunal).

In our view even in an encounter between a health professional and a person purely for the purposes of an evaluation for a court there remains obligations to the person as patient [patient from the Latin for to suffer, who in this instance suffers the intrusions of an examiner who cannot for the evaluated entirely cast off the guise of physician or healer]. The solution is to learn to live with the contradictions and accept the dialectic between responsibilities to the patient and obligations to the agencies of society. The result of accepting the duality of the role inherent in assessing a patient's risk of harming others does exclude participation in a process that could increase the risk to the patient of fatal (e.g., death penalty evaluations) or serious harm (e.g., sexual predator laws whose sole purpose is justify a process of prolonging incarceration beyond the expiry of a sentence). It does however legitimate having the dual role of clinician and evaluator in some circumstances. In fact those who totally eschew ever taking on such a dual role, we believe, are at risk of deluding themselves that they can caste off the mantle of clinician for the patient, and become a socially neutral, objective, observer and reporter.

The dialectic between the demands of a professional responsible to the health of your patient and the demands of professional integrity and honesty owed courts and tribunals is almost always possible to resolve. To fail to accept engagement with the conflicts which in reality usually exist for evaluators and clinicians reporting to external authorities is in our opinion an act of self deception in which you become an agent either of patient, or authority, and no longer an autonomous responsible professional.

Some situations make nonsense of the above considerations, notably death penalty hearings. One of us (J.O) has extensive experience of working with those on death row, the other (P.M) only a comparatively slight acquaintance. We are both of the opinion, however, that it is impossible to honestly discharge your responsibilities as clinician, as evaluator, or as decent human being, in such circumstances.

Risk assessment approaches

There are five basic approaches to evaluating the risk of violence:-

1 Probability models based on established risk factors. The risk factors can be derived actuarially from studies of particular populations (e.g. Violence Risk Appraisal Guide (VRAG) and Static 99) or rationally ascertained from the risk literature (e.g. Historical Clinical Risk 20 (HCR-20)).

2 Clinical experience based on recognizing previously encountered (personally or in the literature) patterns associated with future violence. The clinical approach is largely relevant to the avoidance of obvious errors like discharging morbidly jealous men who are threatening to kill their partner.

3 A mixture of 1 and 2 where the risk assessment instrument is employed to guide the appraisal of risk factors and clinical judgment is applied to balance idiographic information with the nomothetic variables as in the structured professional judgment approach of which the HCR-20 is the prime example.[22]

4 The strictly idiographic approach which employs individual profiles of violent offenders to detect those on a similar pathway to attack. The idiographic approach is employed to evaluate the risks of rare events, such as attempts to assassinate a head of state, and has little application in general mental health.

5 A plethora of local risk assessment tools have sprung up. Sometimes it seems as if every psychiatric service, probation/parole service, prison, and security consultant have their own unique sheet of questions which are supposed to establish the future probability of whatever particular piece of nastiness currently concerns the organization. These ad hoc parochial risk assessment protocols have no evidentiary basis or psychometric integrity (even if they incorporate aspects of other properly constituted instruments). It is far better to validate existing

empirically supported measures for use in a particular setting and with a particular population.

The core of risk assessment is the systematic application of probability models usually incorporated in standardized instruments.

The utility of risk assessment

Even given a firmly based knowledge of those factors which in populations may increase or decrease the probability of violent behaviour, there remain theoretical and practical limitations on effective prediction in the individual case. If factors were identified which occurred only in the violence prone and never in the pacific and, if present, were in every case the harbinger of future attack, then the power of the predictive paradigm would be independent of both the frequency of future violence in the population, and of variables which effect the factors' expression in the individual case. In the real world the sensitivity (the accuracy with which the outcome is predicted) and the specificity (the extent to which only those who will act in the predicted way are identified) fall short of 100 per cent. This being so the less common the future behaviour in the population the less specific will become predictions. Equally, the more complex the influences affecting the expression of the identified predisposition the less the sensitivity of the predictive paradigm.

Say we develop a predictive paradigm of 70 per cent sensitivity and 95 per cent specificity (which is feasible). If we set an acceptable level for the practical use of such an instrument that it will not unfairly restrict, or stigmatize, more than 1 person for every 4 correctly designated as candidates for future violence, then the base rate for violence in the population of interest would have to exceed 20 per cent. Even if we accepted one error for each correct designation (which, if the outcome of ascertainment were incarceration or other significant curtailment of basic freedoms, few would regard as ethically defensible) it still requires a base rate of higher than 6 per cent.

This hopefully makes clear that if predictions of the probability of future violence are to be used to significantly restrict the patient's freedoms the base rate of the behaviour must be reasonably high in the group under consideration. Equally, if measures such as long term institutionalization or compulsory community treatment with restrictions on residency and movement are contemplated the degree of violence apprehended must be commensurately damaging. It would be difficult to justify such interventions if what is at issue is embarrassing, or even fear inducing behaviour, which does not involve either assault occasioning injury or gross intimidation.

Statistical approaches to analysing predictive efficiency in populations with low base rates for the target behaviour exist, the most commonly employed being derived from a measure developed for use with radar or signal detection systems, the Receiver Operating Characteristic (ROC) curve.[24, 25] The ROC curve is a graph of true positives (sensitivity) along the y-axis and false positives (1-specifity) along the x-axis. With respect to violence, these correspond to patients who were predicted to be violent those who were predicted not to be violent, respectively. The line running from the lower left corner of the graph to the top right indicates chance prediction, where true positives are equal to false positives (i.e., for every patient predicted to be a violent who in fact becomes violent another patient who was predicted to be violent does not become violent). A curve above this line indicates that, in this case, recidivism is being predicted at rates above chance. The Area Under the Curve (AUC), which is represented as a proportion of the graph that falls under the curve, reflects the proportion of true positives over false positive (e.g. an AUC of 70 would indicate that 70 per cent of those predicted to be violent in fact do become violent). The ROC analyses allow the accuracy of predictions to be established independent of variations in base rate.

In many western nations between 5 per cent and 10 per cent of all homicides and more than 5 per cent of serious crimes of violence are committed by those with a schizophrenic syndrome. The annual risks of a person with a schizophrenic syndrome committing a homicide is however, in the region of 1 in 10,000 and for a crime of violence about 1 in 150.[26] This is because serious violence is, the media notwithstanding, an uncommon event. Though fear inducing behaviours occur with distressing frequency among the seriously mentally disordered the inflicting of serious injury is measured in annual risks of below 1 per cent.[27,28,29] This suggests that risk assessment instruments will not be relevant to predicting serious violence in those with a schizophrenic syndrome. Underlying the homicide enquiries in the UK, and the litigation, particularly in the US, however is the assumption that they can be and will be.

Given their particularly low base-rate of occurrence, attempting to predict who will commit serious acts of violence or murder will inevitably be accompanied by vast numbers of false accusations. Further, in reality we often trade off outcome variables, thus avoiding over ready resort to civil commitment may improve the chances of establishing a long term therapeutic alliance, which in turn may reduce long term risks, albeit at the price of tolerating a higher degree of risk in the short term. Avoiding all probability of any patient committing a future act of violence would involve the use of widespread coercion and move mental health professionals into increasingly custodial and controlling roles.

An alternative argument deserves consideration. In those with a schizophrenic syndrome, for example, it may well be feasible to identify the 10 per cent who will perpetrate 90 per cent of all the future fear inducing and violent acts. In this 10 per cent may be included nearly all of the far smaller number who will commit potentially lethal or seriously injurious acts. In effectively identifying the 10 per cent and managing them appropriately then the risks to the community of damage, including the small chance of serious damage, will be reduced. The majority of those so identified will be a nuisance who can occasion fear, and who may push, punch or kick others.[30] Effectively identifying and managing all patients in the high risk group will lower the overall risk to the community while minimizing the deprivation to liberty of those in the low risk group. This risk group management approach is not perfect and does not increase the ability to identify a particular individual who may commit a heinous act, but it does allow effective management of those at higher risk.

The management ethically and pragmatically justified, must retain some semblance of proportionality between the apprehended insult and the impact of the proposed preventive strategy. In practice this obliges us in most clinical situations not to resort to increased coercion, let alone preventive detention, but to focus attention on

greater support and more active follow up and treatment in the community with a more ready resort to admission during exacerbations of symptoms or social conflict.

Risk assessment instruments

The last 15 years or so has been marked by a wave of enthusiastic advocacy specifically for the benefits of so called actuarial risk assessment instruments (e.g.,[31,32,33]). The advocates of actuarial risk assessment claim directly, or by implication, to be able to identify the likelihood that specific individuals will progress to various forms of interpersonal violence. These risk assessment tools are based mainly on retrospective, though occasionally prospective, studies of specific populations, such as discharged patients and released offenders. Actuarial approaches have the advantages of:-

1 Multiple variables delineating level of risk.

2 Designed to move from group data to individual attribution.

3 Realized in simple objective reproducible rating scales which minimize individual clinician's discretion and therefore responsibility.

4 Focus attention on 'high risk' individuals (principle of targeted resources).

5 Provides protection to clinicians and managers in event of disaster. Nobody can be blamed for the failures of 'science'.

Actuarial approaches are not without their problems. The results, for example of commonly used risk assessment instruments like the VRAG and the Static 99 change little if at all over time and with circumstances. Clearly whatever the Static 99 may indicate the risks of committing further rapes in a fit 25 year old is unlikely to remain the same as he ages and acquires disabilities.[34] Actuarial approaches also almost inevitably revolve around a limited number of variables which exclude uncommon though potential critical factors. Thus, for example, morbid jealousy is not sufficiently common to emerge as an actuarially established risk factor despite studies on such cases indicating a very high probability of significant violence. The structured clinical judgement approach allows the incorporation of such potential modifiers[22]. Actuarial instruments are developed on specific samples constituted in particular places at particular times. This can lead to problems with generalisability and equally important idiosyncratic and false attributions. For example, in both the VRAG[31] and the Classification of Violence Risk (COVR)[35] the schizophrenic syndrome emerges as a protective, or at best a neutral, factor with regard to the risk of violence, despite being associated in many other studies with far higher rates of violence than occurs in the general population.[26] This is because in the VRAG the rate in those with schizophrenia was compared to other offenders, some of whom had severe personality disorders, and as expected it was lower. In the MacArthur study, from which derives the Classification of Violence Risk (COVR),[35] substance abuse was treated as an independent confounder and the rates for the select few who had a schizophrenic syndrome, but were not substance abusers, were used to calculate the level of risk for schizophrenia.

The use of the term actuarial links these approaches to the well established actuarial methods familiar from the insurance industry. In the insurance industry actuaries usually generate their risk groups on the basis of samples numbered in the thousands, with the occasional in the tens of thousands. In the mental health fields the samples are usually measured in hundreds, with the occasional topping the thousand mark.[1] Actuaries in the insurance industry work exclusively with group based predictions. Your car insurance will depend on such variables as the type of car you drive, your age, your gender, your prior driving record, and where you live. That determines the group you fall into for the purposes of costing the policy you request. The actuary is not interested in what happens to each individual only whether the whole group for whom policies have been drawn costs sufficiently less in claims than is received in policy payments to produce the required profit margin. The actuarial method is not designed to assign levels of risk to individuals but to groups, though the cost of an individual's policy will be determined by the risk group into which their policy falls. The precision of the group prediction is determined by the size of the sample and the frequency with which the event of concern occurs. The rarer the outcome of interest (e.g., murder or plane crashes) the larger has to be the sample, and to some extent the commoner the event (minor assaults or fender benders) the smaller the sample. Irrespective of the sample on which the risks have been established as you try to make finer and finer distinctions involving smaller and smaller subdivisions within the original sample so the confidence that can be placed in the estimate decreases. Paradoxically the more intense and detailed the analysis of the original group the less reliably can the derived risk algorithm be applied to those outside the group.[19] The MacArthur study on which the COVR is based exemplifies this problem. The smallest unit is obviously the individual group member and here the inherent variability of the risk prediction will be at its highest.

To take the example of two of the most widely used and best established actuarial risk instruments. The VRAG claims to identify nine groups, known by the unfortunate term 'bins', with a probability of future violent recidivism varying from 0 per cent to 100 per cent.[31, 32] The analysis of Hart and colleagues[36] demonstrates that rather than nine statistically separable groups there are only three statistically distinct groups. The Static 99 claims to separate sex offenders into 7 groups with recidivism rates varying from less than 10 per cent to greater than 50 per cent.[37] Statistically however only two separable groups are generated one with offending rates between 4 per cent and 25 per cent and one between 30 per cent and 60 per cent.[36] In short even ignoring the all important problem of attributing a group risk to an individual within the grouping the Static 99 identifies recidivism with at best 2 in 3, and at worst 1 in 3, chance of accuracy. In this really good enough to damn a person to indefinite incarceration or extended imprisonment?

Unfortunately, unlike other areas that rely on actuarial approaches to decision-making, there has not been a concerted effort in the area of violence risk assessment to pool the results of various studies to obtain the large samples necessary to reduce the variance which would thereby reduce the broad values on the confidence intervals.

[1] There are notable exceptions, such as the Level of Supervision/Case Management Inventory which has accrued normative data on more than 35,000 prisons and almost 80,000 people under community corrections supervision [Andrews, D. A., Bonta, J. L., & Wormith, J. S. (2005). Level of Service/Case Management Inventory (LS/CMI). Toronto: Multihealth Systems.].

As noted in Footnote 1 above, there are some exceptions such as the LS/CMI. Doubtless, over time researchers will pool results to determine the extent to which confidence intervals can decrease, thereby increasing the predictive utility of instruments.

The big question which hangs over the use of risk assessment instruments is the extent to which it is possible, or acceptable, to make attributions about an individual's future behaviour on the basis of their sharing characteristics with those in a group with a known level of risk for such behaviour. In medicine we are so used to using probabilities to dictate our actions that there are dangers in failing to recognize the problems. An 18 year old woman presents with a history of severe central abdominal pain moving to the right iliac fossa associated with anorexia. The probability in those with similar symptoms of an inflamed appendix may be 30 per cent but 100 per cent of the patients are advised to have surgery. Probability is used here to provide advice entirely in the interest of the patient. If the advice is refused the patient is not forced into surgery but is observed and managed non-surgically. Compare this with advice to a court or tribunal considering extending the incarceration of an offender or patient. The offender may have similar characteristics to those with a better than 50 per cent (or even 80 per cent) chance of re-offending and to attribute the group risk to the individual may benefit the community. It will however almost certainly disadvantage the individual who will not be the one accepting or rejecting the advice. It is fallacious to argue that making such attributions from group membership to individual risk is acceptable because it reduces the errors of false positives. It is not possible to benefit the so called low risk without disadvantaging the rest. Even with particularly strong measure of risk assessment, the false positive rates still hover around 25 per cent.

The only strong defence of attributing risk to an individual by virtue of their group membership, which is the essence of prejudice, is that it has better outcomes than not using this approach. But what are better outcomes? Better outcomes could be reduced to the accounting of true and false attribution. On this basis actuarial risk assessment instruments perform better than the best guesses of most experts. In the case of possible appendicitis given the benefits of early versus delayed intervention a high number of false positives is tolerable to avoid even one false negative. When the outcome is imposing further incarceration even the price of one false positive for every true positive, which the Static 99 and VRAG might offer may raise moral and legal qualms. Particularly as given both the true and the false positive will be incapacitated making it impossible to ever know which was which. There is no quick corrective feedback in the world of assessing the risk of future criminality, unlike appendicitis where the pathologist will soon tell you if you were right or wrong. Further when supposedly low risk individuals are discharged or released and re-offend, this will reinforce any tendency to err on the side of caution and incarceration.

Statistical decision theory can support using group membership to attribute levels of probability to individual group members. Though even here sceptics might suggest that the efforts put into Bayesian approaches to make the best of limited data sets might better be expended in enlarging such data bases. What in any case the approach cannot do is decide on utility and moral propriety. It is only relatively recently that the criminal justice system has moved to making the prediction of future offending the dominant issue in determining sentences, parole, and the extensions of sentences (which in and of itself is an entirely new phenomenon). Similarly the emphasis on the risks psychiatric patients present to others is a relatively recent preoccupation for mental health services. The development of standardized risk assessment instruments not only serves these changes but sustains them. The sexual predator legislation in the US, the DSPD and indefinite sentencing provisions in the UK, and the extended supervision/imprisonment laws in Australasia, are all dependent on mental health professionals providing them a veneer of science and objectivity through risk assessments.[38, 39] While providing the courts with information regarding the extent to which the individual's characteristics place them in a general level of risk we by implication give our assent and support to that process. We place a hope for community benefit before an inevitable disadvantage to those with whom we have engaged professionally.

The current state of the science of risk prediction some might suggest delivers only a limited improvement in the decision making process. If they are correct what, if anything, remains in the risk assessment literature of practical value? The short answer is that the establishing of risk factors remains of inestimable value, not in placing labels on individuals but in identifying how to reduce the probability of future violent and criminal behaviours. The standardized risk assessment approaches such as the HCR-20 and the VRAG allow individuals to be assigned to broad levels of high, low or medium probability of future violence. It can be argued whether or not the confidence that can be placed in such attributions justify imposing extended incarceration. They are however of undoubted value in assigning priority for management interventions.

The structured professional judgement measures, such as the HCR-20, also add considerable value by identifying risk factors, such as substance abuse, specific personality vulnerabilities, and the like allowing targeted interventions to reduce the chances of engaging in violence and offending. The exposure of the current limitations in the project of identifying dangerous individuals is bad news for courts, parole boards and governments looking for short cuts to community safety. But for mental health professionals it is merely a reminder that our business is managing patients to reduce the risks for them and for others, not trying to separate the dangerous goats from the mostly harmless sheep.

The limitations on the ability to accurately predict risk also emphasize that in all circumstances where psychiatrists and psychologists are being asked to provide opinions of risk of future violence to courts or other decision-making bodies, they should provide the courts with the information that they can. This would include, for example, the general risk level in which the risk assessment tools would place the patient but it would also extend to an anamnestic consideration of factors in the patient's case that are known to increase or decrease the level of risk. In the end, though, the clinicians need to be clear to state the limitations of their findings and to, of course, leave the ultimate decision of whether the patient meets the criteria of the statute in question to the legal decision makers.

Practicalities of risk prediction and management

Risk assessment and management can be conceptualized as a four stage process:

1 An evaluation of general level of risk and priority for active intervention. In practice this can rarely go further than saying they are in a high, medium, or low, priority or risk category.

2 Identify current risk factors and future hazards which are both potentially remediable and causally relate to increased chances of violence.

3 Develop management strategies to reduce or remove the deleterious influence of these factors.

4 Evaluate the effectiveness of the interventions in reducing subsequent violence.

Each of the above steps will be discussed in turn below.

Evaluating level of risk

Such evaluations should start simple, consider the context, apply clinical and common sense, then potentially progress to the use of a standardized assessment instrument. In both the mentally disordered and general population the majority of violent and criminal behaviour is committed by young males with histories of repeated antisocial behaviour dating back to conduct disorder in childhood. This is often combined with anomie, that lawless disaffection with society in general and contempt for rules and authority in particular. Impulsiveness is concerning[40] but so is the more muted feckless disregard for consequences found in some with a schizophrenic syndrome. Unemployment, living in a high crime neighbourhood, and antisocial peers all add substantially to risk. Similarly substance abuse is a robust maker for the risk of criminality in the disordered and non disordered alike. Specific to the seriously mentally disordered is a refusal to recognize they are ill, and resistance to complying with treatment with often an antagonism to professionals; the 'fuck off and leave me alone' syndrome.

Clinicians must maintain their common sense. A range of situations must be taken seriously. The angry and threatening who can tell you exactly what they plan to do to their supposed antagonist, the frightened who see no alternative to a pre-emptive strike, those who have prepared for violence (weapons, surveillance, put their affairs in order), the actively suicidal who have nothing to lose but still care enough to take a final revenge, and commonest of all those making threats in a manner which creates fear and concern either in the potential victim or those who have been privy to the threats.

Beyond normative data and risk factors, there exist a number of high risk situations and syndromes particular to psychiatry. These include:-

1 Morbid jealousy

2 Some misidentification syndromes

3 Depressed suicidal mothers of young children.

4 Delusional systems focussing on specific individuals believed to present for the patient a serious threat, or a malevolent impediment to their central project.

5 Some stalking situations

6 Confusional states be they toxic or related to dementia or other cerebral impairments (this is the source of a significant proportion of assaults on health staff)

Patients presenting any of the above characteristics require careful assessment and management to reduce the likelihood that risk will eventuate.

When there is an indication that a patient may be at an elevated level of risk for violence, or when the clinician is doing a risk assessment specifically, there is much to be said for becoming familiar with the use of standardized risk assessment instruments. They can direct attention to important areas that require consideration and provide a structure for both gathering and evaluating relevant information. The choice is between the actuarial (e.g., VRAG, Static 99 and COVR) and those employing structured professional judgement (e.g., HCR 20). The former generate fixed risk levels, the latter in their nature are open to further modification in the light of clinical and common sense. Pure actuarial instruments (e.g. Static 99 and COVR) may appeal to the less experienced given their relative simplicity of administration. Their disadvantage even for the expert is, however, that they can function like black boxes which generate evaluations without the user being necessarily aware of how the instrument was constituted and the limitations that should attend its use. Moreover, they have been validated for use in fairly limited contexts and countries. As a result, clinicians may put undue weight on the results the instruments produce, without knowing how the results might vary in the context in which they are working.

Structured professional judgement is more transparent, if more demanding of the user. Where the actuarial measures can be used by relatively poorly trained individuals or in some contexts clerical staff, the structured professional judgment measures must be employed by those with considerable expertize. We favour the structured professional judgement approach of the HCR 20 but readers need to understand that actuarial instruments are perhaps the dominant forms of risk assessment instrumentation, due in no small part to their reliance on static variables and ease of administration. The HCR 20 is designed for use with general and forensic psychiatric patients and it has been found to have utility in general offender populations. Those whose work brings them into contact with specific groups such as perpetrators of domestic violence, child molesters or stalkers should consider using risk assessment approaches developed specifically for these populations (e.g., the Spousal Assault Risk Assessment (SARA),[41,42] Sexual Violence Risk-20 (SVR-20),[43] Risk for Sexual Violence Protocol (RSVP).[44,45]

The end point of a risk analysis in most cases will allow individuals to be placed into one of three somewhat arbitrary groups which encompass both likelihood and potential level of damage:-

i) High Risk – the individual presents a significant risk of committing a seriously damaging act of violence within a reasonable timeframe (less than a year or so). It is generally impossible to quantify the numerical probability for 'high risk' as it will vary across instruments and in different situations from approximately 30 per cent (sexual offending) to 80 per cent (the occurrence of general violence). Management of the risk is required immediately and the level of risk should be re-evaluated periodically depending on the extent to which the individual's personal situation and dynamic risk factors may vary. When the potential violence is of a particularly horrendous nature (e.g. potentially lethal) the timeframe can reasonably be extended to encompass a number of years. In, for example, some predatory child molesters and some who have killed from morbid jealousy, the chances of re-offending may be substantial and continue virtually throughout the offender's life span.

ii) Moderate Risk – the individual presents a real risk of committing a damaging act which might inflict minor injuries and/or significant fear and distress within a year or so. This group also requires their level of risk to be managed though, because the severity of behaviour is less extreme and the time period

perhaps less imminent, the extent of management is less intensive or restrictive. Also in this group can be placed those with a more remote (less than 30 per cent) but not inconsequential (above 5 per cent which in practice is the limit of reliable detection) risk of serious violence. Examples include those at lower risk of committing acts of a more serious nature (e.g., those making viable and tangible threats of engaging in behaviour which would lead to harm).

iii) Low Risk – Individuals who do not present a real risk of harming a third party. Their level of risk and potential for severe risk is low. They do not require any risk management plan beyond normal care and there is no need to re-evaluate the risk at any time in the reasonably foreseeable future. In essence, this category includes everyone not in the High or Moderate categories.

A final re-emphasis. Even using standardized instruments the final evaluation has to take into account factors beyond the figures generated by the black box. Risk evaluation is about formulation not simply calculations.

Identifying factors relevant to decreasing risk

The primarily clinical purpose of risk assessment should be risk management. Risk management is about identifying those factors which mediate the increased risk and modifying them to decrease risk. The structured professional judgment instruments and a good working knowledge of the risk literature will assist the clinician to identify and understand relevant risk factors. As noted above, identification of the dynamic risk factors points directed to potential management approaches. A simplified schema of the mediators and moderators which link having a schizophrenic syndrome to violent behaviour is presented in Fig. 11.14.1 with the basic approach to breaking or attenuating those links represented in Fig. 11.14.2.

The management strategies in high risk groups take us directly to good clinical practice with the addition of few specific approaches. The basic approach to management of high risk individuals can be summarized:

1 Substance abuse. This claims first place not because it is necessarily the most important risk factor but because the presence of the significant abuse of alcohol or drugs can disrupt all other management approaches.

2 Psychopathology. Obtaining adequate control of the delusions, affective disturbances, and hallucinations which predispose in some cases to offending behaviours is the second management imperative. Again without adequate control of the psychopathology little progress is likely in more targeted treatment modalities. High risk groups are often reluctant to comply either with medication or psychological management. This may force the use of compulsory treatment in an inpatient situation for sufficient time for the patient to begin to experience the benefits of amelioration of their active psychosis. Extended admissions (4–12 weeks) also provide an opportunity to establish trusting therapeutic relationships, though equally they can be productive of resentment and even more marked resistance to treatment. Second generation antipsychotics are preferable in this group as they are better tolerated and less likely to further impair cognitive and particularly frontal deficits. Depot medication is useful initially though this currently restricts considerably the choice of medication at least until more second generation antipsychotics come available in depot format. Clozapine can be particularly effective in this group but the level of cooperation required usually prevents its use at least in the early phase of management.

3 Social Circumstances. Discharging high risk patients to disorganized or casual accommodation in high crime neighbourhoods virtually guarantees offending behaviours. Similarly the drift back into contact with substance abusing peer groups increases risk and further disrupts management. Unemployment or just

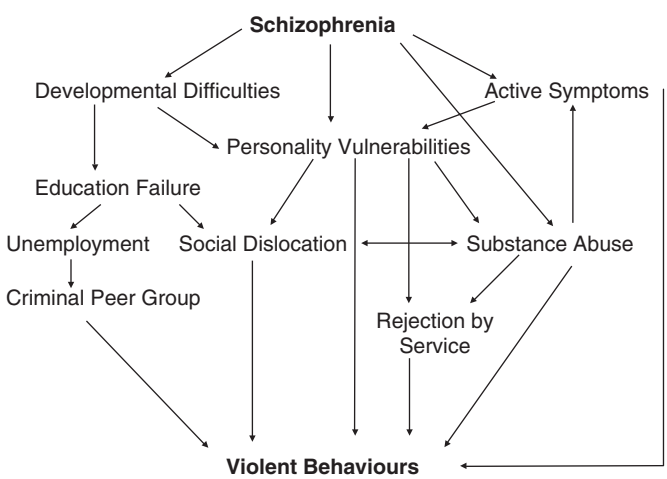

Fig. 11.14.1 A simplified schema of the mediators and moderatory between having a schizophrenic syndrome and behaving violently are illustrated. The very complexity of the nexus between illness and violence offers multiple opportunities for intervening to break the links. [Reproduced from Mullen, P.E., (2006). Schizophrenia and violence: from correlations to preventative strategies. *Advances in Psychiatric Treatment*, **12**, 239–48, copyright 2006, The Royal College of Psychiatrists.]

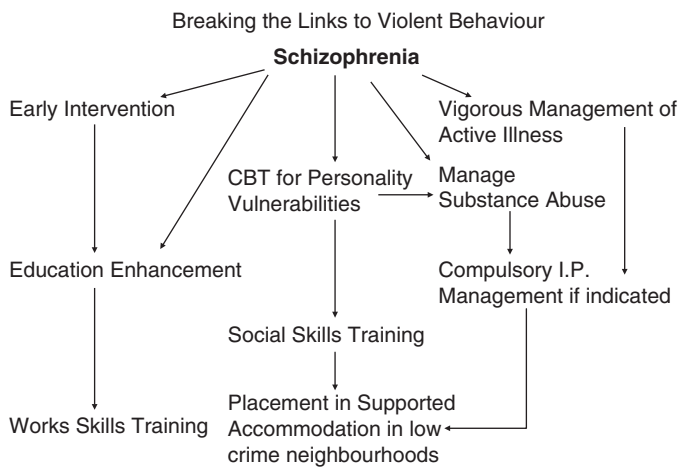

Fig. 11.14.2 Illustrates some of the interventions which could reduce the strength of the association between having schizophrenia and behaving violently. All interventions depend on accepting that it is the services duty to manage both the violence which can emerge from schizophrenia, and those with schizophrenia who are also substance abusing, delinquent and objecting. [Reproduced from Mullen, P.E., (2006). Schizophrenia and violence: from correlations to preventative strategies. *Advances in Psychiatric Treatment*, **12**, 239–48, copyright 2006, The Royal College of Psychiatrists.]

the lack of structure to their days increases risk. An absence of non offending, non substance use satisfactions leaves patients vulnerable to offending. The high risk groups need stable accommodation in low crime neighbourhoods, with active support, and structured recreation with later more long term educational and work related activities. Assisting them into contact with non deviant social groups (via sport, voluntary work, employment, or hobbies) is essential to establish social rewards to replace the pleasures of substance use and crime.

4 Insight. Or more concretely the acceptance of the need to change their attitudes and behaviours which support criminal and violent behaviours is a requirement for more targeted interventions. The stages of change model[46] combined with motivational interviewing[47] can assist in beginning the process. Ultimately it is through establishing a trusting relationship with the therapist and ideally the treatment team that commitment to change and maintaining change is obtained.[48]

5 Personality Traits. Personality disorders may or may not respond to treatment but the traits out of which they have been constructed are mostly open to modification. The objective is not to transform a suspicious, manipulative, insensitive, self absorbed thug into a paragon of the social virtues. It is simply to ameliorate those traits which predispose to antisocial behaviour. Targeted CBT offers a range of options. Wong and Hare's[49] guide to managing psychopathic traits offers a useful source of guidance for developing such programs.

6 Victim Empathy. Assisting the high risk group to understand the impact of their behaviour is essential. Sadly this is often best approached through sensitizing them to the harm they bring on themselves rather than through victim empathy programs, but both are worth attempting.

7 Common Sense and Prudential Wisdom. High risk patients with a schizophrenic syndrome not infrequently lack the mundane capacity to foresee the obvious outcomes of their behaviour. This produces a feckless foolishness. Instilling prudential wisdom in those impaired in this manner is a matter of slow progress in structured interactions which focus on their actual behaviours and the enhancement of the capacity to modify those behaviours in function of their longer term outcomes.

Conclusions

There are significant pressures and obligations—both legal and professional—for mental health professionals to give prominence to risk assessment and management with their patients (clients). The extent to which risk assessment will be more or less prominent will depend largely upon the nature of the professionals work, and the environment in which it occurs. Caring for those with high prevalence disorders in private practice will have little to worry about. For those responsible for patients who are acutely psychotic and of course those working in forensic contexts, the importance of risk assessment will be more significant.

We are at an important crossroads in our level of knowledge about both violence among psychiatric patients and about risk assessment. The fact that the risk for violence among psychiatric patients is not insignificant does mean that risk assessment and management is a legitimate activity for mental health professionals.[45] As such, psychiatrists and psychologists must become familiar with the risk assessment literature and emerging technologies. With appropriate levels of knowledge and training, clinicians can master this complex area in a way that can serve to satisfy their professional and legal obligations. Only with a good understanding of the field can we be protected from the risk of either blithely neglecting the violence risk our patients pose or of becoming so risk averse so as to unnecessarily and arbitrarily restrict our patients' liberty.

In the area of risk assessment, the framing of the main research questions and the articulation of the resulting data is increasingly in terms of actuarial risks and the generation of standardized questionnaires which will generate predictive scores. The technology of risk assessments could become one of the primary mediators of the relationship between the professional and the mentally disordered person. This will radically alter how the patient and their disorders and disabilities are revealed to us. We must not allow the technological focus on risk to replace the importance of the patient's personal and social context. We must not allow it to objectify them and their disorder as an embodiment of a quantum of 'riskiness.' To this end, there is an argument to occasionally separate the formal risk assessment task from that of patient treatment. The roles are blended—as they should be—in most cases, such as caring for voluntary patients and those involuntarily committed for brief periods. When the question of the patient's level of risk is that which will determine their liberty over the long term, however, it is less tenable for a treating clinician to maintain a productive therapeutic relationship with patients while holding the reins on their liberty (e.g., consideration of reviews of indeterminate dispositions, evaluations of risk for sentencing purposes and parole decision-making).

Technology is about performance and control, it is about domination, and the objects of technological manipulation are just that, objects [see[50, 51]]. To the extent that technological approaches to risk assessment come to dominate clinical practice, whatever benefit they may bring, the price will be reframing the clinician's view of their patients as potentially dangerous things. Risk assessment forms part of a major shift in psychiatric practice and theory away from individually based engagements between clinicians and uniquely troubled individuals to a world of standardized best practice. Instruments direct diagnosis, diagnosis determines which system of treatment is to be applied and risk assessments enable us to prevent damage to, or by, the objects of our professional responsibilities. Efficient, effective, properly evaluated performances of mandated procedures becomes the definition not only of the normative but the ethical.

This chapter began with a quotation from a nobel prize winner, it will end with the story of another. In reading the account provided by Nasser of the schizophrenic illness of the Nobel Laureate John Nash images emerge of the complex interactions between illness and the sufferer's humanity, life and even genius.[52] Nash had a devastating mental illness, he was as a result of his illness compulsorily treated and even at times regarded as dangerous. Nash finally entered a stable remission in which he could once more work as a mathematician. This was without the continuing aid of medication or any other form of mental health ministrations. What has this to do with risk assessment and management? Everything. The outcome of an illness such as one of the schizophrenias in an individual case remains enormously difficult to predict. We must, as mental health professionals, act on our estimates of

future probabilities. We should struggle to make our risk assessments and risk management strategies as effective as possible. But in the end we should remain modest about our capacities to perform such predictive and preventive functions and not loose curiosity about what really delivers Nash and many others from insanity and even dangerousness.

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11.15

The expert witness in the Criminal Court: assessment, reports, and testimony

John O'Grady

As an expert witness in the Criminal Court, the psychiatrist ceases to be simply a doctor as a psychiatrist's report and testimony addresses issues on the boundary between law and psychiatry. The law is not primarily concerned with the welfare of the defendant. Criminal law is concerned with justice, fact finding, and the attribution of guilt whilst psychiatry concerns itself with the welfare of the individual, their mental disorder, and its treatment. This chapter will explore the legal framework for expert reports and testimony, standards for such work, the particular ethical dilemmas of this work and provide practical guidance on preparation of reports and testimony.

This chapter draws upon previously published work by the author.[1,2] Expert evidence cannot be understood except in reference to a particular legal jurisdiction. For this chapter the legal system in the United Kingdom is chosen but the general principles will apply to all jurisdictions. Issues specific to Civil and Family courts will not be discussed.

The psychiatric expert witness[3–5]

Witnesses in court can only give evidence of facts they personally perceived and not evidence of their opinion. It is for the court to draw inferences from the testimony of witnesses. The opinion of an expert witness is an exclusion to this general rule because courts need the assistance of experts to consider issues beyond their knowledge.

Lawton L.J in R v Turner established a 'common knowledge' rule governing expert evidence as follows:

> An expert opinion is admissible to furnish the Courts with scientific information which is likely to be outside the experience and knowledge of a Judge or a jury. If on the proven fact, a Judge or jury can form their own conclusions without help, then the opinion of an expert is unnecessary. In such a case if it given dressed up in scientific jargon it may make judgement more difficult. The facts that an expert witness has impressive scientific qualifications does not by that fact alone make his opinion on matters of human nature and behaviour within the limits of normality any more helpful than that of the jurors themselves; but there is a danger that they may think it does … jurors do not need psychiatrists to tell them how ordinary folk who are not suffering from any mental illness are likely to react to the stresses and strains of life.

This seems to limit psychiatric evidence to recognized mental disorder. However, expert advice is allowed which is 'outside the experience and knowledge of a Judge or jury'. The abnormal/normal dichotomy is not a rule of law but guidance. Courts have allowed evidence on a variety of conditions which would not normally be thought of as established mental disorder, for example 'Battered Women's syndrome'.

Particular problems arise for the Court in respect of borderline conditions falling short of recognized mental disorder. Here admissibility will usually be determined by the court's judgement as to whether the expert evidence addresses matters outside the experience or knowledge of a Judge or jury. Generally courts seek to limit evidence to established abnormal conditions.[3,5] Courts have problems with evidence that utilizes leading edge or novel theory or diagnosis. The Court will require evidence that the novel theory or diagnosis is sufficiently organized or recognized to be accepted as a reliable body of knowledge by the profession. The expert will need to demonstrate that acceptance through reference to scientific literature.[3,5]

For medical experts, the Courts are able to establish expertise by reference to qualification and training. Nevertheless, the expert must be able to demonstrate that they have the requisite expertise in a particular case. For example, a psychiatrist trained in general adult psychiatry may not be an expert in a case concerning a person with moderate to severe learning disability.

Immunity from suit

Lawyers and experts enjoy immunity from suit (civil litigation) for their professional work in court.[3] For medical expert witnesses this includes their report and any oral evidence presented in court. That immunity does not extend to immunity from report to the doctor's regulatory body. It does not extend to subsequent actions such as duty of confidentiality in respect of disclosure of reports to third parties. The judgement in the case of GMC v Meadows lays out the legal and public policy arguments for immunity from suit but with regulation by their professional regulatory body.

Reliability of expert testimony

Courts need to assure themselves that an expert witness's evidence is reliable. This creates an obvious problem as the very reason an expert is giving evidence is that they have expertise which the judge or jury does not possess.

Courts have utilized three broad approaches to this problem. The first is to examine the scientific validity of evidence. The second is to devise standards for expert evidence and the third is to regulate experts through formal accreditation systems.

Scrutiny of scientific evidence

The landmark case is that of Daubert v Merrell Dow Pharmaceuticals in the United States courts. That ruling established stringent criteria to judge reliability to include that the technique, body of knowledge, or theory can be tested, has been subjected to peer review and publication, has a known rate of error, is subject to maintenance of standards and controls and is generally accepted by the scientific community. This judgement is problematic for a number of reasons,[6] not least because the court does not have that expert's specific knowledge but nevertheless has to make a scientific judgement on the reliability of that expert's evidence. Judgements are unlikely to be value free determinations and there is a risk that the admissibility of evidence could be distorted by policy considerations or interfere with the use of leading edge science in the court. These considerations have made United Kingdom courts reluctant to introduce a 'Daubert' type test but there is pressure to do so[4,7] with public concern about miscarriages of justice linked to expert evidence.

Regulatory rules for expert witnesses

Courts have defined the standards expected of an expert witness; the landmark case being the judgement of Cresswell J in The Ikarian Reefer. Court judgements have been used to draw up formal rules governing civil family and criminal courts; for UK Criminal courts, the relevant rules are contained in Part 33, Criminal Procedure Rules.[8] The common features are listed in Box 11.15.1 below. The understandable anxiety of the court to ensure experts adhere to these stringent standards may have the unfortunate effect of deterring psychiatrists from providing occasional expert reports for criminal courts.

Box 11.15.1 Common features of regulatory rules for civil family and criminal courts.

Expert Reports should contain:

1. Details of academic and professional qualifications together with experience and accreditation relevant to the opinions expressed in the report (usually as a summary in the introduction with more detail within and Appendix).
2. A statement of the range and extent of expertise together with limitations upon that expertise, particularly declaring when a particular issue is outside his expertise.
3. A statement setting out the substance of all instructions received together with listing all materials provided and considered, upon which the opinion is based.
4. Where there is a range of opinion on matters dealt with in the report, a summary range of opinion together with reasons for the experts preferred opinion (see section below on Report Writing).
5. A declaration of any facts, materials, or investigations which might bear upon or be made against the expert opinion.
6. Extracts of literature or any other material upon which the scientific evidence is based.
7. A statement of which facts are within the expert's own knowledge and which are assumed.**
8. Where an opinion is qualified, a statement to that effect.
9. A statement that the expert has complied with his or her duty to the court to provide independent assistance by way of objective unbiased opinion in matters within his or her expertise.
10. A statement that the expert will inform all parties, including the court, in the event that his or her opinion changes on any material issue.
11. A declaration of truth.

** Courts distinguish true and assumed facts. The only facts the psychiatric expert will routinely know to be true are the results of examination and results of tests or investigations. All other facts will be assumed to be true.[9]

Accreditation

In the United Kingdom, there are a number of organizations to accredit expert witnesses utilizing some combination of direct scrutiny of reports and references from legal teams. None so far have addressed the specific needs of the expert psychiatrist in court. They provide the court with some measure of an expert witness's expertise in legal matters over and above what comes from their basic professional qualification. Critics have pointed out[10] that once registered accreditation is unlikely to pick up poor practice, as experts with years of experience, but not necessarily competence, are unlikely to be refused accreditation. Accreditation is unlikely to prevent expert straying outside their area of expertise. These schemes have not as yet been able to deal effectively with problems of accrediting inexperienced but competent aspiring expert witnesses. To be effective they may require codes of discipline with the attendant danger of attracting vexatious complaints. The Royal College of Psychiatrists in the United Kingdom utilizes a competency based training framework together with standards for continuing professional development (CPD) following training to promote a high standard in medico-legal work. Evidence of completion of such training and CPD is likely to be the most effective way of demonstrating credibility as an expert witness.

Ethics

Dual role

Stone[11] used the term 'dual role' to describe the psychiatrist in the legal context. In Stone's view the role of the clinician and medical examiner for Court are irreconcilable. The evaluee/patient is unable to distinguish the role of the medical examiner as a Court expert from that of personal physician. This result is an inability to protect

themselves from inadvertent disclosure that might adversely affect the outcome in Court. He argued that clinicians cannot help using their therapeutic skills to engage the patient in disclosure. The dual role arises from the use made of the resulting psychiatric evidence for non-welfare purposes. Appelbaum[12] argued that the dual role of psychiatric experts in Court is best managed by understanding that psychiatrists operate outside the medical framework when they undertake forensic Court work and their practice is not governed by the ethical principles underpinning medical practice (beneficence and non-maleficence). Instead he argued that psychiatric experts should operate from a perspective of justice ethics employing ethical principles of objective truth finding and respect for the person (termed autonomy and truthfulness).

If this solution to the Dual Role dilemma were accepted, it would mean that the psychiatrist should not have a welfare/treatment role in respect of the person under evaluation. In the United Kingdom this is untenable[13] primarily because of Mental Health law which provides for diversion to the health system as a sentence following a finding of guilt (Hospital order). Weinstock *et al.*[14] have argued in the United States legal and clinical context the Appelbaum solution[12] is, untenable in that legal context as psychiatrists routinely have conflicting responsibilities thrust upon them where legal or other requirements may take precedence over patient welfare.

Reports addressing sentencing in the United Kingdom place the psychiatrist in a Dual Role position. This is because the psychiatric opinion can result in two outcomes for the evaluee

1 A welfare disposal under Mental Health legislation.

2 Potentially greater restrictions on the defendant, including an indeterminate life sentence where there is expert evidence on mental disorder but no recommendation for a welfare disposal.

Statutes that introduce indeterminate life sentences for public protection based upon assessed future risk of re-offending cause particular problems.[1,13,15] English Courts have, through case law and practice, sought psychiatric evidence when they consider defendants may have mental disorder and where the Court is considering an indeterminate life sentence.[1] Psychiatric evidence on risk will be central to the expert's evidence. The Court may have two options, a Hospital Order in suitable cases or an indeterminate life sentence. The psychiatrists does not have 'a priori' advance knowledge of what the outcome might be in a particular case thus routinely placing the psychiatrist into a dual role in respect of the evaluation.

Calvedeno[16] has pointed out that even where a welfare disposal is recommended, medical evidence in respect of special restrictions to a Hospital Order may lead to lengthy periods in hospital justified not by the need for treatment but by psychiatric judgement on risk in the future. Similar arguments apply to reports to Mental Health Tribunals for patients detained under mental health legislation.

One solution to the dual role is to act only where there is a realistic prospect of benefit to the patient. This leads some psychiatrists to only undertake work for defence teams. In the author's view, this is unethical as it lends itself to bias and deprives one side in the adversarial process of high quality experts.

A theory of mixed duties to address dual role conflict

Doctors are members of society and as citizens have responsibilities, prior to responsibilities as a doctor. The narrow domain of medical ethics does not remove from doctors their duty to consider the interests and rights of other people and to consider the distribution of benefits and risks. Beauchamp[17] proposes augmenting traditional medical ethics with principles he terms justice and respect for autonomy. On that basis O'Grady[1] suggests a framework of mixed duties for expert witnesses in court to address their 'dual role' conflict (see Box 11.15.2).

This approach implicitly requires the psychiatrist to work within a framework of conflicting duties where ethical judgements must balance the welfare of the evaluee against the rights of others and society's legitimate interest in protection from risk.

The psychiatric ethical expert in court is then the one who 'feels the tension' inherent in a dual role and is painfully aware of the conflicting demands of different ethical imperatives.

Risk assessment

Sentencing where public protection is a central issue poses particular difficulties for the psychiatrist as risk assessment becomes central to the court's decisions. Actuarial risk assessments can be particularly dangerous in the legal context. Mullen[18] argues 'The margins of error in every actual or conceivable risk assessment instrument are so wide at the individual level that their use in sentencing, or any form of detention, is unethical'. Whilst acknowledging the significant limitations of risk prediction at the individual level, the Court may nevertheless legitimately argue that evaluation of risk associated with mental disorder is an area falling outside the 'common knowledge' of Judge and Jury. Therefore the court must rely upon psychiatrist's opinion on risk and mental disorder as the psychiatrist is the only witness with the necessary expertise. Using a structured risk assessment methodology may go someway to ensure accuracy, objectivity, and truthfulness. A clear role for the psychiatric expert is to ensure the Court is provided with informed scientific evidence on the limitations of risk assessment and particularly the limitations of utilizing structured or actuarial risk instrument at the individual level[18,19]).

Structure of reports

Receiving instructions

The psychiatrist should understand the legal question to be addressed; where necessary standard text should be consulted (see

Box 11.15.2 Ethical principles to address Dual Role responsibilities

1 Medical ethics:
- Non-maleficence
- Beneficence

2 Justice ethics:
- Truthfulness (objective and subjective)
- Respect for autonomy
- Respect for the human rights of others (balancing the distribution of benefits and risks for the patient and society)

recommended reading). The psychiatrist must ensure that they have the necessary expertise to address the issues for the Court. Trainees must ensure that they are supervised by a suitably qualified senior and disclose this to the instructing party (including disclosure of the supervisor's appointment and qualifications).

The psychiatric expert must ensure that they can meet the needs of the Court as regards timescale for the report and understand that they can be compelled by the Court to give oral evidence; for example when they are on leave. The Court will not do so if the doctor has in advance disclosed dates when they are unavailable. Where there are fees to be paid, the letter of acceptance should state the contractual conditions for accepting instructions.

Rules of evidence in all legislations impose on the expert witness an overriding duty to the court outside of the duty owed to the party instructing them. The psychiatric expert witness has to develop a working relationship with the legal team instructing them but simultaneously discharge their overall duty to the court. One way of conceptualizing the relationship to the instructing party is as a 'consultant' to the legal team, educating them in the meaning of psychiatric findings.[20] Nevertheless it is naive to believe that expert will not be subject to overt or subtle influence by the instructing side.

Psychiatric reports should comply with relevant court rules for example Part 33 of the Criminal procedure rules for England.[8] Notes and documents must be retained for a sufficient period (undefined but at least to until last date for appeal) and disclosed to other experts in the case.[20] The expert should have appropriate indemnity insurance.

The interview

If the defendant is to be visited in prison, arrangements should be made well in advance and comply with the requirements of the institution.

At the beginning of the interview, the examining psychiatrist should explain carefully to the defendant the nature of the doctor's dual role, the limits of confidentiality in producing a medico-legal report and that the Court will have full disclosure of all material known to the report writer (no off record material). It is prudent to obtain a signed record of this discussion and to include it as part of the introduction to the report. Whenever possible, an informant should be interviewed; by telephone if necessary.

Structure of the report

(a) Declarations and introduction

The first section of the report should lay out the instructions received and what was done in order to produce the report. The dates and duration of interviews should be stated, including interviews with informants. For British Criminal Courts, Part 33 of the Criminal Procedure Rules[8] requires certain declarations and statements at the beginning of the report (see Box 11.7.1 above). A section on limitations to the report should be included to record matters such as documents not disclosed or unavailability of informants and state the impact on the expert's opinion.

(b) The facts

The middle section of the report should record briefly the facts upon which the opinion is based and should avoid interpretation which is the proper function of the opinion section. In psychiatric reports, the only facts that are within the psychiatrists own knowledge are likely to be those based on the findings of mental state examination. All other facts are assumed. If structured tests are utilized, they may also constitute facts within the psychiatrists own knowledge.

(c) Opinion

The role of the psychiatric expert is to provide an opinion on mental disorder and its implications for the matters before the Court. The opinion section should then start with a description of the defendant's mental disorder. If there is no evidence of mental disorder, then the privileged exception accorded to psychiatric experts no longer applies (see earlier section). The features that lead to a diagnosis of mental disorder should be described, avoiding jargon, and including mental state findings, so that others can understand how the opinion is reached. The diagnosis should be clearly stated using a recognized classification which for British psychiatrists will be the International Classification of Mental and Behavioural Disorders. Where a condition is described which is not part of such recognized classification systems or where 'leading edge' scientific findings are used to support the opinion that should be justified by disclosure (as an appendix to the report) of relevant literature to support the expert's opinion.

The second stage of the opinion is to translate the psychiatric findings into the legal language employed by the Courts. Terms such as 'diminished responsibility', 'insanity', or 'automatism' have precise legal definitions and the report should address how the psychiatric findings translate to the legal definitions employed by the Court.

Usually there will be a range of opinion and the psychiatrist should indicate the range, giving due weight to alternative opinion before recording the reasons for their own opinion. More than one legal issue may have to be considered together with the range of opinion on each separate legal issue. One helpful mental model is to consider the range of opinion that might be given by other experts if the case were presented to a psychiatric case conference.[20]

At point of sentencing where the court has concerns about public safety, the psychiatrist will be expected to provide an opinion on risk linked to the defendant's mental disorder. The ethical issues arising from that expectation should be thoroughly understood (see section above). It is usual to express a range of opinion (the case conference model) and given reasons for the expert's own opinion.

Where recommendations for a disposal under specific Acts are included, the precise wording of the relevant section of those Acts should be employed.

(i) Opinion on the ultimate issue

There is a common law injunction against a witness expressing an opinion upon the ultimate issue to be determined by the court. Many questions put to psychiatric experts test this rule to its limits. In this author's opinion, psychiatric experts should provide the court with objective evidence upon the mental state in and around the time of an alleged offence but stop short of expressing an opinion on the ultimate issue unless specifically instructed to do so by the presiding Judge.

Confidentiality

Medico-legal work undertaken by psychiatrists is governed by the same rule of confidentiality as applied to other clinical work.

Reports cannot be disclosed to a third party without the consent of the body commissioning the report. Psychiatric reports do not form part of a person's NHS medical record except by the express consent of the individual or their legal representative. Defence solicitors can exercise a right not to disclose a report to Court. Failure to comply with rules of confidentiality can lead to civil action or report to a professional regulatory body.

There may be circumstances where a psychiatrist believes that it is necessary to divulge confidential information to a Court without the evaluee's consent. This could arise:

(a) Where the evaluee refuses to cooperate with the preparation of a report.

or

(b) Where a report is not disclosed but the psychiatrist believes that disclosure is in the public interest.

A psychiatrist who believes that the evaluee is not cooperating with the preparation of a report because of mental illness has a duty to consider whether the evaluee's mental illness could interfere with a fair trial (for example, fitness to plead or lack of consideration of a mental health disposal). The psychiatrist must then make a judgement whether it is in the best interests of the evaluee for sufficient information to be provided to the Court to alert them of the doctor's concerns. Such disclosure will almost certainly be justified in the interests of a fair trial and justice. The doctor will also have a duty to consider whether steps ought to be taken to undertake a Mental Health Act assessment (British law) to enable transfer to a hospital for medical treatment.

The other situation where a breach of confidentiality may be justified is where a report is not disclosed but the report writer believes that the Court ought to consider the report's findings on potential risk to the public. In B.W. v Edgell and Rv Crozier, the Court held that the doctor's duty of confidence did not prevent a psychiatrist from taking steps to communicate the grounds of concern to the court. The strong public interest in disclosure to prevent a court from making decisions based upon inadequate information was held to override the psychiatrist's duty of confidentiality. Where a doctor is considering disclosure in these circumstances, advice should be sought from an experienced colleague, case law, and regulatory body guidance consulted and the doctor should seek advice from their indemnity insurer.

Appearing in court

Advice on practical matters concerning a Court appearance is beyond the scope of this chapter but guidance is available (see recommended reading). Those undertaking regular expert work should consider courses which prepare them for appearance in Court and should understand the legal framework for giving oral evidence in court.[21]

The cardinal rule when giving oral evidence in Court is that although called by one party, the expert witness is not giving evidence for that party's side but is under a duty to provide fair and impartial evidence to the Court even where this conflicts with the interests of the party calling them.

In Criminal Courts, the defendant gives evidence before expert evidence is heard. Experts are allowed, unlike witnesses of fact, to sit in Court and hear the evidence of other witnesses before they, themselves, give evidence. An expert can be called by any interested party in proceedings.

When calling an expert witness, the advocate must elicit the following[21]:

1 The expert's qualifications: If the report has been prepared according to criminal procedure rules, the report will contain a biography setting out the qualifications and experience of the witness. It will then be usual for the advocate to lead this part of the evidence by reference to the biography supplied in the report. It will be perfectly permissible for the other side to call into question the expert's qualifications. This should be met politely by outlining the reasons why the expert believes they have the requisite qualifications and experience to answer the questions posed in instructions.

2 Disclosure of the expert's report: The report will have been pre-read by the Judge and it is usual for the examiner to refer to relevant sections of the report. A report with numbered paragraphs is easier for the Court to follow. The jury will not have read the report and will not usually be given sight of the report. Their knowledge of the expert's report will come from submissions made by either side and through the Judge's questioning and summing up.

3 Advocates are under a duty to challenge disputed evidence. Thus where more than one expert opinion is provided and they differ; the expert must expect their opinion to be disputed. The expert must resist pressure from one party to deviate from or express greater certainty about an opinion they have reached in the written report.

4 An expert witness may be cross-examined as a hostile witness if there is good reason to suppose that they are not telling the truth. Thankfully this is extremely rare. The possibility of deliberate or inadvertent bias must, however, always be considered.

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11.16

Managing offenders with psychiatric disorders in general psychiatric services

James R. P. Ogloff

It has been shown that the prevalence of mental illness among those in the criminal justice system is significantly greater than that found in the general community.[1,2] As presented in Chapter 11.4, for example, the per capita rate of psychotic illness in prisons is approximately 10 times greater than that found in the general community. Tragically, relatively few services exist that provide continuity of mental health care between gaols and the community.[3] This produces a situation where individuals whose mental illness may have been identified and treated in gaol find themselves without services in the community. Typically, only when in crisis do they find their way into general psychiatric services either in community settings or in hospital. This situation has produced considerable stress on already taxed mental health services.[4]

Given the prevalence of offence histories among psychiatric patients, it is important for mental health professionals to be aware of the unique issues—and myths—that accompany patients with offence histories. At the outset it is important to emphasize that the duty of mental health services is to address mental health issues. That ought to be the focus of mental health services. As this chapter makes clear, though, for some patients, there is a relationship between the mental illness and offending and by addressing the mental illness, the risk of re-offending might well be reduced. Moreover, many of the ancillary issues that lead to relapse and destability in psychiatric patients also may lead to offending. Addressing these issues will both help provide long-term stability for patients and will help reduce their risk of offending. As a result, there is a need for general mental health services to acquire expertize to identify and manage patients with offending histories.[5]

This chapter will provide information about the relative risk of offending among psychiatric patients and the relationship (or lack thereof) of inpatient aggression and community-based violence and offending. A framework will be provided for assessing and treating patients with offending histories and issues using a typology of mentally ill offenders. The role of forensic mental health services in bolstering general psychiatric services, and in sometimes providing primary care for mentally ill offenders, will also be discussed.

How many patients have criminal histories?

Surprisingly little research exists that investigates the number of patients entering general psychiatric services who have an offence history. For reasons having to do with privacy, lack of perceived relevance, and professional reluctance, general mental health services do not consistently obtain reliable information regarding patients' offence histories. This is often the case even when the patient has a current community-based corrections order. The two following studies can help shed light on the question of how many general psychiatric patients have histories of criminal offending.

In a study that was conducted to investigate the post-discharge violence of psychiatric patients and the predictive validity of risk assessment measures among almost 193 involuntarily committed psychiatric patients in British Columbia, Canada who were discharged to the community, Douglas, Ogloff, Nicholls, and Grant[6] obtained official criminal histories for all patients who had ever been arrested or convicted of any criminal offence. The vast majority of patients had prior psychiatric hospitalizations (n=184, 95 per cent). Informally, members of the hospital staff were asked what percent of patients they believed had a prior criminal history. Staff, including psychiatrists, estimated that a very small percent of patients would have been arrested or convicted of offences—less than 20 per cent. The review of criminal histories, however, showed that 64 per cent (n=123) of patients had previous arrests or convictions for any type of criminal offence, including 40 per cent (n=78) who had been arrested or convicted of violent offences.

In an Australian study based upon Victorian samples of cohorts of patients with schizophrenia, Wallace and colleagues have found that almost 22 per cent of patients with schizophrenia have a history of offending at some point in their lives.[7] Moreover, eight percent of patients with schizophrenia had a criminal conviction for a violent offence. These percentages increased three-and-four fold when the patients with schizophrenia also had a known substance abuse problem. In a recent study, Hodgins and Muller-Isberner[5] found that one quarter of patients discharged from a general mental health service had a criminal record.

While it is difficult to know exactly how many psychiatric patients across different services have committed offences, the point that may be drawn from the above research suggests that many patients have offence histories—likely more than mental health professionals would expect. The starting point of the chapter, therefore, is that while most psychiatric patients will not have violent criminal histories, many will have offence histories, including the commission of violent offences. Moreover, many more patients will have exhibited violent behaviour that did not lead to arrest or conviction. Therefore, even if they do not realize it, all psychiatrists and other mental health professionals have experience working with patients who have offence histories.

What leads mentally ill people to offend?

Although the reasons that anyone—including psychiatric patients—offends are myriad and complex, a typology of mentally ill offenders is helpful for understanding the reasons they offend.[1] There are three general categories of people with mental illness who offend; understanding the general mentally disordered offender type will enable clinicians in general psychiatric services to provide appropriate treatment. The first, and smallest group, includes those psychiatric patients for whom a necessary and sufficient cause of their offending is the presence of their mental illness and the symptoms the illnesses produce. The second group includes patients who do not offend because of their mental illnesses, *per se*, but due to the concomitant social difficulties that all too often accompany mental illness. The final general group of offenders with mental illness include those patients whose offending occurs irrespective of their mental illness. Each of these groups will be described below.

Patients who offend because of their mental illness

This group is likely the smallest of the three groups. This group includes people who may not be criminally responsible because, as a result of their mental illnesses, they do not know what they are doing, or do not appreciate that what they are doing is wrong. Their offences occur as a direct result of the mental illness. But for the mental illness and the presence of symptoms which led to the patient's offending behaviour, the crime would not have occurred. Their mental illness is both a necessary and sufficient explanation for their offence. They only offend when they are acutely unwell and the offence behaviour is a product of their mental illness (e.g. acting on delusions or hallucinations). Depending upon the jurisdiction in which they reside, they may be found not guilty by reason of insanity or mental illness. They most likely will be housed in secure hospitals rather than prisons following legal adjudication. Typically the illnesses that are present in people who fall into this category are psychosis or serious affective disorders accompanied by psychosis. Many jurisdictions that retain some form of insanity defence specifically exclude the use of the defence by those with antisocial or dissocial personality disorder.

Patients who offend as a result of the sequelae of mental illness

The second general group of psychiatric patients who offend comprises hose whose mental illnesses are a necessary but not sufficient explanation for their offending. It is by far the largest group of psychiatric patients who offend. As is typical for many patients with serious mental illnesses, these patients begin to spiral downward socially as a result of their mental illnesses. They can become estranged from family and pro-social support networks. Their lives become unstable; housing, basic needs, and their need for non-judgmental personal support may go unmet. They may end up being accepted by groups of people who are themselves unstable. They often resort to engaging in illicit drug abuse. These social factors contribute to their resultant offending. While their mental illness may be a catalyst in the course of events that lead to the offending, the mental illness itself is not the direct cause of the offending. Had they not had a mental illness, they likely would not have begun offending. However, by the time they develop offending behaviour, their lives have become so disorganized and their maladaptive coping and survival strategies have become so entrenched as to make the reversal of these processes difficult over the long-term. Psychiatric treatment, while a necessary starting point, will not be sufficient alone to eliminate the offending behaviour.

Patients who offend despite their mental illness

The final group of patients are those who would offend irrespective of the fact that they have a mental illness. Although not as large a group as the one above, many more patients who offend fall into this category than into the first. The fact that they have a mental illness is neither a necessary or sufficient explanation for their offending. Patients in this group are typically characterized by early onset antisocial and illegal behaviour. They differ from other mentally ill offenders by having a pervasive and stable pattern of offending regardless of their mental state.[5] This behaviour almost always precedes the onset of mental illness. While people with a psychopathic or dissocial personality disorder will be included in this group, most of the people in the group will not be so disordered. It is important to acknowledge, though, that the broad range of people that may fall into this group, including the psychopaths, may well develop psychiatric illnesses. We must avoid the tendency to deny this group proper services or to acknowledge their mental illnesses. These patients' mental illnesses may well exacerbate their offending or lead to unusual offending; however, even when they are asymptomatic they may continue to offend.

Aren't psychiatric patients with offence histories unusually burdensome or too dangerous for mental health services?

The perception all too often still exists that patients with offence histories are unusually burdensome or even too dangerous to be seen by general mental health services. While there are doubtless patients, largely those drawn from the third group above, who are burdensome and even dangerous, in the main patients with offence histories are nether unduly burdensome nor dangerous. For example, in a recent prospective study of violence among discharged general and forensic psychiatric patients, Doyle and Dolan[8] found no

[1] Readers are referred to Chapter 11.3.1 (Associations between Psychiatric Disorder and Offending by Thomson and Darjee) for additional information regarding the relationship between mental illness and offending.

significant differences in post-discharge violence rates (both official and unofficial) between patient groups in the UK. Ogloff and colleagues have obtained similar results from separate studies of post-discharge violence among samples of general and forensic psychiatric patients.[6]

One of the concerns expressed in general psychiatric services about patients with offence histories is the risk for aggression and violence they might present during hospitalization. It is often assumed that if a patient has an offence history, particularly one marked by aggression, that the patient will be more likely to be aggressive in hospital. Research suggests, however, that this may not be the case. It is true that over the entire period of hospitalization patients who have more psychopathic traits might have higher rates of aggressive incidents.[9] Research shows that in fact there is no significant relationship, at least for forensic psychiatric patients, between aggression in hospital, aggressive behaviour preceding admission, or violent recidivism.[10]

Analyses of what leads to aggressive behaviour by psychiatric patients suggests that dynamic (highly changeable) factors are responsible and that a functional analysis of inpatient aggression shows that rarely are the acts related to general patient aggression or purely to the patients' mental state.[11] Rather than assuming that patients with forensic histories will be any more or less aggressive than other patients, recent instruments have found useful in assessing patients risk for inpatient aggression.[12] Such instruments should be employed.

Assessment of psychiatric patients who offend

Prior to commencing ongoing mental health care to patients with offence histories, it is important that a comprehensive assessment be conducted, preferably by a psychiatrist or clinical psychologist with expertize and experience in forensic mental health. In some jurisdictions, mental health services may be able to draw upon forensic mental health services to obtain secondary assessments of the patients to assist with assessment and treatment planning.[13] The assessment must address three major components: mental health, substance use, and the presence of criminogenic factors (i.e. factors that increase the likelihood that the patient will re-offend).

First, a thorough mental health assessment is required that includes both a review of the patient's current mental state as well as their psychiatric history. Although seemingly straight-forward, this can be difficult with some patients who have offence histories. All too often now we see young people, usually males, whose mental illnesses are only identified upon admission to gaol or prison.[3] As such, it may be difficult to obtain reliable information about the genesis and onset of these people's mental illnesses.

The second component that must be considered is whether the patients have a substance use or dependence disorder and what role substances have on their mental illness and offending. In mental health generally,[14] and in patients with offence histories in particular,[15] high percentages of patients are substance abusers. Ogloff and colleagues found that 74 per cent of patients in the secure forensic hospital in Victoria, Australia had a lifetime history of substance abuse or dependence. The presence of a substance use disorder is a key risk factor in determining which patients will re-offend (or have a relapse of their illness for that matter). Unfortunately, very often substance use disorders, and their effects on patients, are overlooked in the routine assessment and treatment of patients with mental illnesses.

The final area that must be considered in a comprehensive assessment is the presence of so-called 'criminogenic factors' present in the patient's case. This concept is part of a contemporary well accepted and supported theory of offending known as the Psychology of Criminal Conduct, which was developed by Andrews and Bonta in the 1980s and it has been refined over time.[16] It is a theory concerned with individual differences and variability in criminal behaviour, making it a particularly useful guide for both assessing the risk of reoffending and planning rehabilitation attempts. This emphasizes the complexity of criminal behaviour, thereby acknowledging the contributions of social context, biology, and psychopathology. Criminogenic factors are the subset of dynamic (changeable) risk factors that have been found to relate directly to a risk for re-offending. They are therefore modifiable characteristics, whereby a change in the risk factor equates with a change in the risk of re-offending. These are factors that can affect patients with mental illness just as they can affect people with no mental illness who offend. Examples include having friends who are criminals, developing pro-criminal attitudes, having an anti-social personality, having limited problem-solving skills, and having difficulties controlling anger and hostility (Ogloff & Davis, 2004).[17]

To assist in assessing Andrews and Bonta[18] have developed the *Level of Service Inventory, Revised* (LSI-R), which assesses the presence of criminogenic factors as the basis for offender assessment and treatment.[1] The LSI-R consists of 54-items "grouped into the following domains or sub-components (with the number of items in parentheses): Criminal History (10); Education/Employment (10); Financial (2); Family/Marital (4); Accommodation (3); Companions (5); Alcohol/Drug Problems (9); Emotional/Personal (5); and Attitudes/Orientation(4).[18] While developed for general criminal populations, the LSI-R has been found very useful for assessing the presence of criminogenic factors and general needs of psychiatric patients with offending histories. Recent research findings show that a screening version of the LSI-R reliably identifies risk factors for patients in forensic psychiatric services.[19]

The presence of antisocial personality or dissocial personality is a criminogenic factor that must be considered in the assessment of mentally ill offenders. Unfortunately, antisocial personality disorder as it is defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision[20] is vastly over-represented in psychiatric populations due to the nature of the criteria for the disorder which are essentially the presence of criminality.[21] Thus, great care must be taken to ensure that the diagnosis of antisocial personality does not rely solely on the fact that the patient has a history of offence behaviour. Instruments designed to reliably measure the presence of psychopathy, such as the *Psychopathy Checklist*, can be useful for assessing the aspects of personality and behaviour that comprise psychopathy.[21,22]

Following the assessment of each of the above components, it is necessary to develop a formulation that considers where the

[1] There is a revised version of the LSI-R which includes a section for case management planning, the Level of Supervision/Case Management Inventory and a version for young offenders (Youth Level of Service/Case Management Inventory; Hoge, Andrews, & Leshied, 2002).

patient's mental illness factors into their offending. Drawing on the three typologies of offenders with mental illnesses outlined above, the clinician can determine which category best describes the patient. Because the typologies are general, there will be overlap in characteristics for some patients. In addition to understanding the factors that help explain a patient's offending, the typologies are very important for determining what treatment and management strategies can be most effective for the patient.

The treatment of mentally ill offenders by general mental health services

The typology of mentally ill offenders will be revisited below with respect to the mental health and related services they require to assist with their treatment and management. The three-prong assessment strategy briefly described above will be helpful to identify the range of treatment needs the patient has. To be clear, the primary and even sole purpose of general mental health services is to treat patients' mental illnesses. However, it is useful to consider the presence of an offence history as an indication of the patient's functional impairment. Depending on the group into which the patient falls, the relative efficacy of mental health services alone varies in the extent to which it will satisfactorily address their mental health and offence issues. Most often, particularly for the latter two groups below, ancillary services and forensic mental health services will be required to help ensure the patient's long-term stability and to reduce the likelihood of offending.

Patients who offend because of their mental illness

Although this group of patients may commit horrendous acts, it is as likely that they engage in nuisance offences. Despite the particular type of offending behaviour in which they engage, perhaps surprisingly, their management by mental health services is oftentimes less complex than is the case for the other two groups of offenders with mental illnesses to be discussed below. Generally speaking, the treatment that this group requires is conventional mental health care. As it is the case that the primary cause of their offending behaviour is the mental illness, and the symptoms that it produces, addressing their mental health needs can serve to eliminate the offending. General mental health services are generally well equipped to deal with these patients, though they may feel reluctant to do so. Very often, patients will respond to medication and with supervision their mental state will begin to improve. If treatment in the cases is complex, it will often be because of the mental illness itself. For example, the patients may have chronic psychosis which is refractory to psychiatric treatment.

While the patient's mental illness is the main cause of the offending behaviour in this category, related issues will need to be addressed to stabilize the patient over the long term and to further reduce the patient's risk of re-offending. The LSI-R, noted above, will be particularly helpful in identifying such issues. Common issues include substance abuse, life skills, housing, financial support, and personal support. Services to address these issues will need to be organized to effect long-term psychiatric and behavioural stability.

Patients who offend as a result of the sequelae of mental illness

Just as the complexity of the reason this group offends is greater than with the first group, the treatment they require to stabilize is also more complex. This group is characterized by general disorganization and social damage. As such conventional mental health services alone will have relatively limited effect on patients' mental state and stability over the long-term. Even if psychiatric treatment is effective in the short-term, patients in this group will be likely to return to a chaotic life which eventually may include a return to offending. Nonetheless, the treatment of these patients' psychiatric illnesses is the central component of their care.

The comprehensive assessment approach outlined above will be particularly useful in determining the range of issues beyond mental illness that affects the patients and contributes to their offending behaviour. In particular, the areas of concern identified by the LSI-R are particularly important for informing intervention need. For example, if employment issues, financial issues, accommodation needs and alcohol/drug problems are revealed in the assessment, these issues, in addition to the patient's mental illness will need to be addressed. Not only will addressing these issues satisfactorily lead to a reduction in the patient's risk of re-offending, but it will assist with ensuring stability in mental state over the long-term.

Generally speaking, the greater the number of criminogenic factors that arise from the assessment, the more intensive treatment will need to be to ensure long-term stability. To the extent that needs arise that cannot be addressed directly by the mental health service, these services will need to be sought from appropriate providers in the community. This is where effective case management and service brokerage is critical. All too often psychiatric patients revolve in and out of general mental health services (and the criminal justice system); all the while their underlying needs are not identified or addressed. The vast majority of offenders with mental illnesses can be properly treated and managed by general mental health services if only their related needs and issues can be addressed. Moreover, given that there is a relationship between these patients' mental illnesses and their offending, addressing the mental illness and related matters can help lead to a reduction in offending, although that will not be the purpose of providing them with general mental health services.

Patients who offend despite their mental illness

As with all of the categories of offenders, this group will still require comprehensive mental health care; however, the mental health care will be essentially futile in reducing the patient's proclivity for offending. It is important to note that mental health services still have an obligation to treat these patients' mental illnesses, but addressing their offending issues will be beyond the scope of care or even the expertize of general mental health services. Moreover, addressing the ancillary issues that arise to affect their mental illnesses will be less likely to reduce their offending risk than would be the case for either of the other two groups above. Where possible, patients in this group should be seen by forensic mental health services and they will be candidates for offender rehabilitation programmes offered by contemporary correctional services (in prisons or in the community).

It is important to note that the cautious approach advised is not intended to dissuade services from providing adequate psychiatric care, but to recognize their limitations and to reduce the sense of failure and frustration that occurs when treating patients with such an offence pattern. Despite the nihilism that sometimes exists, there is an ever expanding corpus of firm empirical support to

show that offender rehabilitation can help reduce recidivism,[17,23] however, such services are beyond the scope of general mental health services. Over time, of course, as these patients' offending issues may be successfully addressed, they will be appropriately cared for by mental health services just as other people are. Once an offender not always an offender!

The role and support of forensic mental health services

The thorny question of when forensic mental health services, as opposed to general mental health services, ought to be responsible for a patient's psychiatric care is difficult and depends much on the jurisdiction, the relevant legislation, policies, and practices that are in place. It is never the case that all patients who offend require, or should have access to, forensic mental health services. For the most part, the goal should be to maintain patients in general mental health services to ensure continuity and normality of care. Realistically, though, given the relatively high rate of psychiatric patients who offend or who have offence histories, the level of knowledge and awareness of offence issues among general mental health professionals must increase.

It is still the case in many settings that the mere mention that a patient has a forensic history raises angst and concern about the capacity of general mental health services to care for the individual. This is most often nonsensical, particularly because a relatively high percentage of psychiatric patients in general services have an offence history—whether or not it is known by the service. Ideally, general mental health services should adequately address patients' mental health and ancillary issues to an extent that would actually prevent patients in the first two groups of mentally ill offenders from offending. Very often we in forensic mental health services see patients who had contact with general mental health care yet their problems were exacerbated, they deteriorated and went on to offend. Oftentimes inadequate assessment and identification of the patients' needs is at the core of the shortcoming of their care.

Realistically, despite the oftentimes excellent care provided to psychiatric patients, forensic mental health services will be required. In the first instance, patients found not criminally responsible or who require involuntary treatment during incarceration should be provided service in appropriate forensic mental health facilities. Psychiatric patients with complex presentations, including myriad criminogenic issues, likely will require at least secondary consultation from a forensic mental health service.[13] The best models that exist internationally have forensic mental health services take responsibility for the complex offence-related cases initially with the introduction and eventual transition to general mental health services over time once the patient's offending issues are addressed and stability is realized.[24,25] Continuing general care is the preferred modality of care except in the most difficult cases.

Conclusions

General psychiatry has an important role in providing care to all patients, and under most circumstances this includes patients with offending histories and issues. Given the relatively high rates of offending among psychiatric patients, whether they realize it or not, general services have considerable experience with this patient group. All too often, though, general mental health clinicians do not have adequate training or expertize to systematically assess patients to determine what factors have lead to their offending. The typology of patients who offend presented in this chapter can prove useful for determining the factors that must be addressed to treat the patient. Moreover, an indication will be made as well when assistance may be required by specialist mental health services.

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11.17

Management of offenders with mental disorder in specialist forensic mental health services

Pamela J. Taylor and Emma Dunn

Philosophy and theoretical models

Specialist forensic mental health (fmh) services are for people with serious mental disorders and grave offending behaviour who tend to be rejected from mainstream services. Although often triggered by single high profile cases, these specialist services are among the best planned and commissioned services in psychiatry, founded in evidence of need, risk and efficacy of interventions. They are grounded in a multidisciplinary clinical perspective and often have integrated academic units. They interface both with other clinical services and with the criminal justice service. Good relationships with the local community are vital for establishment and growth.

Mentally disordered offenders have been sources of tension between services at least since the early 19th century. In Britain, the Lunacy Commission argued that it was 'highly objectionable' that offender patients should be detained in a general lunatic hospital, while an 1807 parliamentary select committee noted that 'to confine lunatics in a common Gaol is equally destructive of all possibility of the recovery of the insane and the comfort of other prisoners'.[1] High security hospitals followed about 50 years later.

Funding and commissioning of fmh services worldwide continue to follow oscillations between considered responses to changes in the structure and availability of general services, and responses to single notorious cases. For England and Wales, the Butler Report[2] considered the then increasing gaps in service provision as psychiatric services shifted from mainly institutional to mainly community care. It was the most thoughtful and powerful driver of modern forensic mental health services in England and Wales, presaging the arrival of medium security hospital units. The contrasting path, of case driven service development, is illustrated by the so-called 'Dangerous and Severe Personality Disorder' (DSPD) services,[3] driven by the inquiry into the care and treatment of Michael Stone[4] following his conviction for two homicides and an attempted murder, occurring soon after his discharge from a psychiatric hospital. In the USA, mandated community mental health law (Kendra's Law) and treatment programmes in New York followed a subway killing by a psychotic man,[5] while legislative change necessary for specialist service development in Japan followed a school massacre.[6] Concern that single cases make poor law – and poor health reforms—is tempered by the mutual commitment of government agencies and of practitioners to keep offender patient services under review.

The Department of Health and Home Office report, for England,[7] is a good example of such review, proposing five principles for secure healthcare provision:

i) quality of care and proper attention to individual needs;

ii) community rather than institutional care where possible;

iii) security no greater than justified by the danger presented—to self or others;

iv) maximization of rehabilitation and chances of sustaining an independent life in the longer term;

v) proximity to the patient's own home or family if s/he has them.

It is arguable that only the fifth principle requires an evidence base. Intra-familial violence may contribute to mental health and/or behavioural difficulties, and most violence by people with mental illness occurs within their close social circle.[8] Nevertheless, people have attachments, and the fifth principle is retained for that reason—*and* as a convenient way of anchoring responsibilities for services. The first four principles embody the medical ethic of maximizing autonomy and anticipated the Human Rights Act 1998, which gives effect to the rights under the European Convention to *liberty and security of person* (article 5) and prohibits *degrading treatment* (article 3). The Act also emphasizes proportionally—if it is necessary to breach a right, that breach should not go further than necessary.

Organizational models often founder in a clash between the needs of service users and providers. The ideal is a fully integrated services in which service users move freely between forensic and general mental health services, according to need.[9] General services, however, tend to have to focus on crisis management, and the greater the tensions between specialist and general services the greater the likelihood of ever-longer periods of residency in a physically secure hospital[10] or default to parallel service delivery

reasons for this include reduced availability of non-secure psychiatric beds, perverse incentives in funding in which those in highest security may be funded centrally with minimal local funding burden. Also government caution may dictate that once detained in medium or higher security, every individual must progress stepwise through lower levels of security before returning to the community. Evidence does not support the notion that this stepwise route reduces criminal recidivism,[10] but it has led to the growth of additional tiers of specialist security provision, including 'low security' hospital units and forensic community mental health teams.

The international context

There is insufficient space to explore the international context in any detail. Laws on criminal responsibility, criminal justice and mental health vary between European countries[11] and elsewhere, but many underlying clinical principles are shared. Most countries acknowledge some association between mental disorder and offending behaviours, but there is variation in how this influences prosecution, fitness to plead and stand trial, the extent to which mentally disordered offenders may be regarded as wholly or partially without responsibility for a criminal act, and the extent to which they are treated in mainstream or specialist secure health services, or in prisons, albeit with some health service input.

Our international research group (SWANZDSAICS) drawn from culturally distinct jurisdictions across five continents (Sweden, Wales, the Australian state of Victoria, New Zealand, Denmark, the South African province of Western Cape, Japan, the Canadian province of Quebec, and Scotland) finds a shared therapeutic philosophy in managing offenders with psychosis, but struggles to be therapeutic with sex offenders or people with personality disorder.[12] Other countries however, most notably the USA, seek to separate the business of psychiatry in the courts from any therapeutic endeavour with mentally disordered offenders.[13]

The nature of security

Secure psychiatric hospitals have two overarching aims: improving health and delivering safety for patients and others. In secure hospitals, patients' autonomy is limited in a number of important ways: they may not be allowed to leave the hospital at all, may be confined to a particular area within the hospital, and/or treatment may be enforced. Although these restrictions are undoubtedly at least partly in the interests of the patients themselves, they are commonly also in the interests of others.

The elements of security

Security in a clinical setting is made up of four main elements: physical, procedural, and relational security, *and* treatment. Treatment, including (re)habilitation, becomes vital to safety and security whenever a clear pathway can be shown between a mental disorder and offending behaviour.

Physical security refers to the qualities of buildings: the nature of perimeter walls and internal structures and functions. High security requires at least one high and distinct perimeter wall or fence clear of the main hospital building. In medium-security, the walls of the building alone generally provide the main perimeter, with high fences only surrounding exercise areas which are not entirely within the main building. All specialist security hospitals provide for staff and visitor entry through an 'airlock', using independent locking systems, the external one generally controlled by dedicated security or administrative staff. Ideally, clinical staff contributing to building design, which should ensure good sightlines throughout, while allowing residents a sense of privacy. Each patient has his/her own room, and ideally holds a key to it (with a staff over-ride potential). This enhances his/her safety and sense of personal security, and also the safety of property. In high-security units, cameras may be used for continuous monitoring. The environment should be pleasant, enabling both patients and staff to feel comfortable; small frustrations often trigger violence.

Procedural security provides for a formal set of checks for factors thought to be associated with risk of harm by patients. This includes minimizing patient access to weapons, fire-setting materials, or potentially disinhibiting substances, and preventing absconding. In high security, any communication with the outside world may be monitored; at lower levels of security, such monitoring is determined case by case. Procedures should also guard against potential harm to each patient. Some measures used to prevent or control violence may have 'side effects'. Time-out and seclusion may be necessary, but can be provocative and open to abuse. Physical restraint may sometimes be essential, but if done incorrectly or brutally may damage the possibility of a therapeutic relationship, physically harm or even kill the patient. In the UK, procedures for such measures are subject to guidance both from professional bodies[14] and legislative Codes of Practice (e.g.[15]).

There is insufficient space to detail the extensive range of procedures for ensuring security, so a couple of examples—searching and screening of contacts—must suffice. Searches of the person and of the environment are conducted mainly to minimize access to drugs and weapons. The level of unit security dictates the nature, extent and frequency of searches. In English high security hospitals, no-one is trusted. Staff, professional visitors and social visitors are all searched on entry; many items—such as mobile/cell phones are forbidden anywhere in the hospital. Patients may be searched randomly, but also when moving between areas in the hospital or if there are particular grounds for suspecting they have secreted something that could become a weapon, or acquired drugs. At any security level, patient rooms and other areas may be searched—similarly, randomly or on specific grounds; in high security, patients' possessions are routinely restricted in quantity to facilitate searching. For all such occasions, however, procedures incorporate measures which reflect concern for the individual being searched. Patients must be informed of searches (immediately beforehand if randomly timed) and invited to observe.

Screening of contact with visitors is multifaceted. Visitors may be enticed into aiding absconsion, or be irresponsible in their 'gifts' for the patient; apparently innocuous items may be fashioned into weapons, and they may be under pressure to bring drugs, perhaps disguised in food. There may also be risk of harm to visitors. Telephone calls, mail and personal visits may all be observed, but only in accordance with written procedures. Policies pertaining to visits will refer to classes of visits—for example visits by specific individuals who may threaten or be under threat, or by children, Such visits must be supervised by specifically trained staff.

Relational security skills are founded in therapeutic approaches and, with specific treatments, form the core of hospital security, clearly demarcating hospitals from prisons. It lies in extensive knowledge of each patient, accurate empathy and highly developed

capacities for communicating and working in a clinical team. At best, it not only provides immediate safety and the milieu for change, but it may also facilitate lightening of physical and procedural securities. Effectiveness, however, is reliant on sufficient numbers of adequately trained staff.

Relational security may, however, create anxiety in hospital managers and their political masters, partly because it is more difficult to understand as security than locks and walls, but also due to the perception that its corruption is possible and difficult to predict. Over time, staff may be vulnerable to potentially counter-therapeutic change.[16] Strategies to ensure maintenance of clinical integrity therefore include personal supervision and appraisal, peer review and audit of team- and hospital-wide practice. Access to psychodynamic psychotherapists is not only, or even primarily, for the patients, but also for the staff and the institution.[17]

Treatment as security targets the link between symptoms of mental disorder, most obvious for psychotic symptoms, and criminal or risky behaviour.[18] In contrast to prisons, secure hospitals generally select residents for their treatability. It seems simple then—specific treatment with antipsychotic medication for people with psychosis should bring safety—but matters are rarely so straightforward. Multiple diagnoses are common: at least 25 per cent of offender patients with psychosis have personality disorders established before onset of their psychotic illness, and many abuse alcohol and/or other drugs at levels to qualify for a diagnosis.[18] Over 25 years, an increasing proportion of English high security hospital patients were found to have substance misuse disorders,[18] especially affecting the psychosis-personality disorder group. Substance misuse not fully meeting diagnostic criteria is also common. In the short term, specific treatment for psychosis combined with preventing access to substances of abuse can restore safety. For longer term success and safety, specific treatments aimed at substance misuse are best integrated as part of the overall treatment package,[19] although this is still not common practice (e.g. UK,[20] and Sweden,[21]). This may partly explain the counterintuitive finding[22] that, in the UK at least, there is a preference for admitting people with 'pure' psychosis to medium security hospitals, even though substance misusing people with psychosis would be regarded as a higher risk group (e.g.[23]).

Hospital security in practice

In England and Wales, *high security hospitals* are mandated in statute for those patients who pose an imminent risk of serious harm to 'the public'. Public safety is often construed merely as removing dangerous persons from the community and strictly confining them. During the early stages of treatment adequate protection for staff and other residents is also vital. As patients become apparently safe, it is important that they can be tested out before discharge to the community, but government restrictions often make this difficult, so that patients may arrive at lower security levels ill-prepared for the new challenges; this may be dangerous in itself.

Medium security can be defined only in relation to high and low security services above and below it. The range, quality and quantity of low secure and open provision varies from place to place, so that providers of some medium secure service may have to retain patients longer than other providers and/or provide more parallel services.

The most constant aspect of medium security services is the in-patient unit. 'The aim is for the building design to support the nursing staff, who are the main security barriers.'[24] Procedural security tends to be less stringent than in high security, but relational and treatment security are generally comparable. Nurse: patient ratios should be at least 2:1, and staff from a full range of other clinical disciplines should be available.

Low security relies almost entirely on relational security, but units generally have a locked door, and some procedures relevant to security, such as monitoring of substance misuse.

Service structure

Planning principles

'*Pyramid planning*' applies to specialist fmh services: the most intensive, specialized and secure services, most of which constrain patients' liberty and are the most costly, should be the smallest and at the top of the pyramid. At the base of the pyramid, and greatest in number, should be services linked directly to the local community. Trends in service provision in England and Wales reflect this model, with high secure bed numbers falling, and an increasing number of medium secure services (in mid-2007, 800 and 3 500 respectively for 55 million general population). Low security service provision is also increasing.

Private healthcare facilities. It is generally regarded as unethical to profit from the indefinite institutional detention of people. In Japan, where private provision of mental healthcare was the norm, recognition of service gaps for mentally disordered offenders resulted in the provision of publicly funded, purpose designed facilities for such patients.[6] In the UK, independent provision supplements a shortfall in specialist NHS facilities, providing just under half of all forensic beds. There are several reasons for this, not least the greater flexibility in service planning enjoyed by independent than public sector providers. Nevertheless, all places for detained patients in the UK are publicly funded and independent service providers are subject to the same levels of scrutiny as NHS facilities.

'*Super-specialist*' *provision* is necessary to provide specific treatments and the appropriate milieu for people with some special needs, such as female offender-patients, who almost invariably have suffered prolonged and serious neglect and abuse through childhood. Approaches founded in attachment theory[25] or trauma based work[26] go some way towards meeting their needs. People with a learning disability also benefit from dedicated services,[27] as may children and adolescents.

While there are advantages to specialist developments, most groups needing them tend to be small, and hence there are few units and it is difficult for residents to retain close ties with home, e.g. women resident in the only English high secure provision may be more than 350 miles away from home.

Planning service capacity is difficult. It is often done from utilization figures, but these tend to underestimate requirements by failing to recognize unmet need. While most developed countries have shifted general psychiatric services away from inpatient provision to the community, they have also seen an upsurge in availability of legal and illicit mind-altering substances. This has the probable effect of raising violence levels not only among the mentally healthy, but also among those with mental disorders (e.g.[28]).

Prison populations are growing in many countries. In the USA and the UK indefinite sentences 'for public protection' have been implemented. In England and Wales, the Lord Chief Justice and the Chairmen of the Parole Board have projected an additional

12,000 prisoners under this sentence alone by 2012, all of whom will need some sort of 'treatment' before they can be released[29] (at the time of writing a legal challenge to the lawfulness of such sentences in the absence of sufficient treatment is underway). Surveys of mental disorder among UK prisoners(e.g.[30,31]) suggest that neither generic nor specialist mental health services have kept pace with need in prisons even before this new expansion.

Capacity planning must be dynamic, taking into account assumptions about other services, and socio-economic differences between communities. Statistical models may help, (e.g.[32]) but it is more difficult to allow for changes in sentencing policy.

The service users: assessment, management, treatment and rehabilitation

Principles of assessment

Assessment in forensic psychiatry is considered in Chapter 11.14. In some countries, such as the USA, assessments for the courts are the main tasks for forensic psychiatrists, with some specializing in just one type of assessment and few providing treatment services.

Pre-admission assessment is important for patients destined for a secure hospital. It informs the initial treatment plan, and ensures a safe case-mix and appropriate staffing. Patients admitted to a secure hospital must have a mental disorder which meets legislative criteria for detention. Security requirements are then based on assessment of both mental disorder and criminal/violent behaviour. External factors must also be considered, for example, assessment of victim vulnerability. If, at one extreme, the identity of patient A's potential victim and the circumstances in which s/he might be at risk are clear, and that putative victim can acknowledge and co-operate with safety strategies, then it may be possible to manage risk of harm with little recourse to physical security. If, however, patient B threatens named people who cannot understand, accept or help manage the threat, then physical security may initially be required for her, even though both patients are otherwise similar. A serious, more generalized threat may also mean that initial assessment must be in security.

Admission criteria

Broad agreement on criteria for admission to a secure hospital bed[33] does not necessarily lead to consistency in practice.[22] In England and Wales, only 40–50 per cent of people referred for a specialist secure hospital bed, whether in high[34] or medium security[22] are actually offered one. There are common reasons: i) that some disorders are not widely considered to be treatable, and ii) the high level of security requested is thought not to be necessary. In spite of powers in England and Wales to detain people with personality disorder for assessment or treatment, many medium security beds are effectively closed to them. Few are now admitted to high security hospitals, except to the new purpose designed 'DSPD' units. It seems to be only in the Netherlands that there is a preference for treating people with personality disorder in secure hospitals.[35] There are few data on which to judge the quality of such assessments, but one study showed that 85 per cent of those refused a high security bed remained outside high security during the following 12 months, without incident.[36]

There have been efforts to attain standard ratings of security need. Cohen and Eastman[37] generated a set known as the Admission Criteria to Secure Services Schedule (ACSeSS). Factors considered relevant to admission were (* indicates factors most used in determining admission):

- gravity of recent* and past violence;
- likely immediacy of violence;
- psychopathology/developing behaviour possibly predictive of violence;
- special pathology, for example PD or sexual offending;
- the likely longevity of risk of violence*;
- predictability of the gravity and immediacy of potential future violence.

Collins and Davies[38] developed the Secure Needs Assessment Profile (SNAP), designed for *clinicians* to operationalize clinical judgement, following the first three elements of the security matrix outlined above—need for perimeter, and procedural or relational security.

The practice of using structured aids for clinical assessment of risk of harm to others or to self is growing, with nursing staff finding them particularly attractive. The HCR-20 (20 item Historical and Clinical Risk Management instrument;[39] includes dynamic risk factors, which can both inform management and provide a baseline measure from which progress can be monitored.[40]

Progressing along the pathway to treatment.

When someone has one or more serious mental disorders and poses a serious risk of harm to others, assessment may become a lengthy process. The elements of this must in turn be evaluated and adjusted according to interim outcome measures. Figure 11.17.1 summarizes a typical pathway. The initial assessment is directed at establishing safety—either through advice to others, or through admission to a specialist setting. In the latter, the assessment merges seamlessly into engagement into treatment, directed first at the primary disorder, before working with underlying factors—such as experience of childhood trauma.

The concept of 'treatability'. Under mental health legislation for England and Wales, patients legally classified as having *psychopathic disorder* or *mental impairment* alone cannot be detained for treatment in hospital unless 'such treatment is likely to alleviate or prevent a deterioration' of the condition. This intended safeguard against improper detention has sometimes been used to reject 'difficult' patients. New mental health legislation does not distinquish disorder types and puts the burden on availability of treatment.

Treatability should not be confused with curability. Assessment of treatability should be made in full awareness of the assessor's own attitudes and fears, with primary consideration given to the patient's psychopathology, needs, insight, and preferences and motivation for treatment. The timing of such an assessment may also be crucial. If the consequence of no treatment is more-or-less certain, for example, imprisonment, and this will most likely cause deterioration, then this in itself could justify admission for hospital treatment.

Principles of treatment. First line treatment of mental disorder in a secure setting generally differs little from that used elsewhere in mental health services. It is important to be aware, however, that some treatments have never been systematically evaluated among people whose mental disorders are complicated by violence. For example,

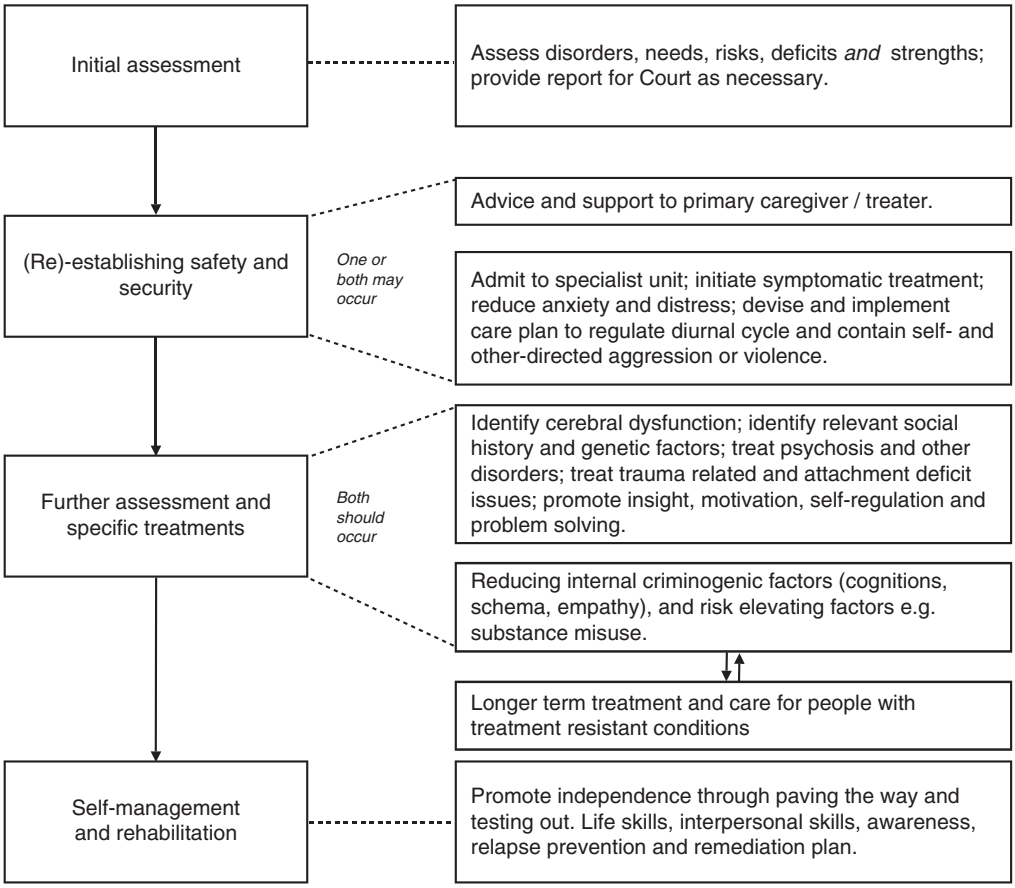

Fig. 11.17.1 Schematic representation of typical treatment and care pathway.

some consider that caution should be exercised in the use of cognitive behaviour treatment to alleviate psychotic symptoms.[41]

Delivery of treatment in secure conditions differs in some important ways from its delivery elsewhere. Intrusive interventions are not unique to forensic mental health services, but the length of application means that staff may need to engage in regular peer and external review to ensure their work remains effective and ethical. Areas for concern include the creation of a climate of compulsion and coercion, and the conflicting roles for psychiatrists using therapeutic skills for legal assessments and dangerousness predictions, as well as true therapeutic engagement.

The *inpatient environment* must be conducive to habilitation and rehabilitation. As well as treatment of specific disorders, the core concern of staff is aiding independent living. A sense of progress, evident in both the physical environment and staff attitudes, encourages hope and consolidates treatment gains. This sense of progress can be achieved in several ways:

- provision of graded living spaces in which patients can experience greater autonomy;
- increasing engagement in work, education and recreation;
- extending responsibility for personal and domestic care tasks;
- phased introduction of supervized activities outside the institutional boundaries, appropriate to the patient's mental state and public safety;
- consideration for and support of family and social relationships, and a basis for developing sustainable social networks. Intimate relationships constitute an important element of this aspect of the work.[42]

Successful return to the community depends on everyone having a sense of security. This will require suitable accommodation, basic financial provision and skills for managing finances, a sense of control over mental disorder, and sufficient mastery of local services to be able to find help in a crisis. Regular reviews of the service user, the services, their use of these services, and the goodness of fit between them, are essential. People regarded as posing a special risk of harm to others may be referred to a multi-agency public protection panel (MAPPP, [43, 44]; see also Chapter 11.16).

Direct involvement of the patient in setting goals for health and for safety creates in the patient a sense of ownership of resultant plans, and is likely to improve collaboration with them. The process of community re-entry is summarized in Fig. 11.17.2, drawing on a grounded theory of discharge from a secure hospital.[45] It lends itself to the development of outcome measures which are more appropriate for an offender *patient* than the almost exclusively used but over-simplistic measure of re-offending. The core concern is movement between pathological dependence and healthy independence, with staff taking an active role in facilitating progress in two phases—paving the way, when skills necessary to the attainment of independence are built up, and testing out, when

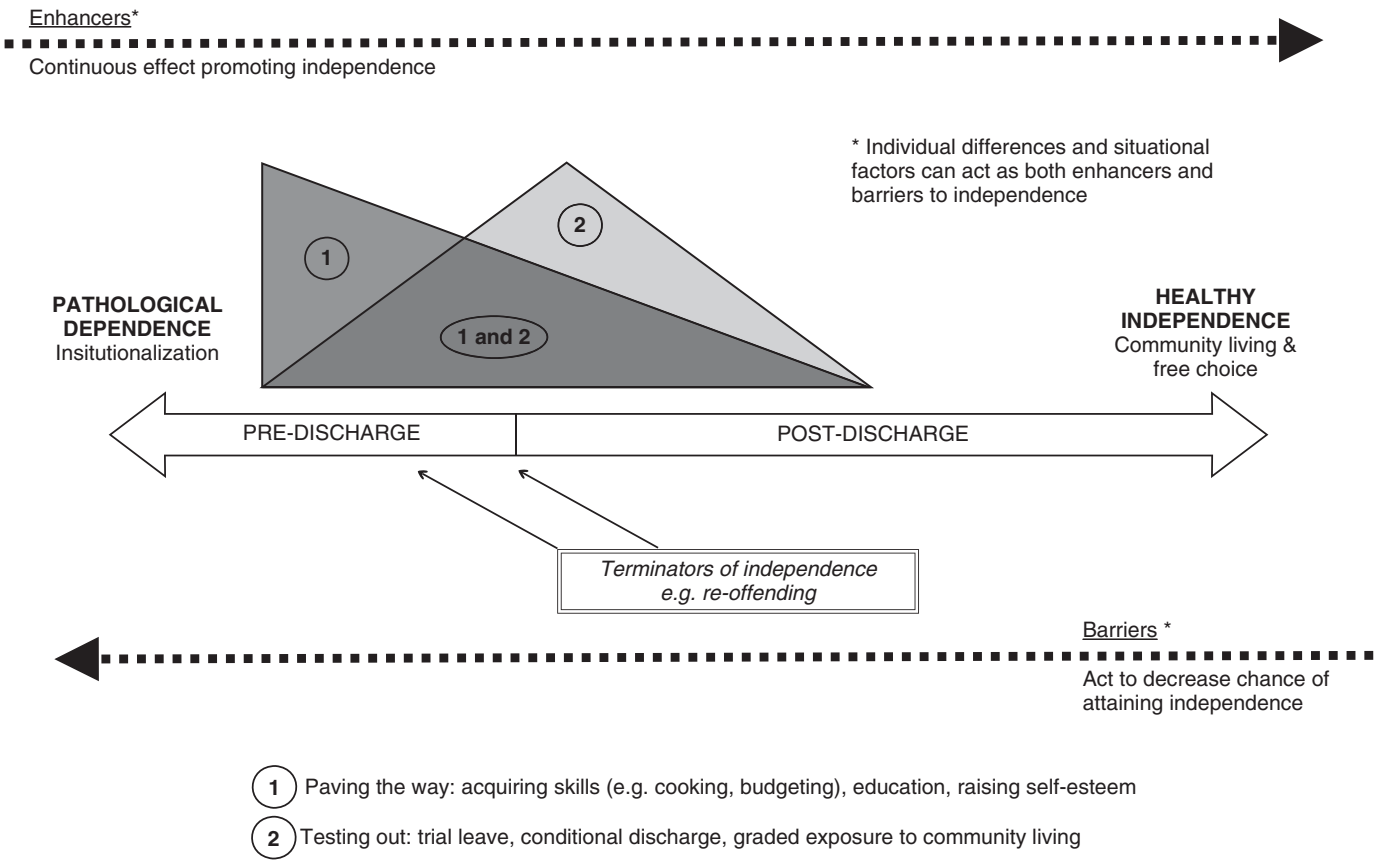

Fig. 11.17.2 Attaining healthy independence while recovering from serious mental disorder and offending: A model.

individuals are given opportunities to prove themselves in situations of increasing trust. Re-offending is important, but as a barrier to gaining or sustaining independence rather than the sole outcome indicator.

Assessing outcomes

Most studies of people who have been discharged from specialist security hospitals have serious limitations, which partly arise from political pressure to focus on the non-clinical outcome of re-offending and inadequate funding of the studies, which are costly and time-consuming. Results of existing studies are limited by:

1 Choice of outcome measures, which are almost solely re-offending, with some reference to mortality and hospital re-admission.

2 Lack of information about the nature of treatment. At best, there is information about length of stay in the institution. Some effects may be inferred, for example the complex model for treating personality disorder in Broadmoor Hospital in England,[46] but most studies simply treat secure hospitals or units as a 'black box'.

3 Social attitudes, substance use, policies/legislation, and provision of specialist services all change over time and between jurisdictions.

Further information

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Index

Note: Page numbers in **bold** refer to main entries. Those in *italic* refer to figures and / or tables. Alphabetical order is word-by-word.

Plates for Chapter 2.3.1

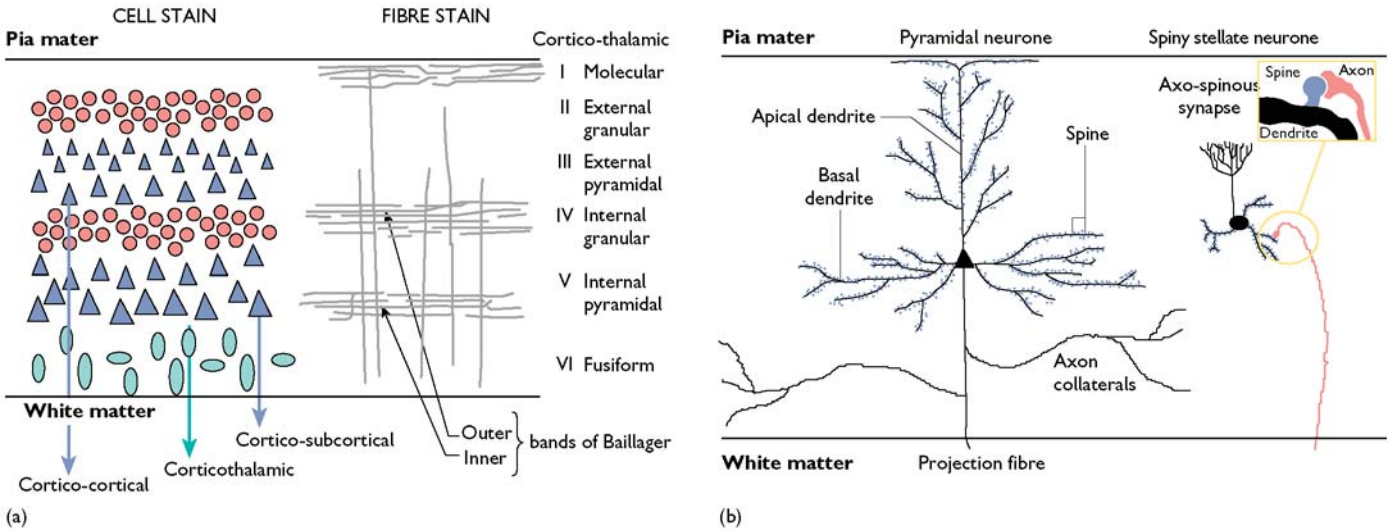

Plate 1 (a) Laminar structure of the cerebral neocortex; (b) excitatory amino acid using spiny neurones of the neocortex.

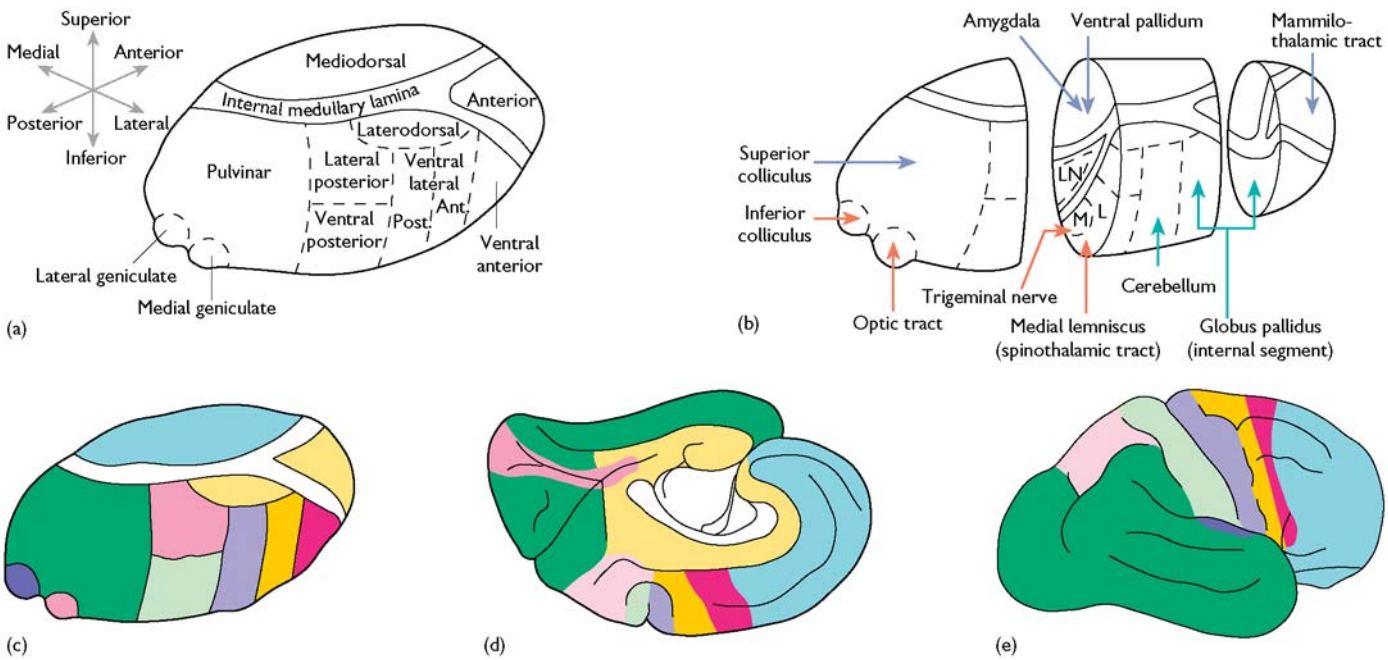

Plate 2 (a) The thalamus is shown as if removed from the brain at the top, with the individual major nuclei indicated. (b) Schematic diagram showing the isolated thalamus divided approximately at the middle of its anteroposterior extent; the arrows indicate known sources of major subcortical afferents to the individual named nuclei. (c) The diagram of the isolated thalamus shown in (a) with the individual main nuclei colour coded. (d), (e) Schematic diagrams of the cerebral surfaces (medial and lateral) showing the regions of cortex colour coded to correspond to the main thalamic nuclei with which they have major connections.

Plates for Chapter 2.3.6

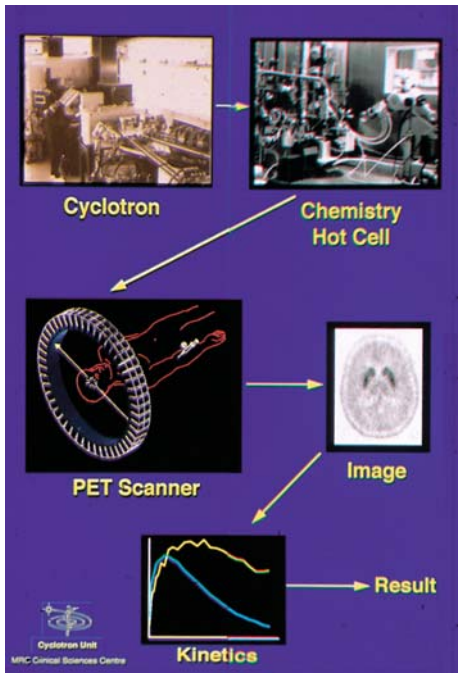

Plate 3 Steps in the production and use of PET radio-isotopes.

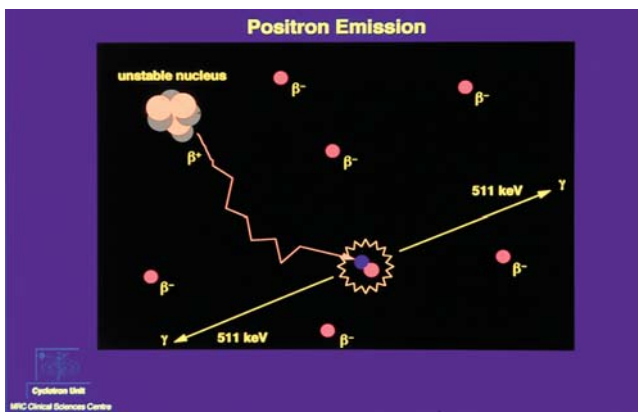

Plate 4 Principles of positron emission: β^- is an electron and β^+ is a positron. Two high-energy gamma rays (γ) are produced on annihilation of a positron by an electron

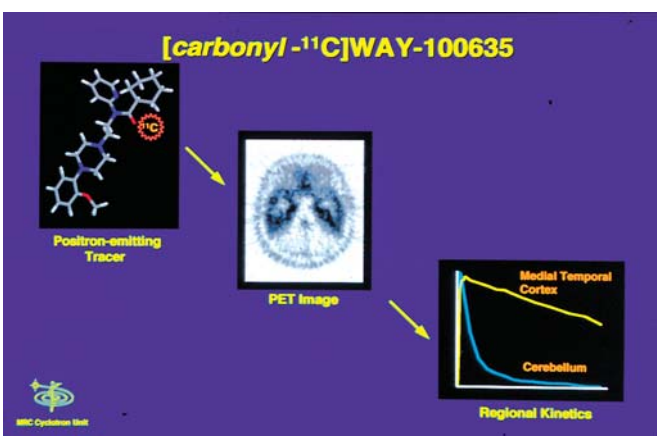

Plate 5 Use of a PET radiotracer to image 5-HT_{1A} receptors in the human brain.

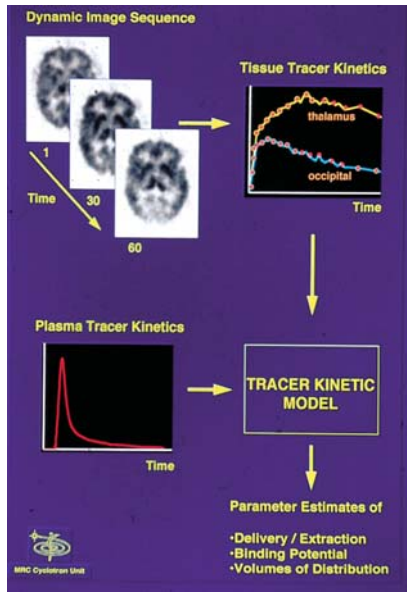

Plate 6 Steps in the data analysis of a PET radiotracer.

Plates for Chapter 2.3.7

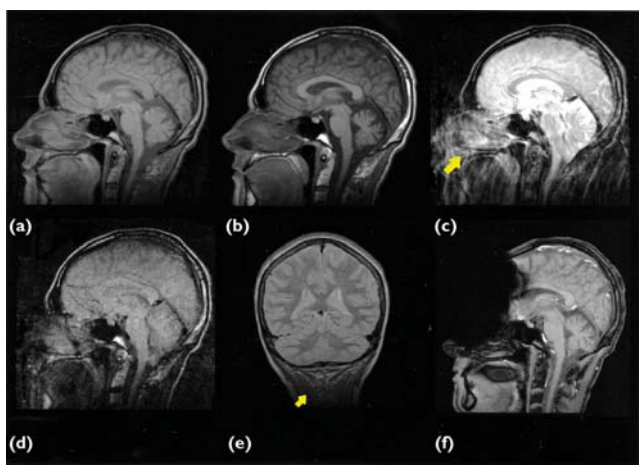

Plate 7 Spin echo MR images and artefacts. Top row, spin echo images: left, proton density image (TR = 2000 ms, TE = 20 ms); middle, T_1-weighted image (TR = 350 ms, TE = 20 ms); right, T_2-weighted image (TR = 2000 ms, TE = 90 ms). The arrow on the T_2-weighted image indicates blurring caused by movement (swallowing) during acquisition. Bottom row, examples of poor tissue contrast and susceptibility artefact: left, spin echo image showing poor contrast due to injudicious prescription of pulse sequence (TR = 350 ms, TE = 90 ms); middle, bulk susceptibility artefact; right, ferromagnetic susceptibility artefact caused by a metallic hairgrip.

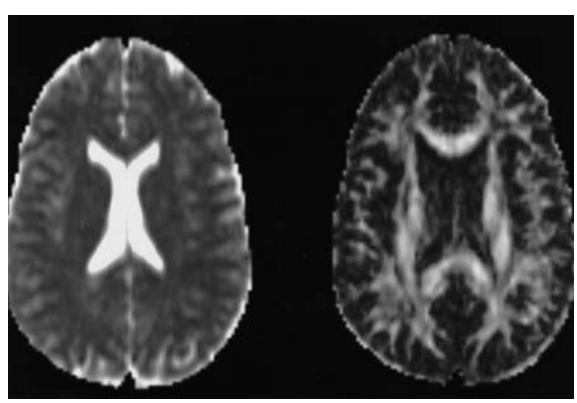

Plate 8 Diffusion-weighted MRI data can be used to generate maps of (left) the apparent diffusion coefficient (**ADC**) and (right) the anisotropy of diffusion. Diffusion of protons is most rapid and isotropic in cerebrospinal fluid, and least rapid and most anisotropic in white matter. White matter is clearly defined by relative hyperintensity in the anisotropy map.

Plates for Chapter 2.3.7 *(continued)*

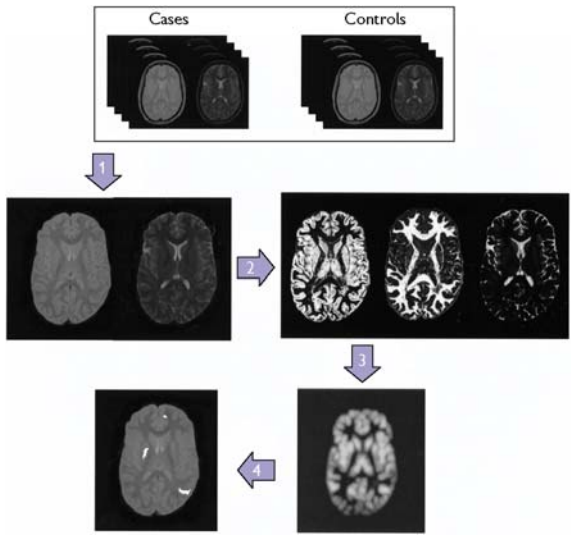

Plate 9 Steps in computerized image analysis. Dual-echo (fast spin echo) data are acquired from several cases and controls in a cross-sectionally designed study. Extracerebral tissue is removed (1) from each image before segmentation or tissue classification (2). Tissue-calssified images are registered with a template image in standard space (3) before hypothesis testing (4). Voxels or clusters, which demonstrate a significant difference in tissue class volume between groups, are colour coded.

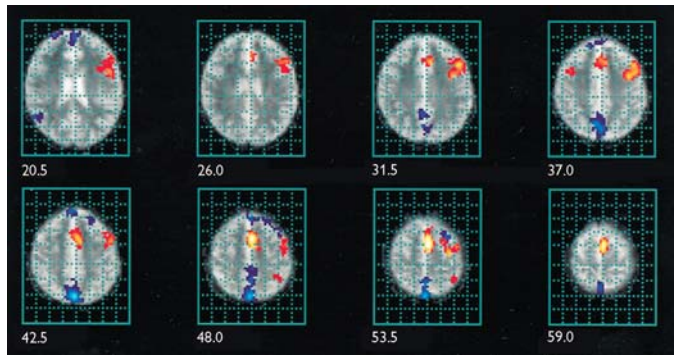

Plate 11 Activation map. Generically activated voxels are colour coded against a grey-scale background of gradient echoplanar imaging data. The grid respresents the standard Talairach-Tournoux Space;[12] z-coordinates for each slice are shown at the bottom left. Colour codes the timing and power of a periodic response to a covert verbal-fluency experiment. Blue voxels show increased magnetic responance signal during condition B (repeat a word covertly); light blue represents a greater power of response than dark blue. Red voxels show increased magnetic resonance signal during condition. A (generate a word beginning with a cue letter); yellow and orange represent a greater power of response than dark red. The voxel-wise probability of a false-positive error is $p = 0.0001$. The main areas activated during conditioin A are the dorsolateral prefrontal cortex, inferior frontal gyrus, and supplementary motor area; the main areas activated during condition B are the medial parietal cortex and posterior cingulate gyrus.

Plates for Chapter 2.3.8

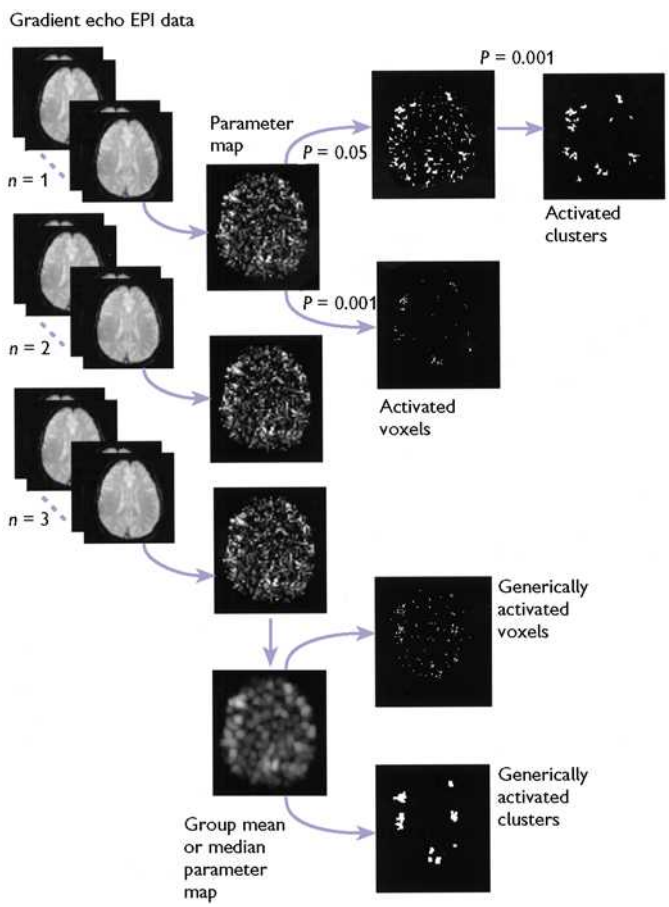

Plate 10 Steps in computerized data analysis. Gradient echoplanar imaging data have been acquired from three subjects under identical conditions. The time series at each voxel is analysed to estimate a measure or parameter of the experimental effect, which is represented as a parameter map. Significantly activated voxels or clusters can be identified in each individual image. Parameter maps can be averaged over individuals and generically activated voxels or clusters identified over the group of subjects. The power to detect activation is enhanced by cluster-level analysis and by combining data from several subjects.

Plate for Chapter 4.1.3

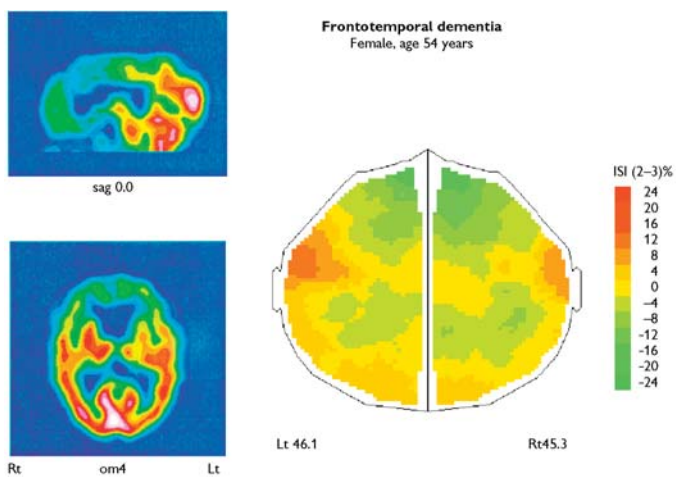

Plate 12 Regional cerebral blood flow (rCBF) measured using SPECT with exametazine (left) and the Xenon-133 inhalation method (right) in a 54-year-old female with clinical signs of FTD. The variation of regional cerebral blood flow is measured with xenon-133, above (red) or below (green) the average flow level, as indicated by the colour code. The patient showed the first signs of personality change, and stereotypy of speech and behaviour at the age of 48 years. EEG was normal, and CT and MRI showed slight frontal cortical atrophy. The regional cerebral blood flow measurement with xenon-133 showed a normal average flow level and marked bilateral, frontal flow decreases. The SPECT scan showed a severe perfusion deficit in the frontal and anterior cingulate cortex. (Courtesy of Department of Neurophysiology, University Hopital, Lund, Sweden.)

Plate for Chapter 4.2.1

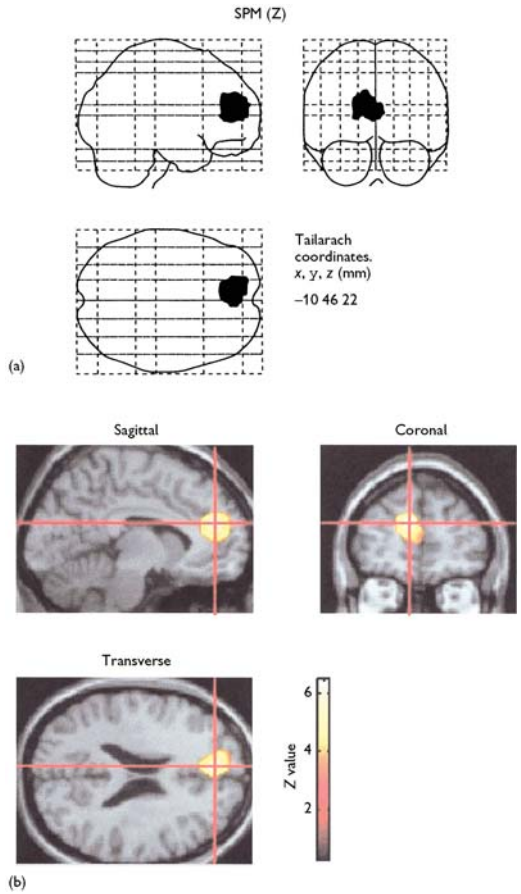

Plate 13 Area of activation during opiate craving: (a) [^{15}O]H_2O PET SPM image; (b) area of activation superimposed on magnetic resonance image.

Plate for Chapter 4.5.6

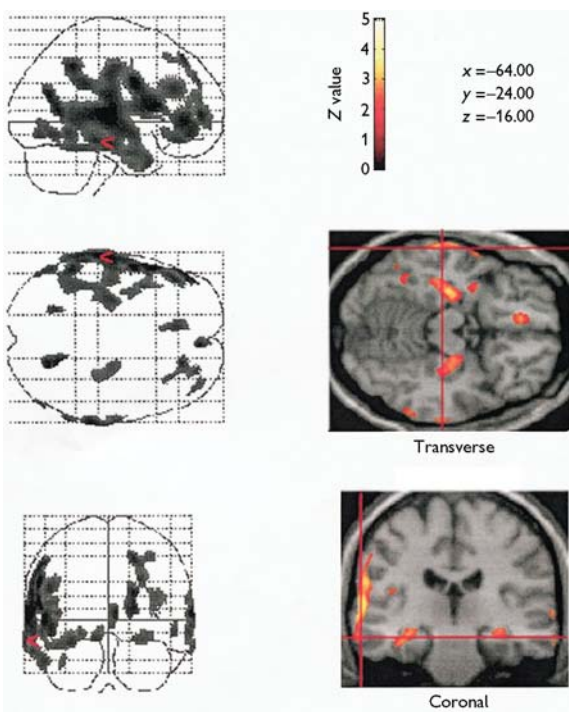

Plate 14 Statistical parametric maps ($p < 0.001$) for reductions in grey matter densities in subjects with chronic refractory depression compared with controls. Effects are controlled for age. (Reproduced with permission from PJ. Shah *et al.* (1998). Cortical grey matter reductions associated with treatment-resistant chronic unipolar depression: controlled MRI study. *British Journal of Psychiatry*, **72**, 527–32.)

Plates for Chapter 6.2.10.4

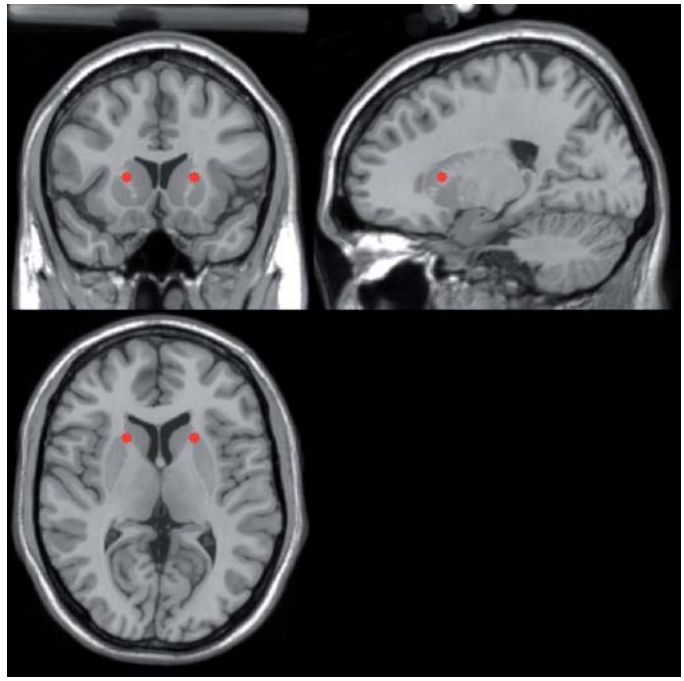

Plate 15 Typical locations of anterior capsulotomy lesions superimposed upon normalized T1 MRI scan. Lesions not to scale.

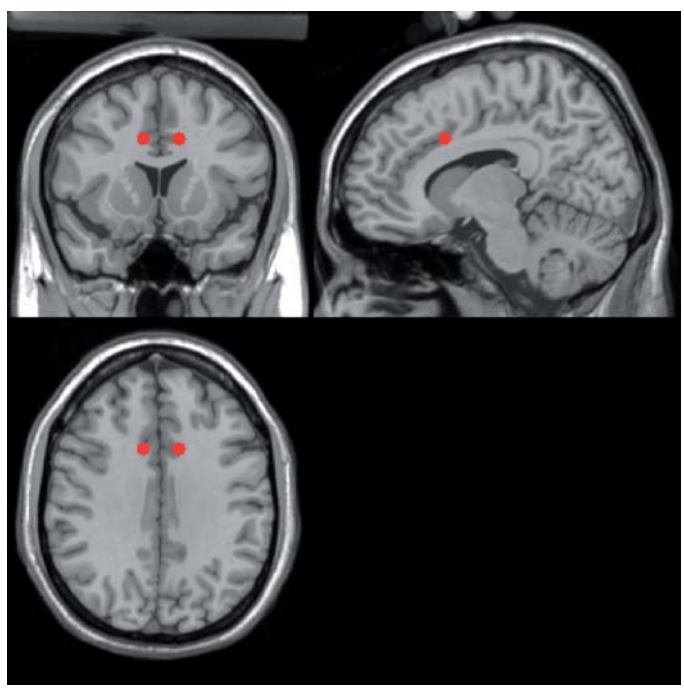

Plate 16 Typical locations of anterior cingulotomy lesions superimposed upon normalized T1 MRI scan. Lesions not to scale.