Edited by NISHA DOGRA, BRIAN LUNN and STEPHEN COOPER

PSYCHIATRY

2nd EDITION

by Ten Teachers

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Preface

Most of those students reading this textbook will not become psychiatrists. However, as doctors, all of you, whether in your foundation posts or later, will encounter patients with significant mental health problems and symptoms of psychiatric illnesses. To carry out your role as a doctor effectively, you will require the necessary knowledge and skills. This book has been written to try to address the needs of all students, whatever their career intentions.

The book is largely based around the core curriculum devised as part of the editors' work for the Royal College of Psychiatrists' Scoping Group on Undergraduate Education. The 'core curriculum' arose from a consensus around this work.

In each subject we have tried to be explicit about why the area covered is relevant to you as a medical student, and each chapter gives clinical examples to illustrate the points made. We have sought to avoid overloading you with details but have instead focused on essential information to help you meet the mental health needs of all patients.

In some chapters, for example 1, 3 and 4, you are asked to reflect on your own perspectives as we consider that many societal and professional attitudes towards those with mental illness need to be challenged. We hope you find that this book helps you to get as much as possible from your clinical placements and by the end feel confident that you could recognize and manage mental health problems when you qualify.

It is worth emphasizing that no clinical placement will be successful unless you practice your skills, and no book can substitute for spending time with patients. However, it is helpful to have a framework around which to organize your experience. While this textbook is focused on the needs of all medical students, we would be delighted if using it during your placements makes you consider becoming a psychiatrist. We would be happy to receive any feedback and sincerely hope the book works as it is intended to.

Nisha Dogra
Brian Lunn
Stephen Cooper

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List of abbreviations

AAMC	American Association of Medical Colleges
ABC approach	Antecedents, behaviour, and consequences approach
Aβ	Amyloid-β
ACE	Angiotensin-converting enzyme
ACE-III	Addenbrookes Cognitive Examination-III
AD	Alzheimer's disease
ADHD	Attention deficit hyperactivity disorder
AIDS	Acquired immune deficiency syndrome
ALT	Alanine aminotransferase
AMHP	Approved mental health professional
AMTS	Abbreviated mental test score
APP	Amyloid precursor protein
APOE	Apolipoprotein E4
ASD	Autism spectrum disorder
AUDIT	Alcohol use disorders identification test
BDZ	Benzodiazepine
BMI	Body mass index
BNF	British National Formulary
BSE	Bovine spongiform encephalopathy
CAMHS	Child and Adolescent Mental Health Services
CB	Challenging behaviour
CBT	Cognitive behavioural therapy
CG	Cytosine-guanine-guanine
Cl^-	Chloride ion
CNS	Central nervous system
CPK	Creatine phosphokinase
CPN	Community psychiatric nurse
CSF	Cerebrospinal fluid
CT	Computed tomography
CVD	Cardiovascular disease
DA	Dopamine
DAT	Dopamine transporter
DISC1	Disrupted in schizophrenia 1
DOLS	Deprivation of Liberty Safeguards
DPD	Dependent personality disorder
DSH	Deliberate self-harm
DSM	*Diagnostic and Statistical Manual of Mental Disorders*
DSM-IV	*Diagnostic and Statistical Manual of Mental Disorders*, 4th edition
DSM-5	*Diagnostic and Statistical Manual of Mental Disorders*, 5th edition
DVLA	Driver and Vehicle Licensing Agency
DZ	Dizygotic

ECG	Electrocardiogram
ECT	Electroconvulsive therapy
EDNOS	Eating disorder not otherwise specified
EEG	Electroencephalography
eGFR	Estimated glomerular filtration rate
EMDR	Eye movement desensitization and reprocessing
EPSE	Extrapyramidal side effects
EUPD	Emotionally unstable personality disorder
FBC	Full blood count
FMR1	Fragile X mental retardation 1
FSH	Follicle-stimulating hormone
GABA	Gamma-aminobutyric acid
GABRA	Gamma-aminobutyric acid type A receptor alpha2
GAD	Generalized anxiety disorder
GDS	Geriatric depression scale
GFR	Glomerular filtration rate
GHB	Gamma-hydroxybutyrate
GP	General practitioner
GPCOG	General Practitioner Assessment of Cognition
GPI	General paresis of the insane
γ-GT	γ-glutamyl transpeptidase
GWAS	Genome-wide association study
HIV	Human immunodeficiency virus
HPA	Hypothalamic–pituitary–adrenal
HSV	Herpes simplex virus
ICD	*International Classification of Diseases*
ICD-10	*International Classification of Diseases*, 10th revision
ICD-11	*International Classification of Diseases*, 11th revision
ID	Intellectual disability
IM	Intramuscular
IQ	Intelligence quotient
IV	Intravenous
LFT	Liver function test
Li	Lithium
LH	Luteinizing hormone
LSD	Lysergic acid diethylamide
MAO-A	Monoamine oxidase-A
MAOI	Monoamine oxidase inhibitor
MBCT	Mindful-based cognitive therapy
MCA	Mental Capacity Act
MHA	Mental Health Act
MHRA	Medicines and Healthcare Products Regulatory Agency
MI	Myocardial infarction
MMN	Mismatch negativity
MMSE	Mini-mental state examination
MoCA	Montreal Cognitive Assessment
MRI	Magnetic resonance imaging
MT	Melatonin
MVP	Mitral valve prolapse

MZ	Monozygotic
NA	Noradrenaline
NARI	Noradrenaline reuptake inhibitor
NCS	National Co-morbidity Survey
NFT	Neurofibrillary tangles
NHS	National Health Service
NIAAA	National Institute on Alcohol Abuse and Alcoholism
NICE	National Institute for Health and Care Excellence
NMD	Neurosurgery for mental disorder
NMDA	*N*-Methyl-D-aspartic acid
NMS	Neuroleptic malignant syndrome
NSAID	Non-steroidal anti-inflammatory drug
OCD	Obsessive–compulsive disorder
OPCS	Office of Population Censuses and Surveys
OSCEs	Objective-structured clinical examinations
PCP	Phencyclidine
PET	Positron emission tomography
PICU	Psychiatric intensive care units
PNFA	Progressive non-fluent aphasia
PWS	Prader–Willi syndrome
PRL	Prolactin
PRN	*Pro re nata* – as needed
PTSD	Post-traumatic stress disorder
QTc	Corrected QT Interval
RBC	Red blood cell
REM	Rapid eye movement
RIMA	Reversible inhibitor of MAO-A
RT	Rapid tranquillization
SAD	Seasonal affective disorder
SCOFF	Sick, control, one stone, fat, food
SIGN	Scottish Intercollegiate Guidelines Network
SIRT1	Sirtuin1
SMAST-G	Short Michigan Alcohol Screening Test-Geriatric Version
SNRIs	Serotonin and noradrenalin reuptake inhibitors
SP	Senile plaques
SPC	Summary of product characteristics
SPET	Single photon emission computed tomography
SSRI	Selective serotonin reuptake inhibitor
SUDEP	Sudden unexpected death in epilepsy
TCA	Tricyclic antidepressant
TD	Tardive dyskinesia
TSC	Tuberous sclerosis
THS	Thyroid stimulating hormone
VLOS	Very late-onset schizophrenia
WHO	World Health Organization
5-HT	5-Hydroxytryptamine
5-HIAA	5-Hydroxyindoleacetic acid
6-CIT	Six-item Cognitive Impairment Test

Digital resources

PDF resources

Visit www.crcpress.com/9781498750022 for the following eResources that accompany this textbook:

- Glossary of psychopharmacology
- Patient assessment handbook

Video resources

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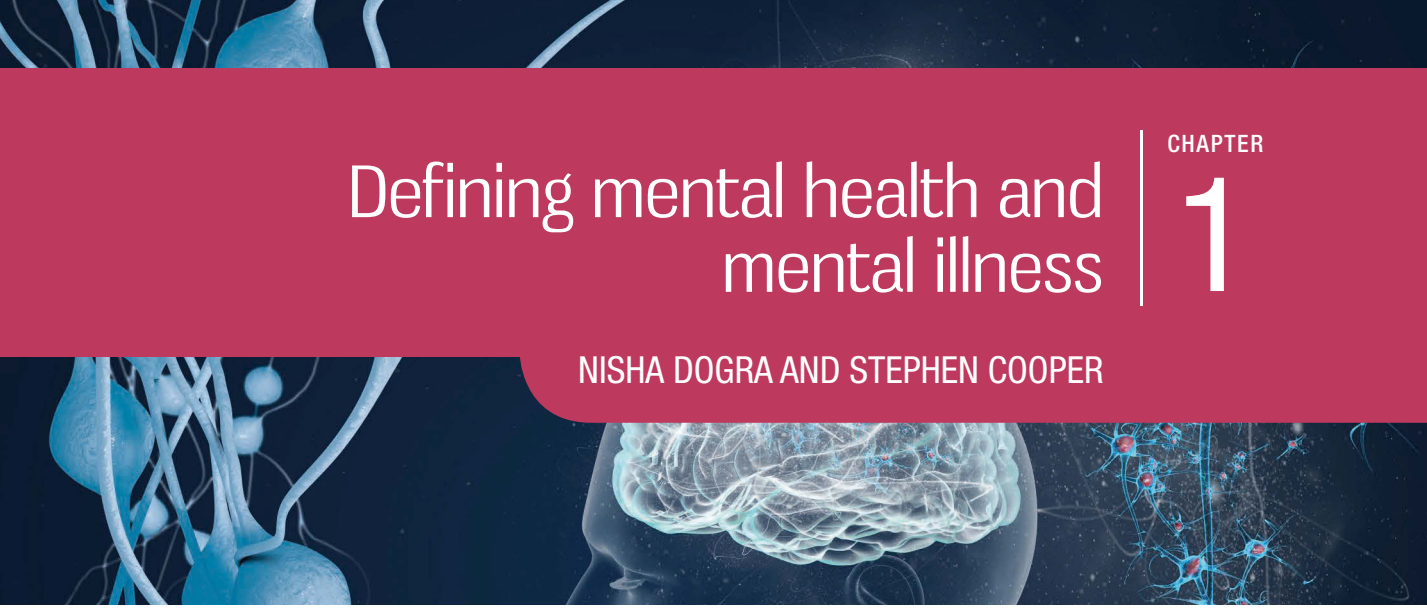

CHAPTER 1

Defining mental health and mental illness

NISHA DOGRA AND STEPHEN COOPER

KEY CHAPTER FEATURES

- Discussion of the terminology around mental health, mental health problems and mental illness
- Outline of the scale of individual suffering from mental health problems and a public health dimension of the scale of the problems
- Define stigma, how it is perpetuated and its consequences on individuals and practice
- The evidence regarding which interventions may reduce stigma
- The steps you may need to take to reflect on your views and their impact on your practice

Introduction

In this chapter, we define mental health, mental health problems and mental illness. This is important because, although it sounds fairly straightforward, our discussion will demonstrate the difficulties that abound with the terminology. The scale of the problem and access to services at a public health level are outlined. We then discuss stigma generally, explore the reasons for it and possible sequelae. We also review the interventions to reduce stigma before asking you to reflect on your own perspective and their potential impact on your future practice.

Defining mental health, mental illness and mental health problems

It is important to state at the outset that there is no widely agreed consensus on the meaning of these terms and their use. Many outside the health arena challenge the terms, and mental illness as a concept is widely challenged by the anti-psychiatry movement (which includes doctors). However, the reality remains that if an individual experiences difficulties which impact on their emotional and inner worlds, their functioning can be affected. Mental health and

EXERCISE 1.1

On your own or with some peers answer the following questions? (You will get more from the exercise if you answer as honestly as you can.)

What is your own understanding of mental health, mental illness and mental health problems?

What sorts of problems do people experience that could be described as mental health problems or mental illness?

How can you tell if someone is experiencing mental health problems or mental illness?

How often do you use words that reflect on patients with mental health in a less than complimentary way?

How do you think you have formed your views on mental illness and what part might your cultural background have played in forming these views?

mental illness can be viewed as two separate, yet related, issues.

Mental health

The World Health Organization (WHO) definition of health is: 'A state of complete physical, mental and social well-being, and not merely the absence of disease' (www.who.int/topics/mental_health/en/). This is supported by the Royal College of Psychiatrists, who have argued that there is no health without mental health. The WHO adds:

> *Mental health is not just the absence of mental disorder. It is defined as a state of well-being in which every individual realizes his or her own potential, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to her or his community.*

Another way of looking at this is that mental health includes how people look at themselves, their lives and the other people in their lives; how they feel about these different components, evaluate their challenges and problems; and how they explore choices. This includes handling stress, relating to other people and making decisions. However, even a cursory glance at the definition raises important questions as the concept is clearly rooted in societal norms and expectations. The way that the normal stresses of life are defined will vary from society to society and within subgroups. The contribution to the community is also societal and culturally based. Perhaps a useful way of viewing the definition in practice is: 'that someone is considered as having mental health when they manage day-to-day living without too much difficulty in a way that satisfies them and fulfils familial and societal expectations of them without causing them undue stress'. Immediately this alerts you to consider the plight of those who do not meet familial or societal expectations as they conflict with individual perspectives and the significant impact this can have on mental health (for example, consider being gay in countries in which homosexuality is illegal, or forced marriages). Culture and its influence on how mental health is understood is discussed later in this chapter in the section 'Mental health: One of many factors'.

Definitions of mental health relating specifically to children have been provided by several bodies and emphasize the expectations of a healthy child. So, a mentally healthy child is one who can, for example:

- Develop emotionally, creatively, intellectually and spiritually
- Initiate, develop and sustain mutually satisfying personal relationships
- Face problems, resolve them and learn from them

This could easily apply to adults and in some ways is developmentally rather than culturally contextual, as these functions apply in whichever society the young person is living.

Mental health problems

'Mental health problems' is a term that encompasses a range of experiences and situations. Mental health might usefully be viewed as a continuum of experience, from mental well-being through to a severe and enduring mental illness. Mental health problems cover a wide range of problems which affect someone's ability to get on with their daily life. Mental health problems can affect anyone, of any age and background, as well as have an impact on the people around them such as family, friends and carers. Mental health problems result from a complex interaction of biological, social and psychological factors. Major life events such as bereavement, relationship break-up or serious illness can impact significantly

on how we feel about ourselves and subsequently on our mental state and health. A minority of people may experience mental health problems to such a degree that they may be diagnosed as having a mental illness. Common mental health problems include anxiety (including phobias), obsessive compulsive disorders, adjustment disorders and milder mood problems.

Mental illness

A mental illness is an illness that causes disturbances in thinking, perception and behaviour beyond those that might be experienced even in an acutely distressed state. They can be severe, seriously interfering with a person's life, significantly impairing a person's ability to cope with life's ordinary demands and routines and even causing a person to become disabled. The majority of people will not experience mental illness, but will undoubtedly experience mental health problems at different times in their lives.

Another common term is mental disorder, and this is often used in the sense that a person who is mentally ill is suffering from a mental disorder – the use is usually in the context of legislation. In practice, most clinicians tend to use mental health problems for less serious disorders and mental illness for more severe disorders. A complicating factor is that subjective components are also relevant. Clinicians may feel that the anxiety symptoms their patients have are fairly mild but for the patients the impact on their life may be significant. Some patients who are seriously mentally ill (for example someone who is manic or acutely psychotic) cannot understand why others think they are ill because from their perspective all is well.

In this book, we use the term mental health problems as that is a widely used terminology, although specific disorders such as schizophrenia, depression and the like are defined as mental illness using the *International Classification of Diseases* (*ICD*) (WHO).

The classification of mental illness

The *ICD* is the international standard diagnostic classification for all general epidemiological and many health management purposes, research and clinical use. It is used to classify diseases and other health problems recorded on many types of health and vital records such as death certificates. However, the way that these diagnostic categories are used in practice varies across the world. The major categories for mental health and behavioural disorders are shown in Box 1.1. The *ICD* is revised periodically and is currently in its tenth edition with the 11th edition being planned.

The *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) is published by the American Psychiatric Association and provides diagnostic criteria for mental disorders. It is used in the United States and in varying degrees elsewhere. There is some consistency between the two classification systems, especially for mental health. However, the *DSM* tends to use broader categories and is considered by some too inclusive in some of the disorders it lists. The multi-axial format used by the *DSM* can be helpful as shown in Box 1.2.

Box 1.1 Multi-axial classification often used in child mental health services (ICD-10)

Axis 1 – Mental health diagnosis
Axis 2 – Developmental
Axis 3 – Intellectual
Axis 4 – Organic/physical
Axis 5 – Psychosocial

Mental and behavioural disorders (F00–F99)

F00–09	Organic, including symptomatic, mental disorders
F10–19	Mental and behavioural disorders due to psychoactive substance use
F20–29	Schizophrenia, schizotypal and delusional disorders
F30–39	Mood (affective) disorders
F40–48	Neurotic, stress-related and somatoform disorders
F50–59	Behavioural syndromes associated with physiological disturbances and physical factors
F60–69	Disorders of adult personality and behaviour
F70–79	Mental retardation
F80–89	Disorders of psychological development
F90–99	Behavioural and emotional disorders with onset usually occurring in childhood and adolescence

Box 1.2 Multi-axial system used in DSM

The DSM-IV organizes each psychiatric diagnosis into five levels (axes) relating to different aspects of disorder or disability.

- Axis I: clinical disorders, including major mental disorders, as well as developmental and learning disorders
- Axis II: underlying pervasive or personality conditions, as well as mental retardation
- Axis III: acute medical conditions and physical disorders
- Axis IV: psychosocial and environmental factors contributing to the disorder
- Axis V: Global Assessment of Functioning or Children's Global Assessment Scale for children and teens under the age of 18

Common Axis I disorders include depression, anxiety disorders, bipolar disorder, ADHD, phobias and schizophrenia.
Common Axis II disorders include personality disorders and mental retardation.

Both classification systems have their limitations but are useful in providing a common language for research and practice and both use the categorical approach as described in the next section.

Entity or dimension?

Kendall (1988) present the relative merits of using categories and dimensions with respect to mental illness. Typically, medicine has used categories, given its roots in the biological sciences. Categorization allows for easier definitions. It enables recognition of someone's symptoms as conforming to a specific clinical concept. However, a dimensional approach allows for greater flexibility. They conclude that where psychotic illness is concerned then a categorical approach may be preferable, whereas in other conditions, the situation is more likely to be changeable, and perhaps benefit from a dimensional perspective. The categorical approach essentially allows the clinician to make a diagnosis based on the presence or absence of symptoms. There are two possibilities, either the patient has the disorder or not.

The difficulties with the categorical approach are that disorders may not present with the whole range of symptoms needed for a diagnosis to be made. However, the presence of those symptoms may still be sufficient to cause significant impairment. The dimensional approach allows for context and specific factors to be accounted for, such as developmental stage and gender.

With respect to emotions and behaviours it can be useful to ask 'when is a problem a problem', as for example, people may have the same level of anxiety but be troubled by it to different extents. Pain is a common symptom in general medicine and may be a helpful analogy. So, we all experience some degrees of anxiety but it is only likely to be viewed as a problem that needs help when it occurs frequently and/or is so severe that it interferes with everyday functioning. The symptom may also be identified as a problem when it begins to impact on those around the patient.

One way of distinguishing between distress associated with adverse life events and more severe disorders, which involve physiological symptoms and underlying biological changes, is to distinguish between mental health problems and mental illness, using a multidimensional model. This has an additional advantage in enabling normal 'distress' (e.g. grief following bereavement) to be recognized as part of the 'human condition', rather than being medicalized and possibly classed as 'depression'. It is suggested that a variety of normal human experiences have become medicalized through an ever increasing range of psychological disorders with virtually every type of behaviour eligible for a medical label (e.g. social phobia, overeating disorder, dependent personality disorder).

Mental health: One of many factors

It is also important to recognize that neither physical nor mental health exists separately; mental, physical and social functioning are interdependent. Furthermore, all health issues need to be considered within a cultural and developmental context. The quality of a person's mental health is influenced by idiosyncratic individual factors and experiences, their family relationships and circumstances and the wider community in which they live. Additionally, each culture influences people's understanding of,

and attitudes towards, mental health issues. However, a culture-specific approach to understanding and improving mental health can be unhelpful if it assumes homogeneity within cultures and ignores individual differences. Culture is only one, albeit an important, factor that influences individuals' beliefs and actions. It can be argued that the aforementioned are rooted in Western perspectives. However, they provide a useful starting point from which to discuss mental health issues.

Incongruence among personal values and familial and wider societal expectations can be a significant stressor especially for young people. People's cultural backgrounds can affect mental health in the following ways:

- The way they think about mental health and mental health problems
- The way they make sense of certain symptoms and behaviours
- The services they choose to accept
- The treatment and management strategies they find acceptable
- The way in which those who have mental health problems are perceived

Some of these factors are mitigated by others, such as the level of education and personal experience.

Interaction between mental and physical health

At this point it is perhaps worth clarifying the common situations in which mental health and physical health issues coincide more obviously.

1. **Psychosomatic disorders:** These are physical illnesses which may be aggravated by psychological factors or in which psychological factors (e.g. stress) may play a part in the aetiology. Examples are ulcerative colitis, asthma, essential hypertension or increased vulnerability to cardiovascular disease in the 12 months after the death of a spouse. Psychological factors are generally not the main cause of the physical pathology and the physical pathology is not being presented unconsciously for the sake of some advantage. These disorders are not discussed further as you will learn about them in other parts of your medical course.
2. **Somatoform disorders (see Chapter 7):** These are disorders in which physical symptoms are presented but for which no organic pathology can be found and in which some form of psychological mechanism is resulting in the (usually) unconscious presentation of physical symptoms. These are thus a direct contrast to psychosomatic disorders.
3. **Psychological symptoms secondary to physical illness:** There are many situations where this may occur and common ones will be highlighted in the chapters on specific mental disorders. Simple examples are mood change secondary to frontal lobe dysfunction in multiple sclerosis and anxiety secondary to hyperthyroidism.
4. **Physical health problems secondary to a mental disorder:** As a group, people with mental health problems experience poorer physical health and life expectancy than the general population (Wahlbeck et al., 2011). The reasons for this are generally multifactorial and some are commented upon in specific chapters. For example, people with schizophrenia have a high prevalence of type 2 diabetes and excess mortality from cardiovascular disease. This is partly due to excessive weight gain affecting many patients, which is partly secondary to the adverse effects of some antipsychotic medications, but lack of exercise, poor diet and often less attention from the health service also play a part. Patients who have suffered from severe eating disorders, even when recovered, may have long-term sequelae such as osteoporosis or premature mortality.
5. **Medication-induced adverse effects:** Most psychotropic medications can have physical adverse effects (e.g. Parkinsonian type tremor with antipsychotics, nausea with selective serotonin reuptake inhibitor [SSRI] antidepressants) and these are described in Chapter 17. Psychological adverse effects can also occur with some medications used for physical illnesses (e.g. depression secondary to corticosteroid treatment or calcium channel blockers).

Sexual disorders and dysfunctions

Sexual behaviour is an important aspect of life. Disturbances can occur that impair normal *sexual functioning* and in an individual's *sexual preferences*. These are largely classified in ICD-10 and DSM-IV because they frequently result in psychological distress. These disturbances sometimes have a psychological problem at their root but may also represent extremes of normal human behaviour or aspects of wider personality difficulties. Thus, in terms of defining the bounds of mental illness they are an intriguing set of issues. It is also worth noting that societal perspectives also influence how some of these aspects are viewed; for example until 1967 homosexuality was in the United Kingdom considered illegal and this remains the case in many countries. This in turn may influence what patients are prepared to share with you as their doctor.

Disturbances of sexual functioning are common and recognition of disturbance in sexual function is relatively straightforward if an appropriate history of sexual behaviour is taken (see Chapter 3). These may have an organic basis (e.g. erectile difficulties in patients with diabetes; dyspareunia secondary to gynaecological problems) and can occur as symptoms of mental illness (e.g. loss of sexual interest in depression). In such situations treatment of the primary problem comes first with appropriate attention to related psychological distress. In some cases (e.g. a newly married couple) appropriate education and advice about normal sexual anatomy and function may be appropriate. You should learn about this in your gynaecological attachment. Where the problem has a primary psychological origin there are a number of recognized psychological approaches. An understanding of these is more appropriate to your post-graduate training and is not discussed further here.

Disturbances of sexual preferences are described as paraphilias and consist of gender identity disorders (e.g. transvestism and transsexualism) and disorders where sexual arousal is unusual or involves use of inappropriate objects or people (e.g. fetishism, paedophilia, voyeurism, sexual masochism). Behaviours such as paedophilia and voyeurism clearly result in behaviours that are entirely inappropriate and illegal. Some, such as sexual masochism, may be regarded as acceptable in some sections of society. Others, such as transsexualism, can represent a genuine need for gender reassignment, with hormone treatment and surgery being offered where appropriate. The detailed assessment and management of these problems is outside the scope of this book. As a student and newly qualified doctor your responsibility is to be able to take an appropriate sexual history and recognize these problems. Their management will almost always be by specialist services. However, thinking about these issues should illustrate to you the complexities in defining the boundaries of mental health problems and illness.

The scale of the problem

The prevalence of specific disorders will be covered in the chapters relating to those disorders. Here we outline the scale of the problem in terms of broad figures using information provided by the WHO. The purpose is not to overwhelm you with figures but to impress upon you that the scale of mental illnesses and their impact on individuals and society is not insignificant. You should also be aware that psychiatrists treat only a small proportion of those who have mental health problems, with many more being treated in primary care, but the vast majority receiving no treatment at all.

The WHO has consistently argued that the economic and personal costs of mental illness are huge. For example, estimates made by the WHO in 2002 showed that 154 million people globally suffer from depression and 25 million people from schizophrenia; 91 million people are affected by alcohol-use disorders and 15 million by drug-use disorders (Figure 1.1). One in four patients visiting a health service has at least one mental, neurological or behavioural disorder, but most of these disorders are neither diagnosed nor treated. It is also important to note the high prevalence rates of mental illness in those suffering from chronic physical conditions such as cancer, cardiovascular diseases, diabetes and HIV/AIDS. The mental illness can lead to non-compliance with prescribed medical regimens and poorer prognosis. Stress, depression and anxiety are common reasons for absenteeism from work but may often not be addressed as illness and may be hidden to prevent stigmatization.

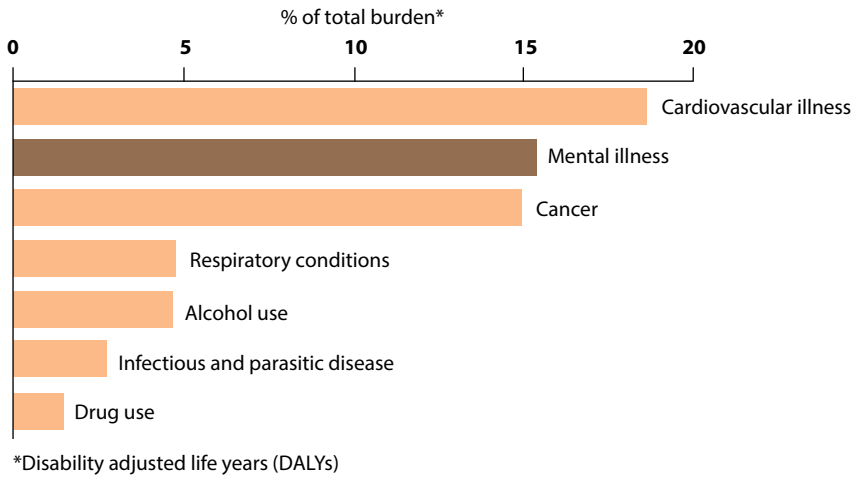

Figure 1.1 The burden of disease: established market economies (1990). (From Murray CL and Lopez AD (eds), *The Global Burden of Disease*, Harvard University Press, 1996.)

An often cited figure by the WHO is that one in four people in the world will be affected by mental or neurological disorders at some point in their lives. Only a minority will go on to have enduring and disabling illness but it is well established that mental and behavioural disorders have a large impact on individuals, families and communities. Individuals suffer through the symptoms they experience because they may be unable to work when they are unwell and often suffer from discrimination even after they have recovered. There is an associated 'loss of productivity' with economic impact on individuals and society. A number of studies have reported on the quality of life of individuals with mental disorders, concluding that the negative impact is not only substantial but sustained. It has been shown that quality of life continues to be poor even after recovery from mental disorders as a result of social factors that include continued stigma and discrimination.

Families commonly bear the burden of mental illness as they often provide support and care to family members who are mentally ill and also manage the negative impact of stigma and discrimination. The nature of mental illness often means that family relationships are affected in addition to the stress caused by disturbed behaviour and disruption to normal family life. In many parts of the world the cost of treatment is borne by families and not the state. Costs resulting from mental illness can be viewed as direct (that is the costs related to providing care and treatment for the disorder) and indirect (costs related to loss of productivity in work, school and home).

Anti-psychiatry

This term was coined in the 1960s in response to a movement led by Laing and Szasz, who essentially questioned the validity of psychiatry and the use of diagnoses that they felt were subjective. Their perspective was that medical concepts were being inappropriately applied to normal human behaviour. There was also considerable opposition to some of the treatments applied (e.g. antipsychotic medications and electroconvulsive therapy [ECT]). However, whatever one's perspectives about psychiatry, the suffering that many people experience cannot be dismissed. A major example of a criticism of psychiatry by the anti-psychiatry movement is that earlier versions of the classification systems included homosexuality as a disorder. This is no longer the case in any classification system.

Yet, psychiatry, and indeed medicine, are very much products of the various societal relationships, so it seems rather strange to single out some disorders (e.g. schizophrenia) over others and deny their existence. Just because we do not fully understand the aetiology of a disorder does not necessarily mean that the disorder does not exist. At least in the United Kingdom, the so-called 'anti-psychiatry movement' now regards itself more as a 'critical psychiatry movement'.

Myths about mental illness

Myths about mental illness abound across the world in all societies. The myths often seek to not only explain the cause of the behaviours exhibited as part of mental illness but also demonstrate the ignorance there is about mental illness. While myths persist, individuals with mental illness often delay seeking treatment or families fail to access appropriate treatment. Box 1.3 highlights some common myths and Box 1.4 gives the facts about the myth. Before moving to Box 1.4, try the exercise in Box 1.3 and see how many myths form your knowledge base about mental health and illness. It is the fear of the unknown that causes such myths about mental illness. The endurance of myths leads to stigma. The Royal College of Psychiatrists' website provides further information on many of these issues (www.rcpsych.ac.uk).

Stigma and mental illness

In this section the aim is to explore the concepts of, and the relationship between, stigma and mental illness. One possible reason for both conceptual

Box 1.3 Common myths about mental illness

As you read through these myths, think about which statements you agree with and what basis you have made your decision on.

- Young people and children do not suffer from mental health problems.
- A person who has had a mental illness will not recover.
- Mentally ill people are violent and dangerous.
- Mental illness affects others and cannot affect me.
- Mental disorders are caused by a personal weakness in character.
- Mental illness is a single, rare disorder.
- Psychiatric disorders are not true medical illnesses like heart disease and diabetes.
- People who have a mental illness are just 'crazy'.
- Schizophrenia means split personality, and there is no way to control it.

Box 1.4 Common myths about mental illness and the facts

Myth: Young people and children do not suffer from mental health problems.

Fact: It is estimated that between 10% and 25% of young people under the age of 18 suffer from mental health problems impacting on their ability to function at home, in school or in their community (see Chapter 14).

Myth: A person who has had a mental illness can never recover.

Fact: People with mental illnesses do recover and resume normal activities. Recovery depends on appropriate treatment and psychosocial factors and many people function well between episodes of illness.

Myth: Mentally ill people are violent and dangerous.

Fact: The vast majority of people with mental illnesses are not violent or dangerous but are often vulnerable. On average about 55 people a year are killed by someone with a psychiatric illness at the time of the homicide.

Myth: Mental illness affects others and cannot affect me.

Fact: Mental illnesses are surprisingly common and do not discriminate – they can affect anyone. One in four people will experience a mental illness at some point.

Myth: Mental disorders are caused by a personal weakness in character.

Fact: Mental disorders are caused by biological, psychological and social factors.

Myth: Mental illness is a single disorder.

Fact: Mental illness is not a single disease but a broad classification covering many disorders, as shown in this chapter.

Myth: Psychiatric disorders are not true medical illnesses like heart disease and diabetes. People who have a mental illness are just 'crazy'.

Fact: Brain disorders, like heart disease and diabetes, are legitimate medical illnesses. Research shows there are biological factors that can in combination with other factors cause psychiatric disorders, and they can be treated effectively (see Chapter 2).

Myth: Schizophrenia means split personality, and there is no way to control it.

Fact: Schizophrenia is often confused with multiple personality disorder. Actually, schizophrenia is a brain disorder that causes disordered thinking and perceptual abnormalities (see Chapter 9).

confusion regarding mental illness and reluctance to seek help is that the stigmatization of mental illness continues to be a worldwide phenomenon. The WHO considers that mental health provision was severely under-resourced in many countries because of stigma, apathy and neglect. Change may be happening but it is slow.

Definition of the concept of stigma

The word 'stigma', used to convey negative views about those with mental illness, originates from the Greek tradition of branding slaves with marks (stigmata) to identify them. Social stigma is a 'mark of infamy or disgrace; sign of moral blemish; stain or reproach caused by dishonourable conduct; reproachful characterization' (Webster's Dictionary).

Stigmatization is a social construct, and through this process those with mental illness are identified as being somehow different and having less worth. Much of the work about stigma has until quite recently been survey-based and focused largely on schizophrenia. Stigma can be seen as an overarching term that contains three components that interlink: problems of knowledge (ignorance), problems of attitudes (prejudice) and problems of behaviour (discrimination).

The extent and impact of stigmatization of adult mental illness

It is difficult to be clear about why we as a society continue to have such negative views about those with mental illness. It may be a way of creating a sense that those with mental illness are different from us and thereby reducing our own fears of becoming like 'them'. It is therefore perhaps not surprising that negative attitudes towards mental illness are largely culturally non-specific and commonplace across the world. Stigmatizing processes can affect multiple domains of people's lives, having a dramatic bearing on the distribution of life chances in such areas as earnings, housing, criminal involvement, health and life itself.

Healthcare staff attitudes generally tend to be similar to those of the lay public. Medical students have been shown to be critical of those whom they believe play a part in the development of their problems, for example self-harm, eating disorders and substance misuse. Patients are often very critical of general practitioner attitudes. However, they also complain about negativity from mental health professionals, especially those patients with personality disorders and substance misuse problems.

It is worth adding that not only are there negative views about those who have mental health problems but also about staff who work with them.

EXERCISE 1.2

Reflect on some of the stereotypes and attitudes towards mental illness and psychiatrists you have come across in your medical career.

Children, mental illness and stigma

From the sparse literature available on stigma in relation to children and mental illness, it appears that adolescents' attitudes towards mental illness tend to be negative and stigmatizing. Our work in Nigeria with young people shows that such attitudes transcend culture.

EXERCISE 1.3

Think about the last three times you have seen mental illness depicted on TV or in the newspaper. How did the media portray mental illness?

Did the articles:

- Stereotype people with mental illness (for example assuming they are all violent)?
- Minimize the difficulties faced by people with mental illness and/or the illness itself?
- Patronize people with mental illness?
- Assume that people with mental illness are somehow different from 'normal people'?
- Perpetuate other myths?

Media, mental illness and stigma

Popular images of mental illness are both longstanding and stable. Two large-scale literature reviews have suggested that the media can be regarded as an important influence on community attitudes towards mental illness. It is considered that there is a complex and circular relationship between mass media representation of mental illness and public understanding. Negative media images promote negative attitudes and the resulting media coverage feeding off an already negative public perception. It is also thought that negative images will have a greater effect on public attitudes than positive portrayals.

Thornicroft explored newspaper, television and film portrayal of mental illness and found it to be largely negative, although there were occasional exceptions. All media leaned heavily towards depicting those with mental illness as being dangerous. There was a tendency to use mental illness generically as opposed to referring to specific diagnoses. Disappointingly, children's television programmes consistently linked violence to mental illness and used derogatory terms. This may suggest to young children that these are acceptable ways in which to refer to those who have mental health problems. There is an argument that media representations matter because they play an active part in shaping and sustaining what mental illness means as suggested previously. Another relevant factor is that by its very nature, the media is highly visible and hard to ignore. Further, the tabloids' use of derogatory language may legitimize its use in our everyday language.

The media appear to give greater priority to their own needs to entertain and cause headlines. In the process, they may be drawn to using the shortcuts that stereotypes provide without challenging the damage they might be causing.

Interventions to reduce stigma

There is generally limited evidence about effective interventions to reduce stigma. Large-scale interventions, such as high-profile campaigns, are often difficult to evaluate. In the United Kingdom there have been several such campaigns (e.g. The Royal College of Psychiatrists' campaign, Every Family in the Land). There is little evidence to indicate that these have successfully changed public or personal attitudes. Although much of the work to date has focused on adults, there are increasing efforts to address the issue among younger populations, with some evidence that knowledge and attitudes can be improved.

There is as yet little evidence that anti-stigma work takes place in many medical schools and, where it does, how well it works to reduce the negative views that medical students have about mental illness. We discuss this more in Chapter 4.

It has been argued that the best way forward to tackle stigma may be to focus on discrimination rather than merely knowledge. In a way this also makes sense from the medical student educational perspective. *Good Medical Practice*, published by the GMC, places on doctors a responsibility to provide equitable care irrespective of various patient characteristics, including any diagnosis they may have. So irrespective of your own views you need to ensure you provide good quality care to all patients including those that have mental health problems.

Summary

There is still considerable debate around the terminology used in psychiatry. We suggest it is important to understand the principles of when someone is 'mentally healthy' and when they may have mental health problems that may be severe enough to be defined as a disorder. It is clinically more useful to take a multidimensional approach rather than the traditional categorical approach used in medicine. The impact of mental illness in the broadest sense for individuals, their families and society is significant and cannot be ignored. Yet despite the fact that we are all vulnerable to developing mental health problems, the stigmatization about mental illness pervades all cultures and societies and continues to be a challenge. Efforts are being made to address some of the stigmatization but progress is slow. Perhaps, you might want to conclude this chapter by returning to the exercise at the beginning of the chapter and asking, are you ready to challenge yourself?

Further reading

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CHAPTER 2

Personality, predisposing and perpetuating factors in mental illness

NISHA DOGRA AND STEPHEN COOPER

KEY CHAPTER FEATURES

- Outline of key aetiological factors: biological, e.g. genetic, organic, trauma, physical health problems; psychological, e.g. personality traits, developmental stage, attachment and ability to form and maintain relationships; social, e.g. environment, culture, family and wider sociopolitical factors
- Disorders of personality

Introduction

In this chapter, we outline the key relevant aetiological factors that play a part in the development of mental health problems and mental illness. Traditionally doctors like to consider aetiological factors for individual disorders but this is more difficult in mental illnesses for a variety of reasons. As discussed in Chapter 1 in the section 'Entity or dimension', there are difficulties in creating clearly defined syndromes to investigate as we have to define these on the basis of symptoms and behaviours, which often cross over different diagnoses. The factors we wish to 'measure' can be problematic, for example, brain biochemistry is not as readily accessible as blood glucose, and environmental stress or family factors involve subtleties that can be difficult to encompass. Mental health problems usually arise out of a complex interplay between biological, psychological, social and environmental factors rather than a single aetiological factor, i.e. aetiology is usually multifactorial. Each of these types of factors will be considered in turn. We will also consider protective factors.

It can also be useful to consider whether at a particular point in an individual's illness relevant factors are predisposing, precipitating and/or perpetuating. Biological and psychological factors may be more likely to predispose an individual to particular problems whereas psychological and social factors are more likely to precipitate and perpetuate problems. Aetiological factors are important to consider, as identification can help plan more effective interventions.

It is almost impossible to discuss aetiological factors without the issue of nature versus nurture being raised. Although the academic debate raises interesting points, in practice, especially in psychiatry, it is a combination of these factors. Trying to home in on single aetiological factors is usually unhelpful.

Biological factors

It is perhaps best to consider biological factors in three main groups: (1) physical disorders and insults that may secondarily cause a mental illness; (2) genetic factors; and (3) changes seen in studies of brain structure, neuropharmacology and functional imaging. These will be described in turn. Although it is unusual in psychiatry to have a single aetiological factor, this clearly is the case for some learning disability disorders such as Down's syndrome. A disorder such as Huntington's chorea, which is caused by a specific genetic disorder, can have psychiatric manifestations, but the presentation of these may be influenced by other factors such as the social circumstances and alcohol or drug abuse.

Physical disorders

Table 2.1 provides a list of some situations where a physical disorder or insult may give rise to the presentation of a psychiatric disorder, sometimes as the

Table 2.1 Physical disorders and insults that can be important in the aetiology of some psychiatric presentations

Physical factor	Related disorder or features
In utero and birth problems	
Intrauterine exposure to toxins, e.g. alcohol	Fetal alcohol syndrome Attention deficit hyperactivity disorder (ADHD)
Perinatal, brain injury at birth; premature birth	ADHD May impair intellectual abilities
Severe head injury	Mood disorders Frontal lobe disorder
Infection	
Syphilis	Dementia
HIV	Dementia; substance misuse; depression
Viral encephalitis, e.g. from herpes simplex virus (HSV-1)	Amnesic syndrome, behavioural disturbances, irritability, depression
Neurological disorders	
Neoplasm and other space-occupying lesions	Personality changes Mood disorders
Poorly controlled temporal lobe epilepsy	Schizophrenia-like psychosis
Post myocardial infarction (MI)	Depression is common after MI. Important to recognize, because morbidity and mortality are increased in the presence of depression
Endocrine disturbance	
Hyperthyroidism	Anxiety
Hypothyroidism	Depression
Addison's disease	Depression
Cerebrovascular	
Stroke	Depression, mood variability/abnormal emotionality, anxiety Changes in personality
Arteriovenous malformations	Depending on the site, may sometimes cause mood disorders or anxiety disorders
Iatrogenic	
Corticosteroids	Elation and depression
Anabolic steroids	Aggression, changes in personality
Methylphenidate	Can cause psychosis (even in very young children)

sole or main manifestation and sometimes as a secondary diagnosis. This is not a comprehensive list and aspects of this are complemented in Chapter 12 on organic disorders. It is always important to consider physical disorders in behavioural presentations and ensure appropriate and adequate investigations. Side effects of many medications can be the cause of the symptoms associated with psychiatric illnesses so a comprehensive drug history is an important part of any assessment.

Genetics

Evidence for genetic factors in the most serious mental illnesses was really the first fairly replicable indication that biological factors were relevant in aetiology. Genetic links were initially established through family and then twin and adoption studies. Table 2.2 describes the genetic risks relating to some common mental disorders.

It is useful to consider the example of schizophrenia to understand the relevance of different types of approaches. Initial population and family studies showed that where a family had an affected member the risks were greater for other family members, with first-degree relatives carrying greater risk than more distant relatives. However, this of itself does not prove a genetic link. As well as sharing some genes, families also share the same environment. Thus, it could be argued, and was by some, that aspects of how family members interacted with each other might equally be the cause of the higher risk of schizophrenia in first-degree relatives. (For example, it was suggested that abnormal patterns of communication by parents might lead to behaviours in the children that were similar to features of schizophrenia.) One way to circumvent this problem is to study twins. Monozygotic (MZ) twins share 100% of their genetic material whereas dizygotic (DZ) twins share only 50%. However, twins, whether MZ or DZ, are born and usually brought up in the same environment. Thus, differences in concordance between MZ and DZ twins, when one of a twin pair suffers from schizophrenia, are most likely to be due to genetic rather than environmental factors. Where one twin has schizophrenia the concordance in MZ

Table 2.2 Genetic risk for some major mental disorders

Disorder	Concordance rates for twins and risk to other family members
Schizophrenia	Concordance rate for twins: monozygotic (MZ) 48% compared with 17% for dizygotic (DZ). 9% of siblings and 17% of children of someone with schizophrenia may develop schizophrenia (general population approximately 1%). Second-degree relatives are 3–4 times more likely to develop the disorder than the general population.
Bipolar disorder	Concordance rate for twins: MZ is 40%–70% compared with approx. 16% for DZ. 5%–10% of those with a first-degree relative with bipolar disorder may develop a bipolar disorder (general population 0.5%–1.5%) and also more likely to develop unipolar depression. 50% of those with bipolar disorder have one parent with a history of depression.
Depression	Concordance rate for twins: MZ is 46% compared with 20% for DZ. Having a parent or sibling who has had depression increases risk by 1.5–3 times than those with no family history.
General anxiety disorder	Twin studies suggest a genetic contribution. 25% of first-degree relatives of those with general anxiety disorder will be affected.
Obsessive compulsive disorder	Twin studies suggest a genetic contribution.
Autistic spectrum disorder (ASD)	Concordance rate for twins: MZ is 30%–60% compared with DZ 0%–6%. Some variants more genetically linked. This is only the case for when the ASD is not linked to a specific genetic disorder such as Angelman syndrome or Prader–Willi.
ADHD	Some studies report 82% concordance in MZ twins compared with 38% for DZ twins. If a child has ADHD there is a fivefold increase in the risk to other family members.

twins is around 45% whereas it is only around 14% for DZ twins.

Another way to control for environmental factors is through studies following up children one of whose parents had schizophrenia but who had been adopted away from their natural parents almost immediately after birth. Their outcomes were compared with those of adopted children whose parents did not suffer from schizophrenia. Results of such studies indicate the expected population level of schizophrenia in the control children but rates of schizophrenia in the children whose biological parents had psychosis would be that expected in first-degree relatives. The rationale here is that there is no reason to suspect any major difference in the nature of the environments into which both groups of children are adopted.

What you will have noticed here is that even for MZ twins the concordance for schizophrenia is considerably less than 50%. This suggests that genetic risk itself is not always sufficient to cause schizophrenia and that certain environmental factors must also operate. Thus, from a clinical perspective, it is important to emphasize to patients and their families that although major mental disorders carry a genetic risk, there is no inevitability that the children will also have the disorder. When there is a family history of mental illness, especially schizophrenia, behavioural disturbance in children can be misinterpreted and the child given a particular script that is hard to escape.

Genetic epidemiology approaches, such as described above, have established that genetic risk is a factor in many mental illnesses. Since the 1980s, investigators have turned their attention to use of modern molecular genetics and biological techniques (Box 2.1) and have found a number of chromosomal regions and some genes that seem to be linked to schizophrenia (see below), severe mood disorders (e.g. unipolar depression SSIRT1; bipolar disorder CANA1C) and alcohol dependence (e.g. GABRA2). It has become fairly clear that for most mental illnesses there are not just going to be only one or two genes involved, each with a large effect. For schizophrenia, for example, at least eight genes (e.g. neuregulin, dysbindin, DISC1) have been reasonably well replicated in studies as being associated but, because schizophrenia (like other mental disorders) has a multifactorial aetiology, in any individual the presence of the pathological allele of one of these genes is likely to be only one of the factors increasing their risk of developing schizophrenia and will rarely be the sole cause. Because each potential gene is of small effect, and because diagnosis can be imprecise, these studies require many hundreds of patients and controls, making them difficult to carry out. A further problem is that we do not know the functional effects of many of these genes in the brain. Thus, the available knowledge has not yet helped to understand a pathway by which these disorders are caused and thus cannot yet lead to prevention or pre-emptive treatment.

Box 2.1 Three main approaches to study of genetics in mental illness

1. *Genetic epidemiology* asks:
   a. If relatives of cases have a risk in excess of the population baseline?
   b. Is this increased risk attributable to genes or environment?
2. *Molecular genetics* asks whether certain alleles are more common in affected than unaffected individuals
   a. Linkage studies – are a trait and large chromosome segments inherited together in families?
   b. Association studies – are small segments of DNA more common in affected than non-affected individuals?
   c. Genome wide association studies (GWAS) use knowledge of genome maps, automated techniques and genetic samples from large populations to screen the whole genome for probabilities of relevance for specific genes
3. *Molecular biology* looks at the effects of genes and their regulation

Brain structure, neuropharmacology and functional imaging

Serious consideration of biological factors in mental illnesses (other than those described in the 'Genetics' section of this chapter) began towards the end of the nineteenth century as interest in neuropathology expanded. Studies were reported of the examination of post-mortem brain samples from patients who had

suffered from various types of psychotic disorders as well as from those who had suffered from dementia and other neurological conditions. Whereas studies of the dementias began to reveal the neuropathology of these conditions, studies of the so-called 'functional' psychiatric disorders were unsuccessful. (Psychiatric disorders without clear evidence of neuropathological abnormality were for many years described as 'functional' disorders because they could only be recognized as disorders of brain function without measurable evidence of physiological or other biological abnormality as was possible for disorders such as dementia.) This type of approach was hampered by inadequate techniques for measurement of what we now know are subtle abnormalities, by a lack of clear diagnostic criteria and by the complicating effects of brain injury, vitamin deficiencies and other such factors that often afflicted patients with chronic mental illnesses over a 100 years ago.

The early twentieth century saw a preoccupation with psychological factors and psychoanalytical approaches. However, the development of effective biological treatments (electroconvulsive therapy [ECT] in the 1930s; antipsychotics in 1952; antidepressants in 1957), improvements in chemical/biochemical techniques and increased understanding of biochemistry led to a steady resurgence of interest in biological investigation of mental illnesses during the 1950s, which accelerated exponentially over the following five decades.

Initially many studies were aimed at trying to find biological markers for various mental illnesses. For example, patients with schizophrenia were examined for putative psychotogenic substances that could theoretically be produced in the brain, from known metabolic pathways, and which, if in excess, might cause psychotic symptoms. The transmethylation hypothesis suggested the possibility of increased concentrations of 3,4-dimethoxyphenylethylamine, which Friedhoff and van Winkle identified on chromatography of urine samples, from patients with schizophrenia, as the famous 'pink spot'. Although many interesting results were initially found, these could generally not be replicated by different investigators with their own patients and sometimes turned out to be secondary effects of drug treatments (as was probably the case for the 'pink spot').

During the late 1950s and early 1960s a more fruitful approach emerged of trying to understand how the now available effective drugs worked. For example, in 1957 Arvid Carlsson demonstrated that dopamine (DA) was a neurotransmitter, a finding that later led to development of the first effective treatment for Parkinson's disease. Of relevance to psychiatry, he also demonstrated, in 1963, that antipsychotic drugs had profound effects on the DA system. This finding began the cascade of research leading to the DA hypothesis for schizophrenia proposed by a number of scientists in the early 1970s. (In 2000, Carlsson received the Nobel Prize for Physiology and Medicine for his lifetime of work on various aspects of DA function.) Similarly, working from the effects of antidepressant drugs, in the 1960s Schildkraut and Coppen, respectively, proposed noradrenergic and serotoninergic hypotheses for depressive illness. Although current knowledge suggests that the early versions of these hypotheses require considerable modification, this approach led to a greater interest in trying to understand neurochemical processes in the brain and how these might be disrupted in mental illnesses.

Success in demonstration of the relevance of particular neurotransmitter pathways, the development of more sophisticated brain imaging techniques and the development of molecular approaches to neuropathology encouraged renewed attempts to understand other types of biological factors in the aetiology of mental illnesses. For example, although early pneumo-encephalographic studies demonstrated increased lateral ventricular volumes in patients with chronic schizophrenia it was not until the advent of computed tomography and magnetic resonance imaging, respectively, in the 1970s and 1980s that this type of finding could be properly and safely investigated in large numbers of patients at different stages of illness. This has led to the establishment of consistent evidence of brain structural changes in schizophrenia and mood disorders, to the resurgence of neuropathological and molecular studies of post-mortem brain tissue from patients using modern techniques, and to hypotheses such as disturbance of brain development as an aetiological factor in schizophrenia.

Schizophrenia

A wide range of biological and cognitive disturbances have been found to be associated with schizophrenia.

Table 2.3 summarizes some of the structural brain changes observed that have been relatively consistent in their association with schizophrenia in recent neuroimaging and neuropathological studies. These changes are small in absolute terms, and sizes of the brain structures involved overlap considerably with those in non-schizophrenic subjects. Thus, such changes are only seen when comparing a large number of patients with control subjects and cannot be used to help make a diagnosis of schizophrenia. These structural abnormalities have been demonstrated in patients with established illness but some appear to be present at the onset of the illness and to progress over time. Along with other evidence, for example of people with increased obstetric complications during gestation of people subsequently developing schizophrenia, the effects of maternal influenza during pregnancy increasing the risk of schizophrenia in the child (by 1%–2%) and the presence of increased frequencies of a number of minor physical anomalies that are determined in utero (e.g. palmar creases and craniofacial abnormalities), these findings suggest that in many cases an important factor may be neurodevelopmental disturbance.

Other studies have suggested disturbances of physiological function (Table 2.4). These changes are often best demonstrated when patients are asked to perform specific cognitive tasks (such as certain memory tasks or verbal tasks) which will normally activate particular brain regions. It is also clear that patients with schizophrenia have a variety of cognitive impairments. These are particularly in the areas of executive function (planning and decision-making), attention and memory. Such impairments have often been found to correlate with both the 'negative' symptoms of schizophrenia and poor outcome in terms of social functioning.

During the 1960s and 1970s a combination of laboratory studies, clinical observations and post-mortem studies suggested that the 'positive' psychotic symptoms of schizophrenia (see Chapter 9) might be due to overactivity of the DA system and primarily to an underlying increase in DA type 2 (D2) receptors (Table 2.5). The advent, in the 1980s, of techniques such as single photon emission tomography (SPET) and positron emission tomography (PET), which allow measures of neurochemical function in live human beings, did not provide consistent confirmation of the first version of the DA hypothesis, which suggested an underlying increase in DA receptors.

However, subsequent, more sophisticated studies have suggested that there may be an underlying tendency towards DA overactivity in the striatum and underactivity in the prefrontal cortex of the brain

Table 2.3 Structural brain changes found in schizophrenia

Finding	Comment
Reduced overall brain volume	Meta-analysis of 58 studies suggests 2% reduction in cerebral volume
Increased volume of cerebral ventricles	Particularly the temporofrontal aspect of the lateral ventricles
Reduction in volume of the hippocampus	Possibly greater on the left side
Reduction in volume of the hippocampus	In discordant monozygotic twins, the affected twin has reduced hippocampal volume
Reduction in volume of parts of the frontal and temporal cortices and possible alterations in gyral folding	Findings in some studies relate to particular symptoms

Table 2.4 Disturbances of brain function

Finding	Comment
Reduction in amplitude of some 'event-related' potentials, such as P300 and mismatch negativity (MMN)	These are complex EEG measures that indicate disturbances in how individuals process information
Disturbances in regional cerebral blood flow measured using PET or functional MRI	Evidence that some of these changes relate to particular groups of symptoms
Altered pattern of blood flow when performing specific cognitive tasks	Suggests some alteration in the normal pattern of connections between different brain regions

Table 2.5 Evidence suggesting altered dopamine transmission in schizophrenia

Finding	Comment
Potency of antipsychotics in binding to dopamine (DA) receptors correlates with their clinical potency.	Ability of these drugs to bind to other receptors (e.g. serotonin, histamine) does not correlate with clinical potency.
Amphetamine abuse can induce some schizophrenia like symptoms.	Amphetamine stimulates the DA system.
Clinical trial in schizophrenia of alpha- and beta-isomers of flupentixol showed benefit only for the alpha-isomer.	Only the alpha-isomer blocks DA receptors. This isomer reduced positive psychotic symptoms.
Increased density of DA receptors in post-mortem brain tissue from patients.	Initial studies agreed but then evidence emerged that this effect might be caused by the long-term antipsychotic treatment most patients had received. Little or no change seen in most studies of patients free of antipsychotic drugs for some time before death.
Studies using PET to measure DA receptors in vivo in drug-naïve patients fail to find consistent increases.	Problem of small size of many of these studies. Meta-analysis suggests there may be a small increase.
Single photon emission tomography (SPET) and positron emission tomography (PET) studies used to show that patients release more DA than control subjects when given amphetamine.	Suggests some dysregulation of the DA system in patients with schizophrenia.
PET studies show greater increase in DA receptor binding in patients with schizophrenia after depletion of DA.	Confirms dysregulation of the DA system in patients with schizophrenia.
Cannabis induces release of DA in the striatum.	Increasing evidence that cannabis abuse increases the risk of developing schizophrenia.

in schizophrenia. The knowledge that DA is the key to how we attach importance or 'salience' to events or thoughts fits well with the evidence that patients with schizophrenia demonstrate inappropriate salience compared with healthy subjects. Such abnormal salience could form the basis of delusional thinking. These findings also suggest a mechanism by which cannabis abuse might increase the risk of developing schizophrenia as cannabis can induce release of DA and may thus alter the salience attributed to events.

There has also been evidence of dysregulation of the excitatory neurotransmitter glutamate. For example, drugs such as phencyclidine (PCP), which block glutamatergic *N*-methyl-D-aspartate (NMDA) receptors, can induce psychotic states. Glutamate is the main excitatory neurotransmitter in the brain and general dysfunction of this, or dysfunction of certain major pathways, could affect functioning in many brain regions and secondarily disrupt other transmitter systems, such as gamma-aminobutyric acid (GABA) and DA. Post-mortem studies have shown alterations in the glutamatergic system in the hippocampus. These findings are consistent with other neuropsychological and neuropathological data.

The second-generation antipsychotic drugs have potent effects on serotonin or 5-hydroxytryptamine (5-HT) receptors, principally 5-HT_{2A} receptors. This may account for some of their benefits and has stimulated research on the 5-HT system in schizophrenia. At present, there is no conclusive evidence for any specific abnormality but it may be a system through which other transmitters can be modulated in certain brain regions relevant to particular symptoms.

Depression

Until the 1990s, the main focus of investigation into the biological aetiology of depressive illness was into disturbances of neurotransmitter function, particularly 5-HT and noradrenaline (NA). Table 2.6 summarizes much of the evidence in favour of a serotonergic dysfunction. Like the schizophrenia story, this comes from a variety of laboratory, clinical and post-mortem brain studies. A useful paradigm used to investigate mood change, in both patients and healthy individuals, has been the tryptophan depletion paradigm (experimental approach). This paradigm depends on the fact that the amino acid tryptophan is the essential precursor for the formation

Table 2.6 Evidence suggesting altered serotonin function in depression

Finding	Comment
Reserpine may induce depression.	Reserpine depletes neurones of 5-hydroxytryptamine (5-HT) and noradrenaline.
Patients respond to selective serotonin reuptake inhibitors (SSRI) antidepressants.	These drugs have fairly specific effects on the 5-HT system.
Cerebro spinal fluid (CSF) levels of 5-hydroxyindoleacetic acid (5-HIAA) (the main brain metabolite of 5-HT) are reduced in depression.	This suggests reduced release of 5-HT. It is particularly found in those with previous suicide attempts.
Tryptophan depletion may induce depressed mood in recovered patients.	Artificial reduction in serotonin induces depressed mood lending strength to the idea that 5-HT function may be reduced in those with depressive illness.
Reduced secretion of the hormone prolactin following stimulation of the 5-HT system.	Suggests altered function of 5-HT receptors controlling this.
Post-mortem brain studies suggest reduced 5-HT_{1A} receptors and increased 5-HT_{2A} receptors.	
Molecular imaging studies using PET suggest reduced 5-HT_{1A} receptors in depression.	
PET studies less consistent with regard to 5-HT_{2A} receptors.	

of serotonin. An important enzyme in this process, tryptophan hydroxylase, is not saturated with tryptophan at physiological concentrations, so any reduction in availability of tryptophan will result in a fairly rapid fall in serotonin levels in the neurons and synapse. Subjects are asked to drink a mixture of amino acids, normally present in the proteins we eat, which do not contain tryptophan. This results in a dramatic, short-term fall in brain serotonin and will induce a temporary relapse of depression in recovered patients (rapidly reversed on being given tryptophan in a normal meal) and in some individuals with a familial genetic risk of depression.

The paradigm of tryptophan depletion is also used in a variety of other ways to help us understand what may be happening in depression. In conjunction with measures of cerebral blood flow it is found that reducing serotonin function in the brain, by tryptophan depletion, alters activity in the orbitofrontal cortex, an area also implicated in other studies of brain structure and function in depression (Table 2.7). It has also been demonstrated that subjects who have been tryptophan depleted may show changes in emotional processing similar to those that may occur in depression.

A similar but smaller body of evidence also suggests that there may be dysfunction of the NA system in patients with depression, although this may be more specifically linked to particular symptoms or types of presentation.

Table 2.7 Disturbances of brain structure and function in depression

Findings
Reduction of grey matter volume in the orbitofrontal region of the cortex
Reduced volume of the hippocampus in patients with a long history of depression
Reduction of blood flow in orbitofrontal cortex
Increased orbitofrontal cortical blood flow following tryptophan depletion
Reduced blood flow in the caudate nucleus

A further interesting series of findings relate to the hypothalamic–pituitary–adrenal (HPA) axis. It has been known for many years that patients with depression have elevations of plasma cortisol compared with a healthy population, although not sufficiently high to make this diagnostically useful. A large proportion of these will fail to suppress cortisol after administration of dexamethasone. Magnetic resonance imaging (MRI) studies indicate enlargement of the adrenal glands in depression. There is also a blunted adrenocorticotropic hormone response to corticotropin-releasing hormone. This suggests some abnormality of receptor function normally designed to detect elevated cortisol secretion and thus a dysregulation of the HPA axis. Stress will normally cause

activation of the HPA system and can be a precipitant of depression. Thus it may be that there is an inability of the HPA system to switch off following stress, and hypercortisolaemia could thus play a part in the development of depression. Elevated cortisol may also be damaging to the hippocampus and thus MRI findings of reduced hippocampal volume in some patients are consistent with this.

Bipolar disorders

Intensive investigation of the biological changes in bipolar disorders began a little later than such studies in schizophrenia. However, the past 10 years have seen consistent evidence emerging for brain structural changes that seem to differ a little from the pattern seen in schizophrenia. In particular, there has been evidence for reduction in the grey matter volume in parts of the prefrontal cortex, enlargement of the lateral ventricles and enlargement of the globus pallidus. Like the situation in depression, there is also evidence for reduced cerebral blood flow in the orbitofrontal cortex and anterior cingulate gyrus. However, in a state of mania patients seem to experience an increase in blood flow in these regions.

All three monoamine neurotransmitters have been implicated in bipolar disorders. There is evidence for an underlying underactivity in the 5-HT system. In states of mania, there is evidence for overactivity in both the DA and NA systems. Disturbance of the HPA axis has also been demonstrated, with evidence of more marked change in the depressive phase of bipolar disorders than in unipolar depressions.

Anxiety disorders

Over the past 20 years, evidence has accumulated for the importance of biological factors in some types of anxiety disorders. It is more difficult to study this group of disorders because of diagnostic overlaps and coexistence of different anxiety disorders in many patients. However, it is clear that benzodiazepines (BDZs) and a variety of antidepressant drugs are effective in treating anxiety in many, though not all, patients. The effectiveness of BDZs led to investigation of the possibility of altered sensitivity of the GABA–BDZ receptor complex in some anxiety disorders. In patients with panic disorders the GABA–BDZ receptor antagonist flumazenil has been shown to induce feelings of anxiety and panic while having no effect in non-anxious control subjects. This suggests that there may be some alteration of function of GABA–BDZ receptors in these patients. This effect is not seen in patients with post-traumatic stress disorder (PTSD). However, in PTSD and panic disorder patients the drug yohimbine (an alpha-2 noradrenergic receptor agonist) can induce feelings of panic, while only inducing milder anxiety in control subjects, suggesting altered function of the NA system.

These are examples of the evidence for biological abnormalities in anxiety disorders. It is not feasible here to summarize the principal evidence for biological factors in all of the anxiety disorders. However, it is crucial to recognize that while many developmental, social and psychological factors are important we must remember that for many mental illnesses it is the interplay of all of these potential aetiological factors that is important – an interplay where the balance of each will differ from patient to patient.

Psychological factors

Individual personality traits and personality types are significant factors in the development of mental health problems. This encompasses temperament type, which is often used for children. The individual's early development and attachment to carers may influence their ability to develop appropriate and meaningful relationships and may be significant aetiological factors. The developmental life stage that someone is at may also be an important factor.

Personality types

Temperament is used to describe the way individuals and especially children behave. It does not attempt to explain what causes the behaviour. Temperament is present from birth and influences how we behave towards people and how we see our environment. This helps understand why some individuals are able to manage many stressors whereas others develop problems in the face of what others may see as minor stressors. It is also important to note that our behaviour may lead others to behave in particular ways which may then establish helpful or non-helpful coping strategies. There is strong evidence that some temperament types in children, which become

personality traits as they mature, are more strongly linked with some mental health problems than others. Problems in childhood are more likely when the child has a temperament that fits less easily with parental styles.

In terms of thinking about personality types, it may be helpful to use the *International Classification of Diseases* (*ICD*) categories of personality disorder (Box 2.2). That is not to say that everyone with those traits develops problems, but identifying the types of personality disorders helps relate the personality characteristics to the most likely mental health problems. (Issues around personality disorder are discussed in Chapter 16.) Young people under the age of 18 years cannot be diagnosed as having a personality disorder, and the term emerging personality disorder may be used. However, given that adolescence is a stage of life when young people are establishing a sense of who they are, some of the behaviours, especially those of the emotionally unstable type, may be transient.

On the whole, personality traits are enduring but that does not mean individuals cannot learn ways of managing the behaviours around certain traits. Most of us will not fit the full criteria for a specific personality disorder but most, if not all of us, will have some of these traits. A helpful way of looking at these is to use broad categories are as follows:

- Odd/eccentric types (such as paranoid or schizoid)
- Emotional, erratic types (such as dissocial, histrionic, emotionally unstable)
- Anxious/fearful types (dependent, anxious, anankastic)

Individuals in the first category tend to be loners and can struggle with relationships. Individuals with such traits (as opposed to the disorder) are more likely to present with problems when these personality traits interfere with daily living. They may present with depression and/or feelings of persecution. The emotionally unstable group of traits are most likely to present with deliberate self-harm, aggression, substance misuse and co-morbidity. Unsurprisingly, those with anxiety and obsessive traits are more likely to present with anxiety and phobic type of disorders. However, just because they are predisposed to these types of disorders does not mean that these are the only disorders they may have. There are other recognized types of personality, although not classified as disorders, such as depressive (unsurprisingly characterized by chronic moroseness, worry and negativity) and passive aggressive types. The latter can be very difficult to deal with in practice as they will not openly raise or address issues but instead act out their hostility. For example, they may disagree with a management plan suggested by the doctor. However, rather than say so they will not comply with the treatment and then claim it is ineffective. The way we think about personality has come some way from when the way people physically looked was used to define their personality!

It is important to recognize that someone with anxiety traits who is supported by addressing some of the issues may learn to manage their anxiety (even though it is unlikely ever to completely go away). However, if the anxiety is managed with excessive reassurance and support of avoidance strategies, the situation is likely to be exacerbated. Particular personality types may be more prone to use particular kinds of defence mechanisms as discussed in Chapter 3, but we all use different defence mechanisms to manage situations. Stress may arise after experiencing adverse events or may result from poor or ineffective coping strategies. Stress in those with

Box 2.2 ICD classification of personality disorders

F60 Specific personality disorders

Paranoid – includes previous categories such as sensitive and querulant personality

Schizoid – not the same as schizotypal disorder (related to schizophrenia)

Dissocial – more used term is antisocial, psychopathic or sociopath

Emotionally unstable – either impulsive or borderline type

Histrionic (was hysterical)

Anankastic (was obsessive)

Anxious (also called avoidant)

Dependent (was asthenic, inadequate or passive)

Other specific personality disorders, including narcissistic personality disorder

F61 Mixed or other personality disorders

anxiety traits may be more easily identified as it is expressed most obviously.

Attachment

It was after World War II that Bowlby wrote about attachment of babies to their mothers thus reflecting that at the time he wrote, mothers were the primary caregivers. Attachment refers to a specific type of biologically based relationship which provides a secure base from which children can explore the world. Attachment relationships can be classified as either secure or insecure. Essentially with secure attachment, the child learns that their needs are met by their caregiver and thereby learns trust. This is not the case for insecure attachments. Three types of insecure relationships have been identified: ambivalent, avoidant or disorganized. Insecure attachment relationships, coupled with high levels of parental anxiety, are associated with increased risk of anxiety disorders in children. Those with ambivalent insecure attachment may develop relationships with strong passive-aggressive patterns of behaviour. Disorganized insecure attachment may lead to short-term superficial relationships in which there is little trust.

Individuals who have secure attachment and a good model for functional relationship are more likely to develop and maintain healthy relationships. Over time it has become clear that the particular person the child develops the relationship with is less important than the nature of the relationship itself. That is, the relationship does not have to be with the biological mother as first hypothesized. A relationship that is warm and positive with someone familiar may be enough. See Chapter 14 for more information.

Self-esteem

The way we view ourselves can influence how we view what happens to us. Individuals with a good sense of who they are and positive self-esteem are less likely to be impacted on by some environmental factors. If they experience a life event, they are less likely to feel persecuted or picked on and more likely to see it as just one of those things. Children and adults with poor social skills find it difficult to develop relationships, so may find themselves excluded and socially isolated. Social skills can be improved through training.

Intelligence quotient

Generally, children with lower cognitive levels are at increased risk of antisocial behaviour. Research suggests that lower intelligence quotient (IQ) is a risk factor because such children have poorer problem-solving skills and are more vulnerable to family adversity. This risk is carried on into adulthood. Having special skills can sometimes mitigate other risk factors. For some individuals talent is also a way of escaping socio-economic disadvantages and the risk inherent within them, which is discussed later.

Life stages and life events

The life stage is important to consider as some behaviours are appropriate at some stages but become a problem if they continue. This can be developmental stages or following life events.

Life events could arguably be environmental factors but are included here as we all experience them. Whether a life event becomes a precipitating or perpetuating factor will depend on the interplay between the life event, an individual's personal characteristics and other factors. The most stressful life events are bereavement, divorce and moving house or job.

The experience of success or failure at dealing with expected life events can also shape our world view. If one set of events such as exams are managed well, we learn that exams are okay and are less likely to be stressed by future exams. However, if we manage events ineffectively and are distressed by the experience, the next time we come across the same event we are likely to remember our previous failing. It can also be the case that being mugged reinforces our predisposition to believe that the world is a dangerous place and thereby reinforces any anxiety traits. Although having an episode of mental illness does not inevitably mean problems will recur, the likelihood of further problems is increased.

Social factors

There are a variety of social factors with many subcategories (Box 2.3). The major categories are environmental, life events, cultural and sociopolitical factors. Each will be considered in turn. Disentangling genetic from environmental influences is not easy.

Box 2.3 Aetiological social factors

- Culture
- Religion
- Family
- Neighbourhoods and social networks
- Work and school
- Socio-economic disadvantage
- War/conflict

Bronfenbrenner's model of layered contexts is a fairly widely used model to help understand social factors. The layers are the microsystem, mesosystem, exosystem and the macrosystem. The first layer (microsystem) is relationships that the individual is actively involved in, for example parents, siblings and peers. However, these interactions depend on context and personal characteristics. The mesosystem describes how the different components of the microsystem come together. The exosystem is the wider local community such as the neighbourhood. The macrosystem is usually remote from any specific individual but may still be a major social influence, for example socio-economic policy regarding child rearing, benefits for those who are ill, education and health policies, and wider cultural and political contexts. It is worth noting that various factors may influence differently at various stages of an individual's life. Some adverse family factors may be mitigated by external factors in older children. The broad political and cultural aspects are not covered in detail in this chapter as they vary from context to context. However, it is important to recognize their potential significance.

Environmental factors

There is a wide range of environmental factors and the influence of these will be individually and contextually variable.

Cultural factors

One of the difficulties in thinking about culture is that it can often mean different things to different people. It is beyond the scope of this text to enter into the debates about the meanings of culture. In this context culture is not predefined but takes into account the various aspects of an individual's life that are important to them, so can include ethnicity, geographical identification and religious perspectives.

Culture in itself (no matter which definition is used) does not cause mental health problems. However, it can strongly influence what is perceived to be a mental health issue, what treatments are sought and potentially even the support provided. There is some evidence that the prevalence in some groups is higher in one context than in another. For example, Afro-Caribbean migrants in the United Kingdom have higher rates of schizophrenia than the same population group in the West Indies. This suggests that the risk is neither entirely genetic nor social but an interplay of the two that manifests itself differently dependent on context.

Religion

Religion can be a protective factor against developing mental health problems but this has to be understood in context. For many people, if religion challenges their sense of who they feel they are it can be considerably stressful, especially when there are strong differences within the family. A very obvious example of this is homosexuality.

Families

Families are a crucial social factor as they can provide enormous support to those with mental health problems but they can also cause considerable stress. Not long ago, parents were thought to be responsible for their children developing autism and schizophrenia. As with culture, families do not cause problems *per se* but may be significant factors in the development of problems. The nature of what a family is varies considerably. As more and more families move away from the traditional nuclear family, the stresses that families bring change. The reconstitution of stepfamilies brings challenges within that process. An additional factor to consider is extended family, and the various supports and stresses that this type of arrangement might provide. Our relationships with our parents and families often form the template for all our other relationships, no matter how much we might wish they did not.

The impact of families will be considered for children, working age adults and older adults as the

relationships differ. For children parents can be significant factors through the following:

- Parenting styles
- Parental relationships (parental hostility rather than whether parents live together or not seems to be a key factor)
- Parental mental or physical illness. Maternal postnatal depression may be particularly relevant, as if untreated it may impact on bonding and attachment
- Abuse (children who experience abuse may externalize their hostility and develop aggression whereas others may internalize their feelings and self-harm, and present as anxious or depressed)
- Unresolved parental issues related to their own upbringing
- Differing expectations of what the child wants and what might be expected of them

Other factors for children will be if there are siblings who have chronic physical health problems or a learning disability. Sibling relationships can also be relevant, although relationships in childhood do not always indicate the type of relationship siblings will have as adults.

For working adults, families can be relevant aetiological factors through the following:

- Their relationship with their partner
- Relationships with and demands of their children
- Relationship with their own parents, especially when their parents become less independent or suffer a loss

For older adults families are relevant in the following ways:

- Issues around ageing and whether older people feel supported by their family
- Bereavement and loss of partners and siblings

It is often the case that the staff working with children identify that parents need to address the child's issues. Staff working with adults may feel that if child services just got the child sorted, their patients would be less stressed. The reality is that the key issue is how this dynamic plays itself out. How each player in the scenario can be helped to understand their role and the impact it has on others may be a more productive way forward. Another way of looking at it is that a depressed mother may not be able to respond warmly to her child and the child then responds by defiance, which then makes it even more difficult for the mother to respond. Breaking such a cycle can be difficult but identifying the relevant factors is important to devise an appropriate intervention plan.

The way that families interact and communicate can affect all members of the family and it is often the case that what impacts on one person has some degree of influence on other family members. It is generally agreed that clear communication, direct style and being emotionally responsive to family members while allowing them some autonomy is ideal. Few families achieve this on an everyday basis and go through life finding mutually acceptable ways of managing.

It can be easy to dismiss the impact of families on adults. Research in the latter part of the twentieth century found that families who were overly expressive played a part in the relapse of schizophrenia. The family members may have unwittingly been factors in perpetuating the illness. This is not the same as saying the family is responsible for the illness.

Families are an important factor in how we view ourselves and the parts we are allowed to play. It can be very difficult to change the narrative, so if you have been labelled as shy within the family context it may be difficult to be anything else.

Domestic violence in families can cause mental health problems for the abused partner (usually female) and for the children. Males may especially model their behaviour on their aggressive fathers.

EXERCISE 2.1

As you go through your psychiatry placement, consider how people describe their families and whether they identify them as a strength or stressor. Think about how you feel about your own family and how that influences your expectations of patients and their families. In your placement after psychiatry, repeat the exercise and see if there is any difference in the range of ways people view their families.

Neighbourhoods and housing

The places in which people live can have significant impact on their mental health. As discussed in the section 'Socio-economic disadvantage' below, poorer people live in less desirable areas and may have to tolerate high levels of crime, violence and risk to their personal safety. Neighbourhood disadvantage is associated with higher rates of major depression and substance abuse disorder. Those suffering from mental illness may also experience a loss of financial security and be over-represented in poorer areas.

However, this is also dependent on social networks and support within the neighbourhood. Social isolation can be a risk factor for depression, especially in older adults. Migrants may find living in areas with high levels of similar migrants more of a support than those who are not migrants, as for the latter it may represent greater and more enduring socio-economic disadvantage. The value of support networks should not be underestimated. Such support may help mitigate the consequences of poverty, disadvantage and discrimination.

Work and school

Individuals spend a considerable amount of time in either work or school. Many manage this without any problems. However, the demands of work or school can provide considerable stress, as can having to develop working relationships with colleagues and peers. For older adults the loss of work may bring other losses such as a purpose and a sense of being valued, social contacts as well as reduced financial security.

Life events (which are not a normal expected part of life)

Trauma, sexual assault and being a victim of crime may all decrease the threshold for someone developing mental health problems, especially anxiety and depression. However, most people who experience trauma do not suffer from PTSD or develop other problems.

Sociopolitical factors

Socio-economic disadvantage

A key factor is socio-economic disadvantage, which brings with it a range of stressors that in turn impact on the development of some mental health problems. This factor is significantly associated with development of mental health problems in children and adults and is relevant across cultures. It is not within our scope to discuss what constitutes poverty. However, whichever way it is measured or defined, there is no doubt that poverty is linked to social inequality and these factors coupled together impact on the overall health of individuals. There is higher mortality and morbidity in deprived communities than in less deprived ones. Being poor means that meeting tasks of daily life can be challenging. Poor people have less access to safe neighbourhoods, decent housing and jobs and to health and education. The disadvantage therefore becomes cumulative. Poverty can cause mental health problems but may also be a consequence of them. Children from disadvantaged backgrounds are less likely to perform well in schools, are more exposed to unstable family life and are more likely to have behavioural problems. Adults from disadvantaged contexts are more likely to develop mood disorders and may experience more anxiety (which may well, for example, be justified if they live in violent neighbourhoods). Substance misuse problems are also more prevalent among lower social classes but it may be that spending what money they have on substances leads to greater problems than substance misuse in more affluent groups.

War and conflict

Besides causing considerable anxiety, war and conflict also lead to refugees and asylum seekers. The potential trauma experienced (not just from bombings but associated insults such as rape and torture) and the following displacement (and associated losses) are considerable stressors and increase the risk of mental health problems. War and conflict may also lead to oppression and denial of human rights, which increase the likelihood of developing mental health problems. War also carries a risk of PTSD for those actively involved as soldiers.

Resilience

As well as being able to identify risk factors that increase the risk of developing problems, it is also important to be aware of how individuals can be helped to develop resilience (Box 2.4).

Box 2.4 Factors promoting resilience

Personal characteristics

Positive self-esteem

- Secure attachments with an ability to trust others
- Have experienced authoritative parenting which gives the individual a sense of mastery and self-sufficiency
- Experiencing success
- Interpersonal abilities such as social skills, problem-solving skills, impulse control and a range of coping strategies

External factors

- Being safe
- Positive and supportive relationships
- Sense of belonging through religious or other affiliations

Source: Modified from Ahmed AS, *Advances in Psychiatric Treatment,* 13, 369–375, 2007.

Summary

In this chapter, we have reviewed the major aetiological factors that play a part in the development of mental disorders and mental health problems. There is no single aetiological factor in any major mental disorder. Rather, it is the complex interplay between the biological, psychological and social factors that leads to problems. In terms of understanding what causes mental health problems, despite the progress made, we still have some way to go. From a practical perspective because individuals are different and see the world in such different ways, the environmental factors will always influence people differently. In your clinical practice, remember that whatever the genetic risks, anyone with enough environmental challenges can develop problems.

Further reading

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CASE STUDY 2.1

Clare is 16 years old and presents to her GP with anxiety about her upcoming exams. She is struggling to concentrate and focus on any revision. She often begins by making a list but then becomes overwhelmed by what lies ahead. She is often tearful and relates this to a recent bereavement. Six months ago her paternal grandmother to whom she was very close passed away after a long and painful illness (breast cancer).

Clare was a normal pregnancy and delivery. There were no early concerns regarding her development and she started school without any major problems though took a while to settle. Her father has struggled to cope with the loss of his mother harbouring feelings of guilt as he does not feel he helped his mother as much as he could. Over the last few months he has tended to shut himself off from the family and started working longer hours. He has been more irritable and his relationship with Clare has suffered. There is no family history of mental illness although Clare's mother reports she can struggle with new situations and that her husband finds change difficult. The family do not really talk about the grandmother although when she was alive they had frequent and regular contact.

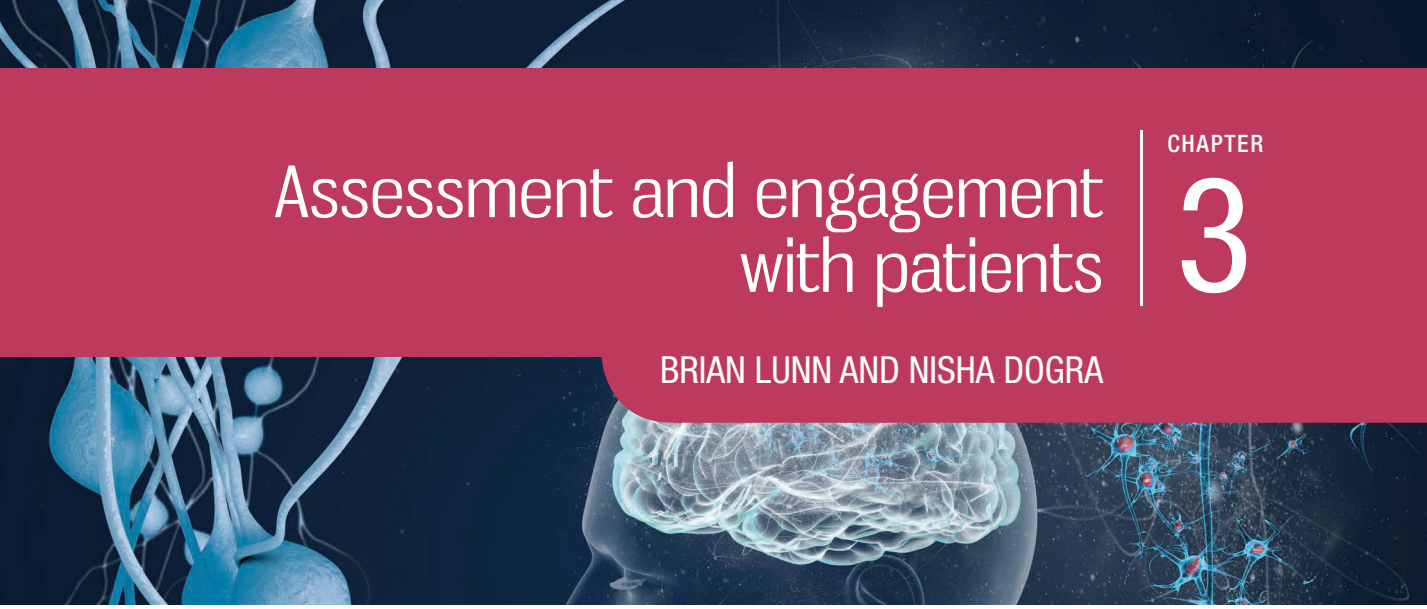
Who in this scenario has mental health problems?

From Clare's perspective, what are the possible aetiological factors?

Once you have identified the factors draw a table outlining what type of factor it is and whether it is predisposing, precipitating and/or perpetuating.

What would change if her father has developed an alcohol misuse problem?

What might be the aetiological factors to consider in this case?



CHAPTER 3

Assessment and engagement with patients

BRIAN LUNN AND NISHA DOGRA

KEY CHAPTER FEATURES

- Key elements of psychiatric history
- Mental state examination
- Cognitive examination
- Physical assessment and investigation
- Explaining the importance of integrating cultural issues into the assessment and management process
- Establishing effective working relationships with patients
- Outlining principles that are useful in developing therapeutic relationships with patients
- Defence mechanisms
- How you can develop your own communication style

Introduction

The first part of the chapter deals with the content of psychiatric history taking and mental state examinations. The second part of the chapter deals with some of the principles of developing rapport with patients and also looks at the process of developing a therapeutic relationship. First encounters with patients with mental health problems are often daunting for medical students. One significant concern is that taking a psychiatric history is in some way different from assessment in other areas of medicine. It is actually the case, however, that the psychiatric history has much in common with taking good histories in any area of medicine. The history needs to be taken in a systematic way using good interview techniques and with a desire to understand the patient's experience. Histories will only be comprehensive if they are taken by someone who has a sound knowledge of the signs of mental illness, such as

delusions and hallucinations, and knowledge of how to clarify exactly what the patient is presenting with.

History

In whatever setting it occurs, history taking is a process of obtaining information and testing out hypotheses with the aim to fully understand the nature of the patient's experience. This is only possible when the focus is not too narrow, as in many cases, psychiatric and physical problems are present at the same time and interact with each other: physical disorders may have a significant psychological component and psychiatric disorders may present first to a non-psychiatrist. It is therefore important that psychiatric symptoms are explored in all patients, and that physical and psychiatric disorders are considered together. All doctors must therefore be comfortable asking about psychiatric symptoms.

The basic principles of taking histories in patients with psychiatric symptomatology are set out in Box 3.1.

Every history should start in as an open format as possible as this is the only way that a patient can explain what is concerning them. It is important not just to accept what the patient says at first at face value as there may be things they are embarrassed about or not realize the significance of. Questioning patients about information can be a delicate learning process between patients and doctor, as patients may feel they have already given you the key facts. However, it is often helpful to explain that you need to ensure a shared understanding of how they are using terms, given that terms such as depression may be used differently by the public and health professionals. Once you have identified what you think the main issues are, summarize this list and check that you have identified everything and understood what the issues actually are.

Box 3.1 Basic principles of history taking

- Begin with open questions
- Shift to more directive (closed) questions to clarify what the patient means and what their symptoms are
- Establish a timeline setting out the sequence of the patient's experiences and relevant events
- Allow the patient to talk
- Try and obtain an understanding of the patient's experience
- Be empathic

Once the problems have been identified, there needs to be a shift to the use of more directive questions to test out hypotheses about what the patient is experiencing and to seek clarification of their symptoms. To use an analogy that may be more familiar to medical students, if a patient complains of pain, the student would normally ask about the nature of the pain, its site, any radiation, how the pain varies in response to stimuli such as exercise and certain foods, etc. As they get more experienced they would include questions about psychosocial factors, e.g. if the onset of the pain coincided with significant life events. This does not imply that the pain is solely of psychogenic origin, but acknowledges that several factors may be relevant. In the same way, if a patient complains of hearing voices the student would need to ask where the voices were heard, what was the content of the experience, when it occurred, what was the quality of the voice and so on. This would then be followed by exploring the patient's thoughts on how they might understand the voices.

Establish a timeline

As psychiatric disorders quite often have an insidious onset, establishing a timeline is vital. Patients and their relatives may confuse the chronology of illnesses, mixing up the consequences of early illness with potential causes. The most useful tool for a historian here is to constantly clarify and use fixed time markers, e.g. birthdays, annual holidays such as Christmas, to identify symptom onset and progression.

Time to talk

This is common to all histories, as the key to all effective histories is not making the patients feel they are not valued but making the patients to talk about their experiences in full. The person taking the history acts as a facilitator, directing the conversation to allow a full elucidation of patients' issues. This also enables patients to build up trust, as we will discuss later in the section 'Establishing effective working relationships with patients'.

Understand the patient

This is the whole point of the history. Without understanding the patient's experience there can be no

successful intervention. It is all too easy to jump to conclusions about what patients mean. An example might be the use of the term 'paranoid'. Laymen (and many professionals) may misuse this term to mean 'anxious', e.g. 'I'm feeling paranoid about my exams'. So, even when you think you know what a patient means you will need to clarify this to ensure that you actually do. It is also important here to consider cultural issues, for the manner in which symptoms are expressed can vary with language and culture, making history taking more complex. This is covered in more detail in the section 'Integrating cultural issues'.

Be empathic

Empathy can be difficult when those with psychiatric disorders have experiences that are so far removed from life experiences of the person taking the history. Ensuring understanding and non-judgemental responses can be difficult. An awareness of this, and a willingness to challenge one's own emotional responses and prejudices is therefore essential.

Finally, one of the most important points for any historian is to gain verification or alternative views regarding what they have learned from the patient. The best way to do this is to seek out collaborative histories from those who have witnessed the patient's presentation. This need not be restricted to relatives or the patient's partner but may include a work colleague, ambulance staff or a police officer. When the informant is emotionally involved with the patient or the consequences of the patient's behaviour, the principles of history taking outlined previously apply.

The key areas for inquiry are contained in Box 3.2.

Box 3.2 Key components of a psychiatric history

Mode of presentation

How did the patient present? For example, self-referral, referred by a professional, brought by police, etc.

Legal status

Are they informal or detained under mental health or other legislation?

History of presentation

What is the problem, what is the timeline, what events have occurred in the preceding period and subsequently?

Patient perspective

How does the patient conceptualize the problem?

Family history

Might include not only medical and psychiatric histories of family members as appropriate but also comments on their personalities, work history and relationships with the patient and others.

Personal history

This covers the development of the patient including such elements as the patient's gestation and birth and early childhood, including developmental milestones, schooling and relationships.

Work history

This covers whether the patient has been able to fulfil their potential and hold down jobs, how the patient functions at their job and relationships within that context. Factors at work that may impact on mood and anxiety may be relevant to explore.

Relationship history, including sexual history

In this part of the history the patient's relationships are explored to identify mitigating and risk factors in terms of support available or stressful relationships. It is also important to undertake a sexual history and identify if there are any issues related to sexual behaviour and/or sexuality that may impact on mental health. It is important not to let embarrassment lead to omissions of this part of the history as it can be important.

Forensic history

This explores any trouble with the law.

Current circumstances

Factors that offer support or may impact on compliance are important to identify.

(*Continued*)

Box 3.2 (*Continued*) Key components of a psychiatric history

Previous psychiatric and medical history

Collect all information about current and previous health problems including a medication history.

Use of alcohol and recreational drugs

Persistence is often required to get as accurate a picture as possible. Questions about money spent can be useful. Ensure that symptoms and signs of dependence are asked about.

Premorbid personality

This can be difficult as patients often focus on how they are in the 'present'. Asking about how they were prior to the onset of problems is useful as is asking them how others see them, not just how they see themselves.

Current medication

Make sure that both prescription and self-prescribed medication/remedies are included.

Corroborative history

This should be obtained from more than one source if possible and can include family members, friends, carers, the police and other health professionals.

Mental state examination

Examination of the mental state is one of the core skills that all doctors need to develop. It is the way in which the patient's clinical signs are explored and classified. Table 3.1 sets out the key domains of the mental state examination along with a list of some possible signs that fall under each domain. A brief explanation of each of these is given below but a full exploration of psychopathology is beyond the remit of this textbook. Video examples can be found on YouTube (www.youtube.com/user/psychiatryteacher) and at the Newcastle University website (http://mbbs-psychiatry.ncl.ac.uk/video/index2.html).

Appearance and behaviours

Observation of a patient's behaviour can reveal a great deal about a patient's mental health. Poor self-care may be present due to depression, schizophrenia, dementia, etc. In addition, depressed patients may make poor eye contact and move slowly. Conversely, a manic patient may appear to have boundless energy and have difficulty sitting still, may make flamboyant, expansive gestures and may not respect social boundaries during the interview. Alternatively, they may be irritable, hostile and/or condescending. Observations may be an important aspect of the assessment in those individuals for whom speech is limited (either expressive or receptive) or communication is difficult because of their mental state, for example younger children, those with learning disabilities, those who are psychotic and older patients with dementia.

Speech

Through observation of a patient's pattern of speech and mode of conversation, insight can be gained into underlying thought processes. An example is slow speech, which may reflect slowing of thought (e.g. in depression or frontal lobe disorders). Speech may also be slowed or hesitant secondary to distraction by internal experiences such as hallucinations, or when thoughts are so fast that the patient struggles to keep up verbally with them. Rapid speech can represent rapid thoughts, which manifest in mania and can be termed 'pressure of speech'. Here one may have difficulty interrupting the patient. Remember that when anxious anyone can talk more rapidly and pressure of speech needs to be differentiated from this normal experience.

Other abnormalities of speech include automatic repetition of a phrase (echolalia), word (pallilalia) or syllable (logoclonia) just spoken by the person speaking to the patient. These may be found in organic brain disorders and mental illnesses such as schizophrenia.

Patients may also be reluctant to speak because of other factors such as paranoia. They may also be fearful in case of reprisals, for example in cases where there is domestic violence or child abuse.

Mood

A common mistake in describing mood is to note 'subjective' mood and 'objective' mood. A much more logical way of describing mood is to talk of mood as described by the patient and as observed by the examiner, respectively. This does not add spurious value to the latter simply by labelling it 'objective'.

Table 3.1 Key aspects of a mental state examination

Areas of examination	Example signs
Appearance, behaviour and engagement with the interviewer	Impaired self-care Poor eye contact Restless/anxious Withdrawn/inappropriate
Speech	Socially appropriate or not, e.g. too loud Vocal intonation Poverty of amount Slow Pressured Perseverative
Affect and mood	Depressed Elated Incongruous Blunted
Suicidal ideation	Thoughts of suicide Plans of action
Thought form	Flight of ideas Knight's-move thinking Neologisms Echolalia
Thought content	Poverty of content Obsessional thoughts Overvalued ideas Delusions Thought alienation
Perceptions	Distortions of size, colour or intensity Illusions Hallucinations
Other	For example unusual movements such as dyskinesias or tics

Typically mood can be 'low' (depressed), 'high' (elevated, elated) or 'normal' (euthymic) but can also be rapidly changing (labile) or out of keeping with what the patient is describing, e.g. smiling when discussing the death of a loved one (incongruous). Depressed and elevated mood states are typically associated with mood disorders but abnormal mood states can occur in other conditions, particularly dementia and schizophrenia. The patient's mood may be incongruous, particularly in schizophrenia. Mood disorders may be accompanied by other concomitants of disturbed mood, e.g. anxiety symptoms in depression and irritability in mania. Lability of mood can be a sign of a rare variant of bipolar disorder, an organic disorder or borderline personality disorder.

It is vital in enquiring about a patient's mood to ask about suicidal thoughts and plans. This should be followed for all patients, and should include full exploration of any specific plans together with protective and exacerbating factors. There is a common misconception that doing this can lead to an increased risk of suicide while this is not the case.

Thought form

Abnormalities of thought form need to be clearly distinguished from abnormalities of thought content. Through careful listening to the patient's conversation, the interviewer may pick up abnormalities of form of thought, which may have diagnostic relevance. Pressure of speech, which was described above, can be associated with 'flight of ideas', when the patient skips rapidly from one thought to another that is related in terms of content, meaning or sound (rhyme). The last one is known as 'punning' and is more typical of mania. Flight of ideas needs to be differentiated from 'loosening of associations', where there is no association between successive thoughts. An example of this is 'knight's-move thinking', which at its most extreme can lead to 'word salad' (an incomprehensible jumble of words) that may have some meaning to the patient but not anyone else. Formal thought disorder of this type is highly suggestive of schizophrenia. Neologisms are literally 'new words' that have no generally recognized meaning. They tend to be either completely new or condensations of existing words and are mainly seen in those patients with schizophrenia or structural brain disease.

Thought content

Disorders of thought content include preoccupations, obsessional thoughts, overvalued ideas and delusions. A preoccupation in itself need not be pathological but can be present in mental illness, and if observed should be described. Delusions, in contrast, are defined as pathological. These are beliefs that persist despite evidence to the contrary and that are out of context with the individual's religious, cultural and educational background. Delusions can be defined as set out in Box 3.3.

Obsessions, in contrast to thought insertion (Box 3.3), are recognized as being the patient's own

VIDEO 3.1

Delusions – https://vimeo.com/13209575

Box 3.3 Classification of delusions

Delusions can be classified as primary or secondary. The latter arise from another pathological experience, for example delusions that develop to explain the experience of auditory hallucinations. In contrast, primary delusions arise de novo. This is usually difficult to be certain of. The various subtypes of primary delusion are

- Delusional perceptions in which a normal perception is suddenly interpreted in a delusional manner, e.g. a patient may see a red traffic light and immediately realize that he is the son of God.
- Sudden delusional ('autochthonous') ideas in which an idea suddenly enters the patient's consciousness like a 'brainwave', unrelated to previous real or psychic events.
- Delusional mood, which is a state of perplexity in which the patient has some sense of some inexplicable change in his environment. He senses 'something going on' which he cannot identify, but which has a peculiar significance for him. This can then be explained by a sudden delusional idea.

thoughts. They are recurrent, persistent thoughts, images or impulses that enter the patient's mind unbidden. They often involve fear of contamination or concerns that something has not been done correctly. In the early stages of suffering from these, the patient classically tries to resist the thoughts, but as time goes on they may be unable to do so. These thoughts lead to anxiety, which is often reduced by means of compulsive behaviours. Typically these relate to the nature of the thoughts, e.g. fears of contamination can lead to compulsive cleaning and hand washing. Compulsive behaviours often have a ritualistic nature and are conducted in a 'magical' manner. Obsessional thoughts and compulsive acts are most typically seen in obsessive–compulsive disorder, but can also occur in other psychiatric illnesses including schizophrenia and depression.

Overvalued ideas are thoughts that a patient believes in firmly in certain circumstances but about which the individual can be reassured when in a more neutral context. They are most commonly paranoid in nature. In some senses they fall between preoccupations and delusional thoughts. An example would be someone who strongly feels that when on a bus the other passengers are talking about them but when they are made to look at this in retrospect can accept that it was not really so. These are ideas that frequently cause anxiety to the individual.

Delusions can be further classified by their content, but particular delusions do not suggest particular diagnoses. Certain delusions are, however, more common in particular conditions. Examples include:

- Persecutory delusions where the patient believes that others are out to do them harm.
- Thought alienation, which can take the form of thought insertion, thought withdrawal or thought broadcasting. In thought insertion, the patient believes that thoughts are being put into his or her head. Conversely, in thought withdrawal, the patient believes that thoughts are being 'taken away' by an external agency. In thought broadcasting, the patient's thoughts are being passively broadcast. Although patients sometimes describe this using the term 'telepathy', it should be ascertained whether they believe their thoughts are being broadcast or actively read. The latter is a persecutory delusion rather than thought broadcasting.
- Somatic passivity phenomena are delusions of physical control, i.e. the patient believes that an external agency is controlling or manipulating his or her body or mind. This is more likely in schizophrenia.
- Nihilistic delusions (which include delusions of worthlessness, guilt and poverty) are delusions where the belief is centred on loss. At its most extreme, the patient may believe that they are actually dead. This is often associated with depression.
- Grandiose delusions are where the patient has an over-exaggerated absolute belief in their self-worth or abilities. Although classically seen in mania these also occur in schizophrenia.

Perceptions

Abnormalities of perception include distortions of real perceptions, false perceptions in the presence of a stimulus (illusions) and false perceptions in the absence of a stimulus (hallucinations). Disorders of perception include disorders of size (smaller – micropsia; larger – macropsia; lopsided – dysmegalopsia) or colour. There are various causes of abnormalities of perception, but

typically these are organic and include substance use. Note, however, that transient non-psychiatric disorders such as migraine can also cause these symptoms. Equally, illusions can occur in health and illness and on their own have little diagnostic value. They are most common when consciousness is altered (e.g. delirium), when sensory input is reduced (e.g. in the dark) or when the individual is distracted or anxious. Understanding this has particular therapeutic value as it highlights the need to nurse patients with delirium in a well-lit environment.

True hallucinations have the quality of a normal perception (and to the patient are indistinguishable from such) while arising in the absence of a stimulus. They may occur in any sensory modality (Box 3.4), but auditory hallucinations are the most common.

Box 3.4 Classification of hallucinations

Auditory hallucinations can take any form from simple, elemental sounds to complex speech or music. The most common experience of hearing voices is of second-person voices, i.e. talking to the patient. These are not associated with particular disorders, but in mood disorders will typically be mood congruent (i.e. in keeping with the patient's mood). Voices that speak the patient's thoughts aloud, give a running commentary on his or her actions, or speak about him/her in the third person are suggestive of schizophrenia. Other types of auditory hallucination can occur in organic disorders, mood disorders and schizophrenia, and sometimes in borderline personality disorder (in which they are usually experienced in internal space, do not have the quality of normal perception and are often termed 'pseudo-hallucinations').

Visual hallucinations should always raise the possibility of an organic disorder such as epilepsy, delirium, dementia or drug intoxication, but can occur in schizophrenia and mood disorders.

Somatic (or touch) hallucinations can be a result of withdrawal states (cocaine, alcohol) and include 'formication', which is the sensation of insects crawling over or under the skin. Hallucinations of joint movement (kinaesthetic) occur in benzodiazepine withdrawal. Deeper body hallucinations are often of a sexual nature and can occur in organic disorders and schizophrenia.

Olfactory (smell) and gustatory (taste) hallucinations often occur together, and should always raise the suspicion of temporal lobe epilepsy.

VIDEO 3.2

Hearing voices: a patient's perspective – https://vimeo.com/84379757

VIDEO 3.3

Hearing voices: an observer's perspective – https://vimeo.com/84392483

Other

Patients may show dyskinesias (a wide variety of movement patterns, e.g. choreoathetosis, rocking, pouting, etc., which may be related to antipsychotic medication) and stereotypies (which are repetitive, non-goal-directed movements). The latter are perhaps most commonly seen in schizophrenia.

Cognitive assessment

A description of how to perform a full cognitive assessment would require a chapter on its own and is beyond the remit of this book. Here, only general principles are set out.

It is important to realize that in testing a patient the results of a 'memory' test do not in themselves indicate the nature of a patient's memory, but merely allow the tester to infer information about how the patient's memory might be. Of the various factors that influence test performance most are outwith the tester's control, but being aware of them allows appropriate interpretation of the results and potentially minimization of them. Factors include the environment (e.g. temperature, noise level, interruptions), time of day (this is particularly relevant in mood disorders), the patient's physical state (e.g. pain, breathlessness) and their compliance with testing (e.g. wilful non-cooperation, difficulties with the test). It is important therefore to be aware of these before beginning testing.

All testing must include:

- Conscious state
- Language difficulties
- Concentration and attention
- Auditory/verbal new learning (sometimes misleadingly termed 'short-term memory')

There are a number of commonly used screening tests that can pick up on these areas. A prime example is the mini-mental state examination (MMSE). In using this, as in all tests, the examiner must be aware of the range of correct answers to the chosen tests and the range of scores in a 'normal' population, as well as how to deliver the tests in such a standardized way so that the results can be compared with these normative data. One important issue is that the examiner should not be critical and not respond to 'poor' performance by a patient, for this may also reflect other factors, such as cultural relevance of the information being tested. It is important to understand what tests actually measure. An example is the three-item word list from the MMSE, which assesses more than just memory. Performance is also influenced by factors such as language (comprehension and expression), executive function, concentration and attention. Scores are affected by age and education, and, as a consequence, false-positive results can be achieved in less-educated patients and false-negative results in the well-educated. It is worth remembering that 'absence of evidence is not evidence of absence'; a patient who performs well on a test providing information about memory (e.g. 30/30 in the MMSE assessment of auditory/verbal new learning) does not necessarily have an unimpaired memory.

Physical assessment and investigations

As discussed previously, the separation into physical and psychiatric illness is artificial at best and carries risks of missing diagnoses and the full range of a patient's problems. It is therefore important that assessment of physical health is not neglected in patients presenting with psychiatric problems. It is doubly important because patients with mental health problems are at high risk of physical health problems because of lifestyle factors (high rates of smoking and obesity, and low levels of physical activity), side effects of medication (increased appetite, sedation) and poor adherence to medication. Healthcare services and clinicians are at risk of further stigmatizing these patients by ignoring or unduly minimizing the importance of physical healthcare monitoring and providing appropriate levels of interventions.

Physical examination

All patients require a comprehensive physical examination, which in psychiatric patients does not differ materially from that required in patients presenting with any other health problem. When there are concerns about substance misuse, particular attention should be paid to possible signs of such behaviours, including needle marks and injection site abscesses.

Blood tests

A range of routine tests should be considered at first presentation. These include urea and electrolytes, random glucose concentration and full blood count. Thyroid problems are associated with mood and anxiety disorders, so thyroid function tests should be undertaken. Liver function tests (particularly γ-glutamyltransferase) along with the previously mentioned full blood count (particularly mean cell volume) can reveal harmful use of alcohol. There may be a need for more specific tests where particular disorders are suspected. In eating disorders, electrolyte balance and calcium can be affected. Tests to consider in specific clinical presentations may be syphilis serology, copper levels for Wilson's disease and testing for human immunodeficiency virus (HIV).

When patients have been on psychiatric medication, again particular tests are required. For example if they are taking antipsychotic medication, prolactin levels, plasma glucose and lipid profiles should be carried out or if taking lithium, assessment of lithium levels and renal and thyroid function is required. Use of clozapine requires regular full blood counts because of the risk of neutropenia.

Urine testing

Dipstick testing of urine has routine value. In addition, there may be cases when a urine drug screen may be useful.

Electrophysiology

Electrocardiograms (ECGs) are routine investigation because of the recognized link between psychiatric illness, treatments and cardiac risk factors. Of particular concern is corrected QT interval (QTc) prolongation with antipsychotics. Electroencephalography (EEG) has value when seizure disorders are suspected.

Imaging

Brain imaging can be used to exclude certain organic disorders but is not indicated in all cases. Functional imaging is particularly useful in patients with degenerative disorders.

Integrating cultural issues

There can be an assumption that cultural issues only need to be considered in minority and non-white patients and families. Taking this approach clearly limits the quality of care that other patients receive. Everyone, including patients, has culture, and a good history needs to incorporate relevant aspects as an integral part of the assessment.

Using culture in its broadest sense as defined, for example by the American Association of Medical Colleges may help us deliver tailored care to all patients. The definition used is:

> *Culture is defined by each person in relationship to the group or groups with whom he or she identifies. An individual's cultural identity may be based on heritage as well as individual circumstances and personal choice. Cultural identity may be affected by such factors as race, ethnicity, age, language, country of origin, acculturation, sexual orientation, gender, socioeconomic status, religious/spiritual beliefs, physical abilities, occupation, among others. These factors may impact behaviours such as communication styles, diet preferences, health beliefs, family roles, lifestyle, rituals and decision-making processes. All of these beliefs and practices, in turn can influence how patients and heath care professionals perceive health and illness and how they interact with one another.*

It has been argued that this definition is patient centred and enables patients to decide which of these factors that make up culture are relevant to them.

In delivering care to patients, doctors need to consider the following points:

- They should reflect on their own biases and prejudices to ensure that these do not consciously or subconsciously justify lesser quality for some patients.
- As many factors influence the individual's understanding of mental health and mental health services, at the outset of any assessment the patient's understanding of what they think is going to happen should be checked. Their explanation or understanding for how things are may be very useful for the interventions that are planned.
- Do not be afraid of asking if you are not sure – you just need to ensure you ask respectfully without judgement.
- Do not be intimidated into avoiding difficult questions just because someone is from a visibly different background. Do not assume that because someone superficially looks as though they may be from a similar background that this means they view the world in a similar way.
- Do not assume that because the last person from a particular background believed that mental illness is caused by spirits so will the next patient from a similar background.
- If there are a number of treatment options, do not assume which option the individual will choose. Discuss all the options and ensure the patient and/or family are able to make an informed choice. Different people will need different levels of explanations and time. Working with interpreters can increase the time needed.
- Ensure that you do get the patient's perspective. At times of distress and stress, family members may speak for the patient and make choices that they would make for themselves rather than think about what the patient might truly want.

Using interpreters

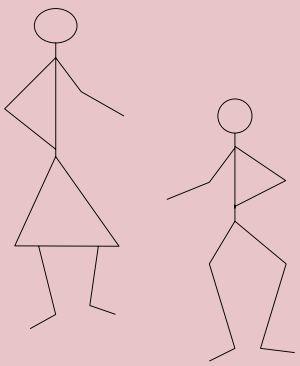
It is beyond the remit of this book to focus on this in detail. However, it is important to make sure that the interpreter is an appropriate person. It is usually not good practice to use children and/or family members as interpreters, except in emergency situations. There are issues about appropriateness of the content covered but also about confidentiality. It can also be stressful, as the presentation will be causing concern for family members too. Using staff who speak the language is preferable to using family members. It can be difficult to find trained interpreters, especially for languages that

EXERCISE 3.1

Practitioner and patient cultural influence on the healthcare encounter.

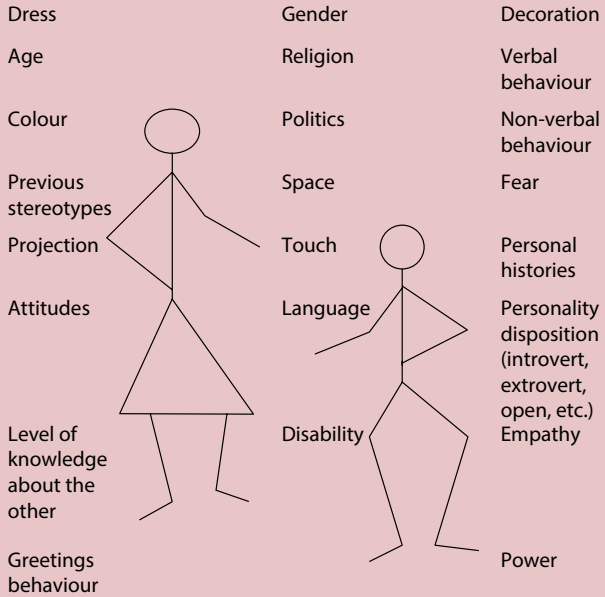
What is it about you and your patients that might influence the healthcare encounter?

Annotate the diagram below with the factors that might influence the encounter.



Exercise answer

Every single factor that makes you the person you are may influence the encounter. There may be advantages or disadvantages to this. All of the factors below and possibly more influence the encounter. You are not a neutral presence in the encounter.



The situation in which they meet.

Why are they meeting?

Is there a previous history?

are not widely spoken. There may be concerns that confidentiality will not be maintained in small communities. It also takes practice to be aware when the interpreter is interpreting what the patient is saying or when they are 'reinterpreting' what the patient has said. When using an interpreter it is important to talk to the patient and not just work through the interpreter. Remember non-verbal language is also important and you can only make sense of this if you communicate with the patient directly.

Diagnostic summary

Having collected all of this information, which may take more than one meeting and include talking with one or more informants, what should happen next? You need to write a structured summary (approximately half an A4 page) in which you identify the key presenting features, an outline history of these, relevant background factors and significant mental state findings. These are then used to construct your differential diagnosis and you should indicate which you think is most likely and why. This then should support your plan of investigation and initial management plan. New information, as it emerges, is then used to review the initial diagnosis and plan of management. This is also known as a case formulation.

Safeguarding

In all medical and psychiatric assessments, keep in mind the need to be alert for potential child or elder abuse as well as domestic violence. Abuse may be physical, psychological, financial, sexual or by neglect. Children, older people and those with learning disabilities and/or mental health disorders are at highest risk of abuse. Abusers are often well known to those being abused with more abuse within families than by strangers. Abuse occurs across all cultural, religious and social classes. Signs of abuse may include unexplained bruises, physical injuries (such as fractures), evidence of neglect or poor hygiene, weight loss or financial issues such as changed documents or money missing from accounts. Sexual abuse may come to light through self-harm presentations or increased risk-taking behaviours and/or apparent changes in behaviour and mood. Psychological abuse in most situations is much more difficult to identify.

Wherever you work there will be clear procedures for dealing with suspected abuse of both children and elders. Students and professionals need to be aware of these and their responsibilities in managing suspected abuse.

Establishing effective working relationships with patients

In this section we consider developing working relationships with patients. Our own sense of culture and our personal values strongly influence our communication styles, so it is relevant to be aware of how some of these factors can interplay with each other to impact on working relationships. We consider different stages of the clinical relationship and how these might be facilitated. We also consider some concepts, such as transference and defence mechanisms, which can influence the working relationship with patients.

Engagement

Engagement is the process by which the clinician establishes a working relationship with the patient and/or their family. Effective engagement is an important aspect of all clinical relationships but absolutely crucial in psychiatry. Although patients may tell you about their physical complaints even if they do not feel engaged with you, they are less likely to share personal details about themselves unless they feel they can trust you and that you are sensitive to their potential difficulties. This means that unless they are engaged with you, you may not get all the information you need.

EXERCISE 3.2

Before we begin, write down the factors you think are relevant when you are establishing a relationship with patients.

What aspects do you think you do well?

What aspects of your own communication style need further development?

There is much that can be done to increase the likelihood of effective engagement. Some of the suggestions below may sound as though they are basic

common sense and in some ways most of them are. However, they are often the things that students forget to do especially when under pressure or feeling uncomfortable. These are essential in psychiatry, but they are also relevant in the practice of medicine in general. Appearing warm and friendly but professional at the same time is important in gaining patient confidence. Research has shown that patients generally like doctors to be formally dressed. This suggests that although the relationships between doctors and patients are changing, with doctors less paternal than they might have been in the past, there are certain expectations that remain. Appropriate dress also extends courtesy to the patient as it reflects to some extent the pride you take in your role.

During the introduction it is important not just to say who you are, and what you intend to do, but it is equally important to check out with the patient what they think is going to happen. It is also important to explain to them at an early stage what information you are obliged to share so that no false expectations are set up.

Active listening

This is more than just listening to what someone is saying. Active listening is the process by which the listener makes the person who is talking feel comfortable and valued. The listener gives the other person the time and space to be able to say what they need to say. When you are trying to take a history and know that you need specific information, it can be very tempting to interrupt too early and make the patient feel as though you are not really listening to them or worse dismissing their views. It takes practice to sensitively bring someone back to your agenda but still be mindful of giving them the time to tell their story.

Reflecting and summarizing

Summarizing (paraphrasing) is the skill of repeating back what someone has said and is a way of checking that you have understood what has been conveyed. Reflecting is the process by which you consider what has been said and may acknowledge how the person feels. It is usually best to be speculative rather than presumptive, e.g. 'that sounds like it was a difficult time' rather than 'that must have been a difficult time'.

Question types

Open questions that encourage more than simple categorical responses are more appropriate to begin with. It is important to give patients the time and space to be able to relate their experiences, so using statements like, 'Tell me what has been happening over the last few days' may be a useful way of beginning. In psychiatry, towards the end of the history, it can be useful to use closed questions to fill any gaps in the history but rarely are such questions effective at obtaining a rich history.

Endings

Finishing a conversation takes skill. It can be particularly difficult for students as they may feel that they have taken and learnt from the interview process but not given anything in return. If the interview has been long and difficult, acknowledge that the person might be tired and thank them for their time. After the interview, it will be useful to reflect back on your performance and also pay some attention to how you felt doing the interview. This will not only help improve your interviewing skills but also help you become more comfortable tackling difficult issues.

Responses to patient histories

Some patients' stories touch us for various reasons and some stay with us. It is important to be able to deal with any issues this raises for you, even if only by chatting to your peers. There are always patients we find difficult; again think it through. The difficulties may be because we are not functioning at our best and/or the patient is difficult. If it is the latter, do not take it too personally as distress can make people less reasonable and cooperative than they might otherwise be.

At times patients may describe events or experiences that resonate personally with you for whatever reason. In these situations, it is even more essential to be aware of the issues of transference and countertransference discussed in the section 'Useful concepts to help develop therapeutic relationships'. If the interview is too difficult to continue because of this, as a student it is appropriate to terminate it or at least take a break while you collect your thoughts.

All patients have the potential to reveal histories that are painful and distressing, and as students learning

how to manage this effectively without detriment to your own health but retaining compassion is important.

Some students may find talking about sexual matters embarrassing or difficult. If this is the case it is important that they practice how to do this so they do not convey embarrassment or judgement to patients. Patients are unlikely to share their concerns about sexual issues if they sense that the professional they are talking to would prefer not to. A sexual history is an important part of relationships and needs appropriate exploration if the patient has concerns. Given some familial, societal and religious views on homosexuality, sexuality can be an important issue for some presentations of mental health problems.

Finally, our own state of mind can strongly influence the outcome of our working relationships with patients. It is consistent with good medical practice to be professional and not allow our personal issues to impact on professional roles. However, pretending that there are no personal issues is not an effective way of addressing this. Being aware of your own issues and feelings is important so you can recognize the potential impact of these when you communicate with patients and colleagues.

Other aspects to consider

Rapport

This refers to the development of a relationship where there is a sense of understanding about each other. In the professional relationship, when a rapport is established there is a sense that both parties are vested in the relationship (in the professional sense) and they are working together towards achieving a shared goal.

Boundaries

Boundaries are a way of defining your personal space and limits, both physically and emotionally. Physical boundaries are limits around physical contact. It goes without saying that physical relationships with patients are inappropriate, as they may compromise the professional relationship. Different people have different levels of comfort around physical contact from unfamiliar people. It is important to be able to offer patients comfort, but be aware that their boundaries may be different from your own. It is probably best to do no more than offer tissues or move closer if someone cries as both will demonstrate that you are attending to the distress but are not running the risk of being misinterpreted. Even taking someone's hand or touching them on the shoulder may be seen as an intrusion by some patients. You should check that it is okay to touch a patient before you do so, but that can remove the element of comfort. It is important to be very aware of the cues the patient gives you about their comfort levels regarding personal space. Experience teaches you much about when physical contact is acceptable or not.

Emotional boundaries are the limits you set around how much of your thoughts and feelings you choose to share with others. This can be more difficult as a student as you may be asked personal questions by patients but feel unable to express a preference not to discuss the issues. Any student undergoing their own personal issues (such as bereavement or relationship difficulties) may find that dealing with psychiatric patients' distress over similar issues has an exaggerated effect on their own emotional world. This may be because it becomes difficult to be clear about which feelings are your own and which belong to the patient.

Boundaries are important for your protection and also for that of patients, so think very carefully before you breach them. Patients have a right to expect that you take responsibility for ensuring that professional boundaries are maintained. They may challenge them, but it is up to you to ensure you are clear about the limits. Social engagements with patients are inappropriate while you are in any way involved in their care. Given you know sensitive information about them, relationships even after the placement may be inappropriate due to the vulnerability of the patient.

Sharing personal information

Sharing personal information may cross some emotional boundaries. It is perfectly acceptable to chat with patients about subjects other than their psychiatric histories. However, it is advisable to keep conversations non-personal. Sharing personal information in therapeutic contexts can be incredibly powerful but requires skill and experience. The outcomes may not be as expected and knowing how to deal with any fallout from sharing personal information should be anticipated. Watch carefully during

your placement to see when colleagues share personal information and how this is done. Sensitive personal information should only be used in a planned manner and not just because you are unable to contain your emotions.

Working collaboratively with patients and their families

Societal and political changes have led to increased expectations that patients now have greater say in their care and management. This applies equally in psychiatry, although psychiatry faces greater challenges than many other areas of medicine with movements (such as the anti-psychiatry movement) that contest the validity of psychiatric disorder, arguing that it is nothing more than a social construct designed to empower doctors.

Within appropriate constraints around confidentiality, in psychiatry there is often a need to collect third-party information as so much of mental illness changes the way people think and understand the world. When interviewing other informants, it is important to keep in mind that people give information that is coloured by their own experiences. This does not mean that they are being untruthful or saying that the patient is incorrect, but that their perception differs. Information about what the person was like before they became ill is information that can often only be provided by family or friends, especially if someone presents acutely unwell.

Useful concepts to help develop therapeutic relationships

Transference

Transference occurs when a person takes the perceptions and expectations of one person and projects them on to another person. Projecting is when you project your own feelings, emotions or motivations into another person without realizing your reaction is really more about you than it is about the other person.

Typically, the pattern projected on to the other person comes from a childhood relationship. This may be from an actual person, such as a parent, or an idealized figure or prototype. This transfers both power and expectation. This can have both positive and negative outcomes. In which situations and whom we place our transference on reveals and illuminates our motives and our thoughts, which would otherwise not be expressed to others or perhaps even acknowledged to ourselves. Our acts of transference may reflect what we want and what we might be trying to avoid, and our hopes and fears. What we read into other people reveals our own prejudices and unfulfilled wishes. Transference occurs on a regular basis and in all relationships, but is particularly useful as a therapeutic tool to promote understanding.

Countertransference

Countertransference is the response that is elicited in the recipient (therapist) by the other's (patient's) unconscious transference communications. The transference generated by patients may evoke responses in clinicians that they need to be aware of. Transference also provides a good idea of what the patient might be expecting from you.

Feelings are easier to identify if they are not congruent with the doctor's personality and expectation of his or her role. Doctors may struggle with transference, as often they need to feel needed and they may encourage this, and only realize the impact once a degree of dependency has been created. If they lack awareness they may react emotionally with irritation, rather than considering the role they might also have played in establishing this dynamic. Awareness of the transference–countertransference relationship allows a more considered response. Being aware of the subconscious patient agenda may help the doctor recognize some of the patient's wishes and fears, and address these openly and sensitively. It may also help explain certain behaviours from both the patient and the doctor. Understanding this also means that the doctor is able to step back and prevent themselves from feeling overwhelmed by excessive patient demand as they have greater awareness of what might be happening.

Transference happens in most relationships and not recognizing transference and countertransference can have a negative impact on the doctor–patient relationship.

Defence mechanisms

These are discussed here as they are important factors that play a part in clinical and other relationships. Although much of Sigmund Freud's work may now be considered out of vogue, except in very specific contexts, understanding defence mechanisms can really help understand human functioning. It is worth highlighting that we all use defence mechanisms and they can be effective coping strategies in the short term. However, over-reliance on them and an unwillingness to explore deeper meanings can impact on relationships and how we feel about ourselves. Freud considered that defence mechanisms are used to deal with anxiety and actions that we may find unacceptable either to ourselves or may be unacceptable socially. There was also an emphasis that at the heart of it lay sexual or violent tendencies. For the purposes of explaining them here, this emphasis is not made.

The most commonly described defence mechanisms are:

- **Denial** – The refusal to accept the reality of a situation, thought or event. An example of this can be when something that someone does not want to happen has happened, e.g. bereavement, a relationship break-up. The initial response can be a denial that the event has taken place.
- **Projection** – This is placing the feelings an individual has on someone else. So instead of acknowledging that you feel angry, you say someone else is angry because you are unable to own or manage your feelings of anger.
- **Rationalization** – This is the mechanism whereby we try and explain something logically, as the feelings we have may not be feelings we feel are acceptable or logical. An example might be saying that you were justified in being cross with a colleague as they were late for a meeting when the real reason might be that you do not like that person.
- **Sublimation** – This is when the energy from feelings about one issue is channelled into another activity. Being angry with someone at work and then using the aggression when you play sport is an example of sublimation.
- **Reaction formation** – This is expressing the opposite of what you might feel or think because to acknowledge the real feelings or thoughts may be unacceptable (either socially or to yourself).
- **Displacement** – This is placing the feelings for one person (or a situation) on to another person or situation because it may be unacceptable to have the feelings for the original person. This may be being attracted to someone who is unavailable and thereby displacing those feelings on to someone else who may be available. It is also often done when an individual is angry with someone in authority but cannot communicate that so takes out their anger on a peer or someone who is less senior.
- **Regression** – This is when someone reverts to a coping style from an earlier stage of development. A temper tantrum in an adult is a very good example of this.
- **Repression** – This is when highly stressful events that led to particular feelings are 'locked' away into our unconscious self. They may impact on our response to other situations but we are not aware of this as we do not 'recall' the event. An example might be someone who was bullied at school but repressed the recall of this. In their workplace or other relationship, they may function in such a way that anticipates they may be bullied because of the earlier events.

It is also important to note that we all use defence mechanisms and they are useful means of understanding the way we communicate our feelings to others. However, if defence mechanisms, transference and countertransference are considered together, sometimes what a person is saying is exactly what they are saying. The fact that we are unable to hear this may be because we are using particular defence mechanisms and are unable or unwilling to hear what the person is saying. A student may say they were badly taught as an explanation of poor academic performance when in fact they had failed to study effectively (rationalization, if we consider defence mechanisms). However, as a tutor it is worth noting that the student may not be rationalizing but may well have not been well taught.

EXERCISE 3.3

Having read about defence mechanisms, think about your own life in the last week or so. Denial, projection and displacement are very commonly used in everyday life. Can you think about if you used them and when?

It may also be helpful to think about when the people you have come across during this time used them or not.

What are the emotions you find most difficult to own?

How might this impact on your future practice as a doctor?

Developing your personal communication style

As a student you have opportunities to watch many different professionals at work. When you observe them, reflect on what works and what is less effective. As you develop your own skills, consider which aspects of good practice you can incorporate into your style. It is rarely effective to simply 'copy' someone else, as patients can tell whether your communication flows or not and whether it is congruent with your style. Communication is a skill that requires continued refinement even after many years in practice. Developing your own style that incorporates your personal style with good professional practice means that you will inevitably make mistakes, but this is an important learning opportunity if you leave yourself open to change. It is also useful to consider which areas you find most difficult to talk about and how this might impact on the way you communicate with patients. It is not necessarily that these issues need to be addressed, but knowing about them enables you to be a more reflective clinician.

Summary

In this chapter we have considered the stages of developing an effective working relationship with patients and some of the factors that may influence this. Communication by its very nature means that there is more than one perspective to take into account. The interview enables you to learn about the patient. Reflection and discussions with peers and staff enable you to learn about yourself. Knowing yourself is useful to help effectiveness at interviewing patients and developing productive working relationships.

To finish this chapter you may find it useful to repeat the first exercise.

Further reading

Casey PR, Kelly B (2007). *Fish's Clinical Psychopathology: Signs and Symptoms in Psychiatry*. London: Royal College of Psychiatrists.

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CHAPTER 4

Making the most of your placement

NISHA DOGRA

KEY CHAPTER FEATURES

- Preparing for the placement
- Maximizing opportunities during the placement
- What teachers expect from you and what you can expect from your teachers
- Passing your exams
- Service provision

Introduction

In this chapter we begin by considering how you can make the most of your clinical placement in psychiatry. We discuss some of the issues related to safety, stigma and fear that often accompany the placement and have been shown to be a concern among students. 'Passing your exams' is a section on passing psychiatric exams, although we are aware that few medical schools now have end of clinical block exams. The section 'Looking after your own mental health and that of colleagues' considers how you might look after your own mental health and that of your colleagues. We also provide evidence of why this is important as a student and once you are practising. We then provide a framework for mental health service provision focusing on those aspects that may influence your placement.

Preparation for the placement

Before you start your placement, it may be useful for you to reflect on your views about psychiatry and patients who have mental health problems. Before you dismiss this as typically 'touchy-feely' psychiatry, just consider recent evidence. There has always been good evidence that non-mental health professionals have negative views about patients with

mental health problems and often their views are as stigmatizing as those of the general public [the work of Jorm et al. (1997) is particularly useful in this area]. A study at one UK medical school found that students had negative views about patients with mental health problems even after their clinical placements. They also had more negative views about those with mental illness than for patients with diabetes. This suggests that clinical placements reinforced rather than challenged their views. A study led by Simon Budd at Leeds found that students agreed that they often felt uncomfortable during their placements and had difficulties communicating with patients in psychiatric services. Most students also agreed that it can be harder to deal with patients who have mental health problems, but despite this did not consider psychiatry to be stressful. However, we are aware that many students can find the psychiatry placement to be a challenging experience.

As Dixon et al. (2008) highlight, some of the concerns may be justified. However, you need to address these concerns as it is important to ensure that your care of patients with mental health problems is not poorer because of your views. Not doing so means you are unlikely to comply with the General Medical Council's *Good Medical Practice* guide.

Dixon et al. argued that although patients with mental health problems may as a group be more difficult to manage than other patients, this does not mean that all or even the majority will be difficult to manage. Indeed, in psychiatry you may learn more about managing the range of human presentations than in any other specialty and this skill will come in useful whatever area of medicine you ultimately end up practising in.

Patients with mental health problems are likely to need more time than other patients, but that should not mean that they receive a lesser quality of care. The media are also likely to fuel the stereotype that patients with mental health problems are more violent. The evidence for this being the case is more questionable, but, rather than remain fearful, it may be better to find out when this is more of a possibility so that you are effectively prepared.

In preparation, think about what your concerns or fears are and how you can address them.

Write down a list of what you hope to learn and take it to discuss with your clinical team (or consultant if there is a named individual). This will also be good practice for when you graduate and will be expected to take more responsibility for your learning. It is also worth reviewing your earlier learning, as some of the basic sciences foundation will be very useful for the clinical placement.

Psychiatry is a specialty in which your clinical skills, including communication skills, are going to be your strongest asset. There are few diagnostic tests, so you really have to use the clinical skills you have. Consider how you might develop your clinical skills and which aspects warrant particular attention.

Read any preparation material you have been sent or asked to read, and before the placement check out the practical details such as where to go and when.

On the day

- Help make a good impression by turning up on time.
- Dress appropriately – this does not necessarily mean formally. You should, however, be smart and well groomed.
- Comply with your university and hospital regulations regarding badges.
- Be sure you know what the issues are with respect to consent and confidentiality.
- Check out what's expected of you (and if you are not sure, ask).
- Be reassured by the knowledge that most of those involved in teaching you psychiatry actually do enjoy it!

During the attachment

You are likely to meet lots of people – try and remember their names. Try and look like you are interested and most clinicians are happy to help you gain the clinical exposure you need. Clinicians may at times be protective of their patients but if you are enthusiastic they are likely to try and ensure you get to see as

many patients as you can. You will impress clinicians if you take the time to be mindful of the efforts made on your behalf.

Prepare for clinical sessions by reading about the conditions you may see. In doing this you are likely to gain more from meeting the patient. You will find that the same condition can have varying impacts on people's lives. This is true for every medical condition but very well-illustrated in psychiatry. There are also great opportunities for seeing the interplay of biological, psychological and social factors in action. However, although textbooks are useful in helping you prepare, textbooks cannot substitute for real experience. It is also worth using the resources of the clinicians you see during your placements. Feel confident enough to ask them questions to help your learning. Most will welcome the opportunity to discuss their work and appreciate your enthusiasm and willingness to learn. They will appreciate that you may have had limited opportunities to see clinical psychiatry in action and will do their best to support your learning. Most clinical teachers will be busy but clinical teaching is an important part of their job and most enjoy it, so make the most of it.

It is unacceptable to access notes of people or colleagues you know personally unless you are directly involved in their care. Make yourself familiar with the relevant local protocols so that you practise safely.

Health and safety

Personal safety

There is some evidence towards a small increased risk of violence among patients who have schizophrenia. This is likely in younger men who are not treatment compliant and lack social support. If a patient is acutely psychotic, their perceptual difficulties make them more vulnerable, but can also increase the risk to others. Any time you feel concerned or unsafe or are not sure, ensure you take steps to make yourself safe. Take the time to familiarize yourself with the safety protocols in place. It is important to realize that the skills learned in assessing risk in a psychiatric setting will be useful in all clinical environments. Rates of assault on staff are higher in accident and emergency (A&E) units than in general psychiatric settings.

Paying regard to the safety of others

If a patient discloses to you that they are considering hurting someone else or themselves, you need to ensure you share this information with the clinical team. Do not assure the patient that they have confidentiality, as you are not able to guarantee this. It is a legal obligation to share information that may put another person at risk, for example, thoughts of violence towards another and potential abuse of others (especially children and other vulnerable groups). As a student, you should ensure you communicate what you have been told clearly and in a timely manner. If a patient asks you about the limits of confidentiality, be honest about this and state the parameters within which you are expected to work.

Attendance

Clinical placements rely on attendance to be able to make the most of clinical opportunities and learning. If you are finding the placement difficult, discuss this as just avoiding it does not help your learning or your future patients.

What to expect from clinical teachers

There are efforts afoot to set baseline standards for clinical teachers in psychiatry. You should reasonably expect teachers:

- To be prepared
- To make time to meet with you to discuss your placement and what you hope to achieve from it
- To provide support regarding the clinical subject by answering questions you may identify when reading about the subject
- To be obliged to report their concerns if they identify problems with your attitude or behaviour

It will help your teachers if you understand that teaching you is often one of many roles that they undertake and even the best laid plans can go awry.

Most of your clinical teachers will understand your fears and/or concerns. They would much rather you discussed them as that can help you deal with them rather than just pretending everything is okay.

Patient expectations

Most psychiatric patients are usually happy to talk with students about their illnesses and the impact of this on their lives. They often value the opportunity of having extended time to be able to share their story and help your learning. Do not take it personally if they decline to be interviewed as they may just not be well enough to do so but may be willing to do so when their health has improved. Even if they have previously consented to talking to you, it can be helpful to check again at the time of the interview. Talking to as many different patients as you can enables you to see the breadth of psychiatric disorders and also how the aetiological factors interplay to lead to similar diagnoses with different outcomes. Patients can help you see that there is a person beyond the diagnosis and that many of them when not ill have hugely varied and fulfilling lives.

It goes without saying that patients need to be approached courteously. Explain what you would need to do and ask what their expectations are. It can be useful at the outset to say what you plan to do with any information you obtain, so that the patient is aware of the parameters of confidentiality and is assured that their details are not shared beyond the clinical context. Given that it will take you some time to take a detailed history, check how much time the patient expects to spend with you and if they are happy for you to also talk to accompanying family members if relevant and appropriate. Talking to carers can provide valuable learning experiences. Some patients and/or their carers may want to recount difficult situations to ensure that as a student you learn from this so you will hopefully provide better care than they might have experienced.

Worries or problems during your placement

In the first instance, any worries or problems should be discussed with your lead consultant. If the worries are about the consultant, then share them with your personal tutor and/or the lead person who is in charge of the psychiatry programme. This may be a clinical academic or an NHS consultant with a particular role for undergraduate education.

Passing your exams

It goes without saying that being prepared increases the likelihood of passing your exams. For clinical psychiatry exams as with others, you will be examined on the process and the contents of your interviewing and examinations. Passing exams is a lot easier if you have had practice interviewing patients. The best practice opportunities will come on the wards, in day hospitals and in clinics, not through reading textbooks. Try and ensure that you balance your learning in terms of the knowledge, skills and attitudes. Patients will usually be quite happy to provide useful feedback and are well placed to say what you could have done to help them open up more or feel more comfortable.

The process

As part of good medical practice, you are expected to treat patients and colleagues with courtesy and respect. It is important that this is demonstrated in your exams. Sometimes under pressure you may fail to do fairly straightforward things that you usually do without thinking, e.g. asking the patient their name. Should you omit some questions, do not panic. Just take a deep breath and remedy the error. In objective-structured clinical examinations (OSCEs) for example, you will often have 7–10 minutes to collect quite a lot of data. Under pressure, you may not attend to the patient, as you are preoccupied with getting the information to pass the exam. Examiners will mark you down if you fail to respond to the patient in an appropriate way, e.g. if you do not follow up on their cues or do not acknowledge distress. Remember that clinical examinations are focused on assessing clinical skills rather than simply being designed for you to exhibit your knowledge.

Contents

A mental state examination should be part of all medical assessments, even if it might often only be very brief. Avoid making any judgmental or pejorative comments, as that is inappropriate.

Looking after your own mental health and that of colleagues

Your own mental health

It is often speculated that one of the reasons there are negative perceptions of those with mental health problems is that it raises concerns about our own vulnerabilities. Apart from psychosis, many of the problems experienced by people with mental health problems are in the range of 'normal' human experiences but vary in frequency and intensity. Differentiating people with mental health problems as different from ourselves may enable us to apply denial and thereby minimize our own risk.

As we discussed in Chapter 1, mental health problems are common. Doctors may not have as many of the social risk factors at play as the general public, but they may have psychological and professional factors that increase their vulnerability. It is worth noting that more than 90% of cases referred to the General Medical Council relating to the health of doctors and concerns about their practice are to do with mental health problems. Doctors and students cope with stress in different ways. It is useful at this stage for you to consider what coping strategies you use and how effectively these work. It is also important to think about how you recognize that you are becoming stressed and how you deal with this early on.

The mental health of colleagues

If you know or suspect that a colleague has mental health problems, try talking to them and encourage them to seek appropriate help. It can be helpful to keep a careful eye on them but be wary of stepping in too often to bail them out as you may just be avoiding the inevitable. If a colleague's problems (for example, substance misuse) are potentially or actually impacting on their job, you have a responsibility to report this. Avoid being tempted into misguided loyalty by keeping quiet; you will ultimately be doing your colleague and patients a disservice. By not raising concerns you may be jeopardizing patient safety and your own career. As a student, there is no need for you to carry anxieties about your colleagues as there are senior staff with whom to share concerns.

Psychiatry as a career

If you are interested in psychiatry as a career in the United Kingdom, visit the Royal College of Psychiatrists website at www.rcpsych.ac.uk and find out more. Also take the opportunity to talk with clinicians on your placement – your enthusiasm will be rewarded. For students not in the United Kingdom, the websites of national psychiatric associations will usually have information on careers in psychiatry and again talking to trainees and senior clinicians will prove invaluable.

Mental health service provision

Mental health service provision will vary across the United Kingdom and internationally. Service developments are dependent on local context (including local history, local health priorities, politics and local staff interest), national policies (where these exist, but in many countries there is little policy regarding mental health) and resources available (staff expertise as well as financial resources).

The United Kingdom has had a dramatic reduction in beds available over the last few decades as the focus has increasingly moved from inpatient care to community-based services. The support that patients actually receive in the community is highly variable.

It is most likely that the community mental health teams that you will come across are multidisciplinary in nature. Very few psychiatric services in the United Kingdom do not have such teams. Working out the roles of various members of the team can be quite confusing, so ask if you have any questions. Most teams will have a psychiatrist, community psychiatry nurses (who have trained in mental health but may also have general nursing qualifications), nursing assistants (who take on some nursing duties but have not completed a nursing qualification), occupational therapists, psychologists (in some areas the psychology service will be independent of the psychiatric service), social workers (it may vary whether these are an integral part of the team or work alongside it dependent on local arrangements), specific therapists who may have a range of different professional backgrounds and administrative support.

Over the last decade or so there has been a proliferation of specialist teams such as crisis intervention, early intervention services for psychosis, assertive outreach and deliberate self-harm teams. This will vary locally and it is worth discussing with patients and clinical staff how such teams have influenced service delivery and impacted on factors such as continuity of care.

Summary

In summary, as with any other placement, you will get more out of the placement if you prepare effectively for it. The psychiatry placement may be more challenging than other placements, especially if there are areas you have anxieties about. Being willing to acknowledge and address these difficulties will mean that whatever your future specialty, you will acquire skills that will benefit your patients and ensure high-quality care.

Further reading

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Mental health legislation

CHAPTER 5

BRIAN LUNN

KEY CHAPTER FEATURES

- An introduction to the principles of mental health legislation
- An overview of compulsion in the treatment of mental illness
- An overview of capacity in mental illness
- Common principles in mental health legislation
- A brief overview of the English and Welsh Mental Health Act (2007)

Introduction

Some of the most contentious and complex issues in psychiatry are those surrounding the compulsory detention and treatment of patients with mental disorders for assessment and treatment. These issues raise important questions such as 'To what extent should a person suffering mental disorder be held responsible for their behaviour (e.g. is it fair to punish a man who smashes a window in response to "voices" over which he has no control as they are part of a mental illness)?' If the individual is not fully responsible then should their freedom be limited 'for their own good' (i.e. should the same man be taken to hospital against his will if he cannot be held accountable for his actions)?

The law must provide practical responses to this type of question and has to balance the needs of the individual with the needs of the community. So, for example, if through a mental illness the man represents a risk to himself or others, is it reasonable to detain him for his protection and/or the protection of others? Before we consider these issues, it is worth highlighting that the definition of what is a mental disorder or illness is contentious. Historically, there have been models of illness used throughout the world that today seem at best bizarre and at worst prejudiced and oppressive, whether used for political reasons or because they were based on what was understood and believed at that time. The importance of defining mental illness within a legal framework is that it protects people regarded as 'odd' or a 'nuisance'

from inappropriate and unnecessary compulsion. People should surely not be considered mentally disordered simply on grounds of 'immoral' conduct, sexual choices or dependence on alcohol or drugs. It is important to remember that psychiatric diagnoses do not necessarily correspond to legal definitions!

Before reading further it is worth considering:

- Are there circumstances in which compulsory admission/treatment are justified?
- If so, when might this be appropriate?
- If not, what are the alternatives?
- Should mental illnesses be treated differently from other illnesses?
- If so, what would be the justification?
- Who should decide whether compulsory detention is indicated?
- How can the individual's rights be protected even if he or she is detained?

Why is this important for you?

It is likely that only some of you reading this will become psychiatrists. So, why is it important that you know about mental health legislation? It is likely that even in your foundation years you will come across patients with mental disorders and/or loss of capacity. Understanding the principles of the relevant legislation is therefore important for all doctors as there are parts of this that all doctors may need to use. It is also possible that you will be in a position where you will be treating patients already detained under the Mental Health Act (MHA). It is not only psychiatrists who may use the MHA; any registered doctor may be involved in MHA assessments. In this case understanding the limits of the act is important.

Case studies

Consider each of the four case histories below and consider how you think the patient should be managed. For each case, it might be helpful to consider the justification for your decision. They will be revisited at the end of the chapter with some comments about how they might be managed within current legislation.

CASE STUDY 5.1

A 47-year-old single woman with multiple sclerosis is referred to local mental health services. Her mother is concerned that her daughter has voiced an intention to commit suicide. The patient, having been brought up in a very religious household, rejected her family's religious beliefs in her late teens when she left home to go to university. She and her parents had been estranged until a few years ago when her father had a serious myocardial infarction (MI). Six months after their reunion her father died from a second MI.

Just after university, the patient developed blurred vision. It was on presenting for investigation that her multiple sclerosis was diagnosed. Her illness has followed a relapsing/remitting course, but over the last 5 years the decline has been steadily accelerating. Currently, she is just recovering from another relapse and now needs to look at moving into living in a more supported environment. Her mother has suggested she moves home.

The patient has always said that when she lost her independence she would choose to end her life rather than live beholden to others. She has recently discussed this with her younger sister who revealed her confidence to their mother. Suicide is an anathema to the rest of the patient's family. They consider her to be depressed given that she is having such thoughts. As such, they have asked that the patient be assessed and detained for treatment if she cannot be persuaded that suicide is wrong.

CASE STUDY 5.2

A 23-year-old man is stopped by the police as he was noted to be wandering the streets muttering to himself. On stopping him, the police found that he was carrying a knife. He is being assessed in the cells by a duty psychiatrist following referral by the police surgeon.

The young man's flatmates describe a gradual pattern of deterioration in functioning that they feel might be related to recreational drug use. The young man talks of his fears of a vague group of 'officials' who are monitoring him and he feels that they will ultimately try to make him a victim of 'extraordinary rendition', taking him away to Pakistan to be tortured. He has told the police that he is carrying a knife for his own 'protection'.

CASE STUDY 5.3

A 38-year-old musician with bipolar affective disorder is in hospital nearing the end of his stay. His admission was compulsory as he had developed another manic episode that had resulted in him running up significant debts and displaying increasingly disinhibited behaviour, culminating in him running down the middle of the main shopping street naked apart from a 'Santa' hat.

He is grateful that he was detained and admitted and is now euthymic. He has had successful spells at home and is ready for discharge but has stated that although he believes medication helped with the acute phase of his illness he intends stopping everything within 3 months of discharge, as he believes that lithium, which he is currently on, blunts his creativity. He no longer requires inpatient treatment.

CASE STUDY 5.4

A 55-year-old man with recurrent major depression is found in his home by his community psychiatry nurse (CPN) in a state of squalor. He is emaciated and in poor physical health. Assessment reveals that he has a severe depressive illness with psychotic features, including profound nihilistic delusions, e.g. the belief that his internal organs have rotted away. He refuses to eat or drink and pulls out any intravenous or nasogastric tube inserted. He will not take medication. In line with treatment protocols, it is felt by his treating team that the most appropriate treatment is electro-convulsive therapy (ECT). His family are supportive of this recommendation, but the patient refuses saying that it is pointless as he is already dead.

Compulsory treatment

The whole area of compulsion in health is a contentious one. There is not even commonality in the legislation between the various legal jurisdictions in the United Kingdom. Over the past few years significant changes in legislation have taken place in Scotland and England and Wales. At the time of going to press, proposals for new legislation in Northern Ireland are being consulted upon.

It is beyond the remit of this chapter to consider all the complexities of the various pieces of legislation in the United Kingdom even if they were not changing. Instead, it is intended to introduce some basic principles and then illustrate one approach by describing the various elements of the MHA as currently enacted in England and Wales.

Consent

It is a fundamental principle of ethical medical practice that patients are only treated with their consent. There are, however, some circumstances where this principle cannot be easily adhered to, for example if the patient lacks capacity because they are unable to communicate their wishes or understand what treatment is being offered. The key elements of capacity to be able to consent are that:

- The patient understands the information presented to them.
- The patient can retain the information.
- The patient can weigh the information in the balance and come to a decision based on this.
- The patient can communicate their wishes (note that this can be through any means and not just verbally).

If the patient cannot fulfil the above criteria, their capacity to give consent is questionable. It is important to remember that the default position under the Mental Capacity Act (MCA) (2005) is that all adults (i.e. people aged 16 years and older) are assumed to have the capacity to make their own decisions, to consent to or refuse treatment unless there is evidence to the contrary. Obviously issues of consent are relevant in all areas of medicine, but as this book focuses on mental health that is where discussion of principles will be focused here.

The interrelationship between the MHA and the MCA is complex and is currently being tested by case law. Most recently, judgement has been delivered emphasizing that when there is the possibility to invoke both acts, the MHA has priority. Capacity is more likely to be regained if the mental disorder is effectively treated. Additionally, the MHA has greater safeguards than the Capacity Act.

Compulsion

There are three key areas where patients with mental health problems may find themselves being considered for compulsory treatment. These are the following:

- When they present an actual or potential risk to others as a result of a mental disorder (e.g. if they are having auditory hallucinations telling them to harm someone; if they are paranoid and believe others are out to harm them whether they are or not).
- When the mental disorder with which they present results in them posing an actual or potential risk to themselves (e.g. suicide, harm from impulsive behaviours (such as overspending in a manic illness) or self-neglect). Hopelessness in postnatal depression can present significant risks to babies as severely depressed mothers may believe that it is kinder to kill the child than for them to be in a bleak and hopeless situation.
- When the risk of deteriorating mental health results in the potential for either of the above two situations developing.

Now of course it is possible that an individual may meet one or more of these three criteria and still have legal capacity, and it is in such circumstances that compulsion is most controversial. In a 1999 Department of Health review, an expert committee chaired by Professor Genevra Richardson highlighted this inequality and suggested it be addressed along with strengthening of the rights of patients to act autonomously in any new mental health legislation. However, agreement around a new mental health bill incorporating all of these complex issues together could not be reached. Many of the principles were adopted in a new Scottish legislation (Mental Health [Care and Treatment] [Scotland] Act [2003]) and into a 2007 amendment of the 1983 MHA of England and Wales. Important changes include a broader definition of mental disorder; it is now defined as any disorder or disability of mind. Previously exempted were 'immorality, promiscuity and sexual deviancy' but now the only exemption is 'dependence on alcohol or drugs'. Additionally, patients with a personality disorder were required to have a 'treatable' disorder to be detained. The requirement of treatability has now been removed. This however raises questions regarding what the functions of detention are. Should doctors be involved in detaining patients for whom there are limited effective treatments and where the main justification is to detain them to limit their freedom so they cannot harm others? Or should this be a non-medical and purely legal decision? Also, how reasonable is it to detain someone on the possibility that they may harm others?

Common principles

Across the various legal jurisdictions, a variety of principles hold true. These include the following:

- **More than one person makes the decisions:** Any decision about a detention of any duration, or treatment, is not taken by one person alone and there should be no conflict of interests for those making the decisions.
- **The period of detention is defined:** Periods of detention are not 'open-ended'. That is patients can only be detained for a set period and if extension of that period is sought, then an independent review is required.
- **Right to appeal is granted:** Patients once detained have the right to appeal to an independent authority which has the right to discharge the patient from their compulsory admission.
- **Discharge is when appropriate:** Once detained that period of detention should be terminated when the patient no longer meets the legal conditions justifying detention, i.e. compulsory treatment ends when the patient has recovered sufficiently.
- **Change from voluntary to detained status:** A patient who is admitted to hospital voluntarily can be detained if they withdraw their consent for the admission or treatment and their condition has deteriorated such that they meet the criteria required for detention.
- **Police responsibilities:** Police on coming into contact with a person who appears to be mentally ill are required to make suitable assessment and treatment a priority whether or not an offence has taken place.

- **Consent when detained:** Patients while detained should still be approached for their consent, and treatment plans should, where possible, be agreed upon rather than just imposed.
- **Offenders:** Those convicted of offences, no matter how serious, have the same right to treatment as non-offending patients. Most mental health legislations have separate, but usually overlapping, systems for offenders.

Having read the above discussion how does this influence (if at all) your original answers to the questions posed above?

Summary of Mental Health Act (2007), England and Wales

Who can detain patients?

One of the fundamental changes introduced by the recent MHA amendments was that mental health professionals other than doctors can now take on the responsibility for managing detained patients. The detention of a patient requires the involvement of more than one doctor and involvement of another professional for the majority of detention orders. For orders lasting longer than 72 hours (Sections 2 and 3) the requirement is for two doctors to make the recommendation and for an approved mental health professional (AMHP) to make the application. One of the two doctors must be one who is recognized by the Secretary of State as having expertise in the diagnosis and management of mental disorders (known as a Section 12 approved doctor). The other is typically a registered doctor who knows the patient (most often the patient's general practitioner (GP) but when they are unavailable, a second Section 12 approved doctor can provide a recommendation, so long as they are not in a relationship (financial, personal or in a subordinate role) with the first doctor. The AMHP is most commonly a specially trained social worker.

The exceptions to the above rules about the make-up of the assessing 'team' are the emergency Sections 4 and 5. In the case of Section 4, there can be one of the two doctors who would normally make up the assessing team and an AMHP. In the case of a Section 5(2) there only needs to be the doctor in charge of the patient's care or their nominated deputy. This means that on non-psychiatric wards the patient can be detained only by their consultant or someone nominated by them from within the same employing organization which may mean junior doctors attached to non-psychiatric teams when on call. This means that a psychiatrist employed by a different trust cannot be called to detain a patient in a general medical ward on a 5(2). Finally a Section 5(4) is applied by a registered mental nurse.

The sections

- **Section 2:** An assessment order allowing compulsory detention for up to 28 days that can be used either in the community or in hospitals.
- **Section 3:** A treatment order allowing detention for up to 6 months that can be used either in the community or in hospitals.
- **Section 4:** An emergency order to admit a patient to hospital from the community for up to 72 hours if waiting for the second doctor to complete a Section 2 or 3 would result in a delay that might cause problems for the patient or others by perhaps exposing them to risks related to possible mental illness.
- **Section 5(2):** An order that allows detention for up to 72 hours of inpatients. This section applies to hospital inpatients and authorizes the detention of a voluntary patient for up to 72 hours allowing an assessment, which may lead to an application for admission under Section 2 or 3 if necessary.
- **Section 5(4):** An order that allows detention of a patient in a psychiatric unit for up to 6 hours to enable them to be assessed for detention by a doctor. Typically, this means that the next section considered would be a 5(2).
- **Section 135:** An order that allows the police to force entry to allow an MHA assessment of an individual believed to be mentally ill.
- **Section 136:** An order allowing the police to remove a person believed to be mentally ill from a public place to a 'Place of Safety' to allow an MHA assessment to take place.

Supervised Community Treatment: This is administered by a Community Treatment Order. The aim is to allow some patients to live in the community while still

subject to powers of the MHA. These patients remain under compulsion and can be recalled to hospital for treatment. Only those patients who have previously been detained for treatment (see Section 3 above) are eligible for a Supervised Community Treatment Order.

Patients involved in criminal proceedings

The MHA allows courts to deal with mentally disordered offenders. Whereas the sections applying to those who appear in the courts still require medical recommendations, the decision whether the prisoner 'deserves' punishment or treatment is for the court.

There are seven sections relating to offenders. The two most important are as follows:

1. **Section 37:** This allows a person convicted of an imprisonable offence to be detained and treated in hospital. The patient is discharged when well, regardless of the length of prison sentence they may have been given if they had not been detained in hospital.
2. **Section 41:** This additional section may be applied to patients regarded as 'dangerous' and who are detained under Section 37. It restricts the discharge of these patients by requiring permission for their discharge from the Home Secretary once they are deemed ready for discharge by the hospital.

Some criminal laws also involve psychiatric reports, for example the verdict of 'not guilty by reason of insanity' or 'unfit to plead' (person too mentally disordered to know right from wrong) results in the individual's detention as if under Section 37 with Section 41 restrictions. The verdict of 'diminished responsibility' can reduce a murder verdict to one of manslaughter.

Vulnerable groups

For young people under 16 years of age, parental consent may be sufficient to detain their children if they fulfil the criteria above. However, it is considered a better practice to use the MHA in some contexts given the safeguards that fall into place for detained patients if the act is implemented.

Individuals with a learning disability may not be able to understand or retain information, in which case it is unlikely they will have capacity to consent to treatment.

Treatment

The MHA applies only to treatment of mental disorder and cannot be used to impose treatment of physical conditions. Unconscious patients are treated under authority of Common Law, which recognizes a duty to save life etc. (Doctrine of Necessity). Restraint of a potentially violent or suicidal voluntary patient, whether in a general medical or a psychiatric ward, would be justified on the same basis. For less urgent cases where the patient lacks capacity the MCA applies.

There are several key principles involved in treating mentally ill patients for their mental illness, which are as follows:

- Voluntary patients cannot be treated for a psychiatric disorder without informed consent.
- Patients detained under Sections 4, 5(2), 135, 136 (72-hour sections) and guardianship cannot be treated without informed consent except in emergencies when the principles outlined above apply.
- Patients detained under Sections 2, 3 and 37 may be given some treatments without consent under the conditions detailed below.

General Treatments

There are no conditions applied to general care and treatments, except as outlined below. Nursing care, occupational therapy and other care may be given to detained patients without consent.

Medication and ECT

Consent or a second opinion is required. To be given ECT or prolonged medication, i.e. for more than 3 months, either the patient must give a consent certified valid by the consultant (remember their wish to leave hospital is considered invalid!) or agreement to the treatment plan must be obtained from a second doctor appointed by the MHA Commission.

Urgent Treatment

If urgent, a course of ECT or medication may be started with a detained patient while waiting for the second opinion.

Irreversible Treatments

This includes treatments such as neurosurgery for psychiatric disorders and hormone implants. Here, consent *and* a second opinion are required. For these treatments, the second-opinion appointed doctor must agree with the plan and the patient must give consent which is considered valid by a three-person panel.

Capacity

Capacity is a person's ability to make their own decisions. In April 2007, the Mental Capacity Act (2005) came into force in England and Wales. Prior to this, decisions on behalf of adults who lacked capacity were made under common law. This act is underpinned by five guiding principles. The principles are set out in Box 5.1.

In assessing capacity, the assessment can only refer to a single decision at a particular moment in time. Assessments have value only when the individual's 'mind or brain' is affected by illness or disability. Capacity can fluctuate. This is an important point as waiting and revisiting a decision may allow an individual to recover capacity and make a decision on their own behalf.

The act sets out two questions that the assessor needs to answer. They are as follows:

1. Is there an impairment of or disturbance in the functioning of a person's mind or brain? If so,—
2. Is the impairment or disturbance sufficient that the person lacks the capacity to make a particular decision?

In defining capacity, the act sets out four things that the individual needs to be able to do to be said to have capacity. These are set out in Box 5.2. The decision is made on the balance of probabilities, i.e. is it more likely than not that the individual lacks capacity? With respect to people with mental disorders, the MCA is used where an individual lacking capacity is not objecting to the treatment. In this respect, the MCA provides a legal framework that supports the provision of care to a person who lacks the mental capacity to deal with their own care or treatment. The decisions made must be made in the patient's 'best interest' (see the fourth principle in Box 5.1). This was introduced as an amendment to the MHA in 2007 and implemented in 2009. The additions are known as 'deprivation of liberty safeguards'.

Box 5.1 The five guiding principles of the MCA

- An assumption of capacity.
- Supporting people to make their own decisions.
- People have the right to make eccentric or unwise decisions.
- Where someone lacks capacity one must act in the person's best interests.
- Where someone lacks capacity any action taken on their behalf must generally be the least restrictive option.

Box 5.2 An individual is unable to make their own decision if they cannot do one or more of the following four things

- Understand information given to them
- Retain that information long enough to be able to make the decision
- Weigh up the information available to make the decision
- Communicate their decision – this could be by talking, using sign language or even simple muscle movements such as blinking an eye or squeezing a hand

Advance decisions

The MCA makes particular reference to advance decisions. These are when an individual sets out what types of treatment he or she does not want to be given if they ever lose the capacity to decide on their treatment. Such advance decisions are legally binding and must be followed by doctors and other health professionals as long as they meet certain conditions as those below:

- They can only be made by an individual who is aged 18 years or over and who has capacity to make it. There are more stringent conditions when the treatment is 'life sustaining'.

- They are not valid if the individual withdraws it or if they subsequently act in a way that is clearly inconsistent with it.

Decisions only come into effect if there comes a point where the individual making the decision loses capacity in relation to the decision(s) in question.

There are two important points to note about advance decisions, as follows:

1. A decision cannot specify a particular treatment to be given in an advance decision. Decisions can only cover treatments they would wish not to be given.
2. A decision does not permit individuals to specify that they should not be detained or treated against their wishes under the MHA.

Revised code of practice

Further changes that reinforce the importance of the Human Rights Act and the importance of capacity were implemented in a Revised Code of Practice to the MHA in 2015. The guiding principles are set out in Box 5.3.

Summary

This chapter has outlined the principles behind mental health legislation, although it should be clear that this is in no way as straightforward as it may initially sound. Although the detail varies within the United Kingdom and also elsewhere, the guiding principles are usually similar and aim to protect both patients who have mental illness and the community if there is risk from such patients. The key points to remember are when the MHA can be applied and when it cannot. If there is no evidence or high suspicion that a patient has a mental disorder, the MHA cannot be used. When the MHA is applied there are certain safeguards in place to ensure that patients' rights are protected.

Further reading

Code of Practice: Mental Health Act 1983 (2015). Easy read version. Available at https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/421541/MHA_Code_EasyRead.pdf (Accessed August 2015).

Mental Capacity Act (England and Wales) (2005). Available at http://www.legislation.gov.uk/ukpga/2007/12/contents (Accessed August 2015).

Box 5.3 Guiding principles as applied to the MHA

- Least restrictive option and maximizing independence: Where possible, patients should be treated without recourse to the act.
- Restrictions that apply to all patients should be avoided: While some restrictions, such as no smoking, might be appropriate for the health and safety of others, restrictions that are for the convenience of the provider, e.g. not allowing use of mobile phones, are not permitted. However, the use of a phone might be restricted for someone who is at risk of using it to 'stalk someone'.
- Empowerment and involvement: The views of patients and where appropriate, their families, carers and others should be taken into account when decisions are made. Patients should be encouraged to make advance decisions.
- Respect and dignity.
- Purpose and effectiveness: Treatment decisions should have clear therapeutic aims and be evidence based.
- Physical healthcare needs: These should be assessed and addressed.
- Efficiency and equity: All relevant parties must ensure that high quality mental health services that give equal priority to physical health and social care are provided.
- All decisions must be lawful and informed by good professional practice. Lawfulness necessarily includes compliance with the Human Rights Act 1998 and Equality Act 2010.

Mental Health Act (England and Wales) (2007). Available at http://www.legislation.gov.uk/ukpga/2007/12/contents (Accessed August 2015).

Mental Health Act (Scotland) (2015). Available at http://www.legislation.gov.uk/asp/2015/9/contents/enacted (Accessed April 2015).

CASE STUDY 5.1

A 47-year-old single woman with multiple sclerosis is referred to local mental health services by her mother. She is concerned that her daughter has voiced an intention to commit suicide. The patient, having been brought up in a very religious household, rejected her family's religious beliefs in her late teens when she left home to go to university. She and her parents have been estranged until a few years ago when her father had a serious MI. Six months after their reunion her father died from a second MI.

Just after university, the patient developed blurred vision. It was on presenting for investigation that her multiple sclerosis was diagnosed. Her illness has followed a relapsing/remitting course, but over the last 5 years the decline has been steadily accelerating. Currently, she is just recovering from another relapse and now needs to look at moving into living in a more supported environment. Her mother has suggested she moves home.

The patient has always said that when she lost her independence she would choose to end her life rather than live beholden to others. She has recently discussed this with her younger sister who revealed her confidence to their mother. Suicide is an anathema to the rest of the patient's family. They consider her to be depressed given that she is having such thoughts. As such, they have asked that the patient be assessed and detained for treatment if she cannot be persuaded that suicide is wrong.

Possible Outcome

The patient if found, as is probable, to still have capacity to make such decisions, and if there is no evidence of a mental illness such as a mood disorder, is not liable to be detained.

CASE STUDY 5.2

A 23-year-old man is stopped by the police as he was noted to be wandering the streets muttering to himself. On stopping him, the police found that he was carrying a knife. He is being assessed in the cells by a duty psychiatrist following referral by the police surgeon.

The young man's flatmates describe a gradual pattern of deterioration in functioning that they feel might be related to recreational drug use. The young man talks of his fears of a vague group of 'officials' who are monitoring him and he feels that they will ultimately try to make him a victim of 'extraordinary rendition', taking him away to Pakistan to be tortured. He has told the police that he is carrying a knife for his own 'protection'.

Possible Outcome

This patient appears to be suffering from a paranoid psychosis. This is a new presentation and it is unclear whether it is due to intoxication with drugs, a drug-induced psychosis or a more chronic illness such as schizophrenia (see Chapter 9). He is therefore liable to be detained for assessment, i.e. a Section 2 as described above. Criminal proceedings are unlikely.

CASE STUDY 5.3

A 38-year-old musician with bipolar affective disorder is in hospital nearing the end of his stay. His admission was compulsory as he had developed another manic episode that had resulted in him running up significant debts and displaying increasingly disinhibited behaviour, culminating in him running down the middle of the main shopping street naked apart from a 'Santa' hat.

He is grateful that he was detained and admitted and is now euthymic. He has had successful spells at home and is ready for discharge but has stated that although he believes medication helped with the acute phase of his illness he intends stopping everything within 3 months of discharge, as he believes that lithium, which he is currently on, blunts his creativity. He no longer requires inpatient treatment.

Possible Outcome

If this was a repeated pattern with the patient being admitted regularly following discontinuation of medication, the clinical team may seek to impose a Community Treatment Order with conditions around his use of medication, accepting monitoring of his mental state and investigations required for mood stabilizers (see Chapter 16). If, however, this was early in his illness it is likely that he would be discharged with an attempt to negotiate a plan involving close monitoring of his mental state and a negotiated phased withdrawal of medication.

CASE STUDY 5.4

A 55-year-old man with recurrent major depression is found in his home by his CPN in a state of squalor. He is emaciated and in poor physical health. Assessment reveals that he has a severe depressive illness with psychotic features, including profound nihilistic delusions, e.g. the belief that his internal organs have rotted away. He refuses to eat or drink and pulls out any intravenous or nasogastric tube inserted. He will not take medication. In line with treatment protocols, it is felt by his treating team that the most appropriate treatment is ECT. His family are supportive of this recommendation, but the patient refuses saying that it is pointless as he is already dead.

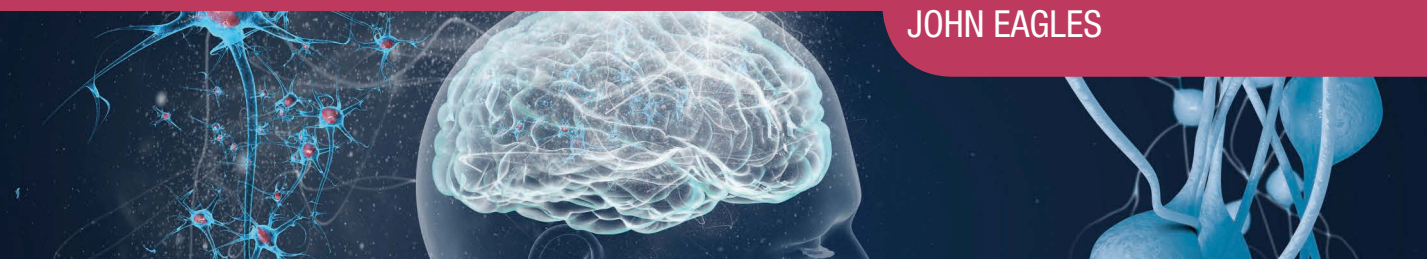
Possible Outcome

As this is a presentation of a patient with a recurrent illness needing admission for treatment he is liable to be detained under a treatment order (Section 3). His illness is life threatening and so ECT is a logical and potentially life-saving treatment. A second-opinion approved doctor's opinion would be sought for this. If there was no chance to wait for this emergency treatment Section 62 might be used.

Mood disorders

CHAPTER 6

JOHN EAGLES



KEY CHAPTER FEATURES

- Depression: history, prevalence, risk factors, clinical presentations, assessment and management
- Depression in older patients
- Bipolar affective disorder: history, prevalence, risk factors, clinical presentations, assessment and management
- Seasonal affective disorder
- Childbirth and affective disorders

Introduction

It is very important for all doctors to be competent in the diagnosis and management of mood disorders. Although patients with bipolar affective disorders will be managed predominantly by psychiatrists and general practitioners (GPs), depression will be seen by all doctors who work with conscious patients. This chapter will deal first with depression, then with bipolar affective disorders and will conclude with brief accounts of other mood disorders. Childhood mood disorders are covered in Chapter 14.

Why is this relevant to you?

Mood disorders (most notably depression, which the World Health Organization (WHO) now cites as the leading cause of disability worldwide) are very common and will be seen frequently by nearly all doctors but are often missed. When a patient is depressed, the quality of their life is usually markedly impaired. Depression often goes unrecognized, and untreated, and so the opportunity may be lost to effect a very significant improvement in the quality of that patient's life.

Depression

History

The term 'melancholia', commonly used until the early twentieth century and still used occasionally to describe severe depression, dates back to ancient Greece, when it was believed to arise due to the accumulation of black (melan) bile (chole). Depression has been diagnosed retrospectively in many historical figures, including Kings Saul and David (from the text of the *Bible*), Rembrandt, Mozart and Abraham Lincoln. For thousands of years, opinions varied as to whether melancholia was essentially a religious or a medical affliction, and it was only in the nineteenth century that a medical model of depression began to hold sway. Some would assert that this process has now extended too far into the 'medicalization of unhappiness' and this possibility will be discussed in the section 'Are doctors prescribing too many antidepressants'. Depression of clinical significance is now termed depressive disorder.

Prevalence

It is difficult to provide definitive rates for the prevalence or incidence of depression for two main reasons. First, as in the diagnosis of hypertension or diabetes, many depressive symptoms exhibit a continuous variation across the population, giving rise to difficulties in knowing where to draw the line between distress and unhappiness on one hand and mild depressive illness on the other. Second, the results of population screening studies will reflect the accuracy of the screening questionnaire deployed, the skills of the interviewers, especially where symptoms are more subtle and are observer rated, and the proportion of the community who agree to participate.

Despite these caveats, enough good studies have been conducted to yield reliable data on prevalence rates of depression. For example, Weissman et al. (1996) deployed tight criteria for depression in 10 countries, finding overall lifetime prevalence rates of 9% and annual incidence rates of 4%. There were wide international differences in lifetime rates ranging from 1.5% in Taiwan, through 9% in Germany to 19% in Beirut. In 2001, Ayuso-Mateos and coworkers screened both urban and rural populations in five European countries, using more inclusive criteria for depression. They found an overall point prevalence rate of 8.6% for depressive disorders. Again, there were marked international differences, which may partly reflect psychosocial disadvantage, ranging from a rate of 2.6% in Santander, Spain, to 17.1% in Liverpool. As Hidaka (2012) has discussed, modern lifestyles may be contributing to an increasing prevalence of depression.

These figures confirm that depression is a common condition in many societies, it causes much suffering (as discussed in Chapter 1) and it constitutes a hugely important challenge to the medical profession.

Risk factors for depression

The main 'risk factors' for depression are summarized in Table 6.1. It is important to note that although these factors are associated with depression, and may often act as predisposing factors, they are not necessarily causative. For example, although depression may well result from the break-up of a marriage, pre-existing depression may have contributed materially to the separation in the first instance. Milder cases of depression may be related to psychosocial factors alone, whereas more severe cases are likely to have a greater biological component. Also, some individuals may develop severe depression in the absence of any social problems (although these may occur secondary to the illness) but as discussed below, in the section 'Biological factors' it can be difficult to separate out environmental from non-environmental factors.

Sociodemographic factors

Rates of depression in childhood exhibit no gender difference, but from puberty onwards, rates in females begin to predominate, outnumbering males during their reproductive years by at least two to one, with rates tending to equalize again among older adults. Possible reasons for this female preponderance may include factors associated with pregnancy and childbirth (leading to postnatal onsets that do not occur in males), lower self-esteem coupled with self-blaming cognitive styles and more difficult sociocultural roles (which may also include an increased vulnerability to adverse life events). Widowed, divorced and separated people have higher rates of depression, but marriage seems to be more protective for men than it is for women. Single men are more likely to be depressed than married men, but several studies have found that single women have lower

rates than married women. People in lower socio-economic groups suffer something of a 'double whammy' in that not only do they experience higher rates of depression but their outcomes are less good. This probably relates to a combination of later recognition or non-recognition, being subject to more perpetuating factors, lesser likelihood of receiving appropriate treatment and lower levels of treatment adherence.

Psychosocial factors

It can be helpful to think in terms of distant and current adverse psychosocial factors. Among distant factors, there is good evidence that the death of one's mother before the age of 11 years increases the likelihood of future depression. As a corollary, any event that adversely affects parent–child emotional bonds is likely to be a risk factor, for example parental separation or dysfunctional parenting. Such factors are difficult to disentangle from the effects of childhood abuse (sexual, physical or emotional), which are also more likely to occur when family function is abnormal or parenting is poor.

Of current and recent factors, adverse life events are of clear importance in precipitating depression. The magnitude of the effect of life events ranges downwards in severity from the death of a spouse, and many of these events are linked by the common theme of loss; this could be loss of relationships (through

Table 6.1 Factors associated with an increased risk of depression

Sociodemographic Factors	**Physical Illnesses**
Female gender	Viral infections
Adult age groups	HIV/AIDS
Marital status (see text)	Cancer
Unemployment	Diabetes
Lower socio-economic status	Cardiovascular disease
Urban area of residence	Stroke
Born after 1950	Epilepsy
Biological Factors	Multiple sclerosis
Genetic predisposition	Asthma
Low birth weight	Chronic obstructive lung disease
Psychosocial Factors	Arthritis
Early loss of mother	Cystic fibrosis
Childhood sexual abuse	**Prescribed Medication**
Bereavement	Steroids
Other adverse life events	Calcium channel blockers
Lack of a confidant(e)	Beta-blockers
Social isolation	Digoxin
Psychiatric Co-morbidity	Opiates
Alcohol misuse	**Miscellaneous**
Generalized anxiety disorder	Chronic pain
Panic disorder	Use of cannabis or psychostimulants
Agoraphobia	Use of cannabis or psychostimulants
Social phobia	Smoking
Somatoform disorders	Gambling
Eating disorders	Pregnancy/childbirth
	Obesity

bereavement, separation or moving house), loss of physical health, loss of livelihood or loss of prestige/self-esteem. Social relationships, in terms of having at least one close person in whom one can confide and a broader network of social support and contacts, are important buffers against depression.

VIDEO 6.1

"Taking a stand": depression – https://vimeo.com/ 20097105

At this point, it is perhaps worth commenting on the terms 'reactive' and 'endogenous' depression, which fell out of common usage during the 1990s. Students should avoid using these terms, although they may hear them used on the wards or read of them in older literature. The term 'reactive' depression was applied to patients whose depression seemed to be secondary to adverse circumstances, whose symptoms were often less severe and who lacked the somatic or biological symptoms that often go along with more severe depression. The term endogenous depression implied something caused by the person's innate biology, arising without clear external cause, and generally associated with greater severity and somatic symptoms. It became clear that it was not possible to clearly separate two such types of depression using either symptom profiles or the presence or absence of precipitating stressful life events, which were found to occur in both. Thus, we now refer to depressive disorders as a single category.

Biological factors

As in hypertension and diabetes, the risk of adult depression is higher among infants with low birthweight, possibly due to dysfunction of the hypothalamic–pituitary–adrenal axis. The size of this effect is relatively small and the genetic component of the aetiology of depression is also small in comparison with environmental factors. No specific gene has yet been found to play a major role and inheritance is very likely to be polygenic. In broad terms, major depression in a first-degree relative effectively doubles the risk of experiencing depression. That being said, the genetic component of aetiology rises with more severe depressive disorders, with a concomitantly increased risk among relatives. In practice, it can be difficult to disentangle genetic factors from environmental ones.

Psychiatric co-morbidity

Co-morbidity of depression and alcohol use is particularly common in many societies. It can be very difficult to know which arose first, with alcohol offering temporary relief from depression before exacerbating the condition in a vicious circle. Primary chronic alcohol abuse may also lead to persistent chronic low mood. All of the anxiety disorders listed in Table 6.1 are more than twice as common in depressed people as in a control population. The importance of recognizing co-morbidity is that the treatment of both conditions has an additive effect, reversing the type of vicious circle mentioned earlier.

Physical illnesses

As shown in Table 6.1, many physical disorders are associated with raised rates of depression, underlining the need for vigilance in general medical settings.

Miscellaneous factors

Of these, the association with smoking, gambling and illicit drug use may be underpinned by abnormal function in the brain's reward mechanisms. The link with obesity is again a complex 'chicken and egg' vicious circle situation. Mood disorders in pregnancy and childbirth will be discussed separately in the section on 'Childbirth and affective disorders'.

Presentation

Given the high prevalence of depression among adults, and the tendency for many people to deny psychological symptoms, doctors should maintain a high index of suspicion, particularly when someone has associated risk factors for depression. For example, a recently bereaved middle-aged woman with painful arthritis has a high risk of developing a depressive disorder. Patients may also need sensitive questioning as they find some of their feelings embarrassing or difficult to share or feel ashamed of not being able to manage.

Clinical features

Many systems for classifying depression have been in vogue at different times, and in different countries, over the past 60 years. None has been without

its strengths and weaknesses and the *International Classification of Diseases*, 10th Edition (ICD-10) criteria for a depressive episode are summarized in Box 6.1. Differences in the severity of depression are worth emphasising. Mild depression has fewer symptoms (as indicated in Box 6.1) and the mood itself is less likely to impact on every aspect of the individual's life and general functioning. In severe depression, not only is low mood pervasive, but the individual is unlikely to function effectively in any area. The other symptoms are also more likely to be severe so that, for example, there will be a greater disturbance of sleep and appetite or more pervasive thoughts regarding suicide.

Depressed mood is, of course, the central feature of depression. It is distinguished from dejection and unhappiness by its persistence and its intensity. Unlike unhappiness, depressed mood tends not to improve in response to positive events such as the visit of a friend or a victory by one's football team. Uncharacteristic tearfulness is common, and anxiety is a frequent concomitant. When people are depressed for the first time they may find depressed mood difficult to recognize, and irritability may be a more evident affect, especially to family and friends. Diurnal variation of mood, whereby depressed people feel at their worst in the morning and improve as the day progresses, is often evident especially in those more severely ill.

Loss of interest in activities such as work and hobbies is characteristic, coupled with a lack of pleasure and enjoyment from previously pleasurable pursuits (anhedonia).

Loss of energy (**anergia**) is a common presenting complaint in depression, giving rise to a differential diagnosis that includes the various physical causes of energy loss and fatigue. The anergia and easy fatigability of depression tend to be coupled with subjectively impaired motivation, whereas an anergic physically ill person will typically feel that 'the spirit is willing but the body is weak'. As a related symptom, **loss of libido** is common in depression.

VIDEO 6.2

"I'm a reasonable man but…": depression and irritability – https://vimeo.com/49319212

VIDEO 6.3

Tired all the time: depression – https://vimeo.com/ 145381771

Impaired concentration and attention are common. For example, patients are often unable to follow a TV programme or focus on a book. Depressed people will often feel that their thinking is fuzzy and inefficient, and will encounter short-term memory difficulties.

Reductions in self-esteem and confidence result from the negative cognitions associated with low mood (mentioned under 'Aetiological models of depression'), and it needs to be clear that low self-esteem did not predate other features before it should be deemed to be

Box 6.1 ICD-10 criteria for depressive symptoms

Minimum duration of 2 weeks (unless very severe)

Core symptoms	Depressed mood Loss of interest and enjoyment Reduced energy with increased fatigability
Other common symptoms	Reduced concentration and attention Reduced self-esteem and confidence Ideas of guilt and unworthiness Pessimistic view of the future Ideas or acts of self-harm or suicide Disturbed sleep Diminished appetite

Mild depressive episode requires at least two core symptoms plus at least two of the other symptoms

Moderate depressive episode requires at least two core symptoms plus at least three of the other symptoms, and symptoms are likely to be 'present to a marked degree'

Severe depressive episode requires all three core symptoms plus at least four of the other symptoms

a depressive symptom. A pessimistic view of the future is also typical of negative depressive cognitions, as are feelings of personal guilt and unworthiness. There are significant gradations in the degree of negativity of such thinking from mild/moderate depressions to severe depression with psychosis. For example, a moderately depressed person may think it likely that he or she will lose his or her job because he or she has not worked hard enough, whereas a severely depressed patient may hold a delusional conviction that he or she faces redundancy and destitution because he or she was late for work on one occasion.

Thoughts of self-harm and suicide are intimately linked with depression, at all levels from transient thoughts of taking an overdose to completed suicide.

Sleep disturbance is an important symptom of depression. Early morning awakening (sometimes defined as 2 hours or more before one's usual wakening time) is characteristic, especially in more severe depressions. Awakening during the night with difficulty falling asleep again (middle insomnia) and problems falling asleep (initial insomnia) are also common patterns.

Loss of appetite, often coupled with weight loss, is a common depressive symptom that again needs to be distinguished from possible physical causes. In depression, eating is often one of the activities that has ceased to be pleasurable. Some people with (usually milder) depressions may gain weight through 'comfort eating'.

Psychomotor changes tend to occur with more severe depression. Retardation refers to apparent slowing of movement and speech, while patients may report a slowing of their thinking processes. Agitation comprises excessive motor activity, often coupled with anxiety. Patients are fidgety and restless, and can find it almost impossible to stay still.

Obsessive compulsive symptoms (see Chapter 7) occur commonly in depression, often as an exacerbation of pre-existing obsessional personality traits.

VIDEO 6.4

"Everything is black…": severe depression with psychotic features – https://vimeo.com/21068453

VIDEO 6.5

Nihilistic delusions – https://vimeo.com/13209654

Psychotic symptoms occur only in severe depression. Most commonly, these comprise **delusions** that can generally be understood as extreme extensions of depressive thinking. Typical depressive delusions relate to guilt, impending catastrophe, poverty, hypochondriasis or persecution (usually as a result of wrongly perceived misdeeds). When auditory hallucinations occur, unlike in schizophrenia, they tend to be relatively brief and in the second person. The content is usually derogatory, consistent with depressed mood. Visual hallucinations can occur (e.g. of threatening faces or the devil) but they are rare and should generally lead to consideration of organic causation (see Chapter 9).

ICD-10 also classifies depression according to whether or not 'somatic' symptoms are present. These are sometimes termed the 'biological' symptoms of depression, and do tend to point towards a more biological, as opposed to psychosocial, causation and can indicate a greater likelihood that physical treatments will prove to be successful. The somatic/biological symptoms of depression are as follows: loss of interest or pleasure in usually enjoyable activities; lack of emotional reactivity to normally pleasurable events; early morning awakening; depression worse in the mornings; psychomotor retardation or agitation; marked loss of appetite; weight loss; marked loss of libido.

An episode of depressive disorder may be a single event in a person's life. However, for many people further episodes ensue and it is then appropriate to describe the individual as suffering from recurrent depressive disorder. This is classified separately in ICD-10 because recurrence may have implications for future management. This category excludes people with a history of significantly elevated mood, and recovery between distinct episodes differentiates it from chronic depression.

Dysthymia refers to a syndrome of chronic mild depression that does not meet the severity criteria for depressive episodes or recurrent depression. Its onset is often in adolescence. The pervasively negative and pessimistic cognitive style has sometimes given rise to the description of 'depressive personality disorder'.

Depression in older adults

Modest increases in rates of depression have been reported to occur in females at the time of menopause and there has been much debate regarding the aetiology

of this and whether it represents a distinct syndrome of postmenopausal depression. Marked menopausal, hormonal changes cause physical symptoms but no clear link has been established between these hormone changes and mood changes. Menopause also constitutes a psychological landmark, signalling the end of a woman's reproductive years. Socially, it may coincide with life events such as children leaving home, perhaps precipitating 'empty nest syndrome'. Thus, depression at this phase of life may be no different in its causation from that at other times but, as during the postnatal phase, there may be unique psychosocial factors that make females more vulnerable.

In old age, somatic symptoms tend to be more common when depression presents. Prevalence rates are similar to those in younger adults, but there are significant differences between older people living independently and those in institutions. Prevalence rates are high in nursing homes, perhaps largely because risk factors tend to cluster in residents: they are more likely to be bereaved, socially isolated and suffering from concomitant (often painful) physical illnesses. Such factors may serve to perpetuate depression in older people, and depression *per se* will also increase the likelihood of, and the necessity for, admission to institutional care. There is a significant association in older people between depression and early mortality.

There is also a complex association between depression and dementia, with each apparently giving rise to an increase in the chance of developing the other. Dysfunction in the hypothalamic–pituitary–adrenal axis may predispose to both disorders. When elderly people become significantly depressed, as much as 15% of them can develop 'depressive pseudodementia'. This presents with cognitive impairments suggestive of dementia but actually represents a severe slowing of cognitive function caused by severe, but often unrecognized, depression. Effective antidepressant treatment results in return to normal.

Diagnosis and assessment and management of depression

Making the first diagnosis of depression

A large proportion of consultations in primary care are with patients diagnosed as depressed. Scottish government statistics for 2012–2013 estimated that around 400,000 such consultations occurred that year. Depression was the most common reason for consultation with a GP in the 25–44-year-old population. However, although more severe cases are usually detected, there is consistent evidence that only about half of the depressed patients who consult in primary care are actually diagnosed with that condition. There are several reasons for this low detection rate.

Older depressed people are less likely to be diagnosed in primary care. Men, although being less likely than women to consult their GP when they become depressed, are more likely to be diagnosed when they do consult. Somatic presentations may hinder the diagnosis of depression. A WHO study screened primary care attenders across 14 countries, and found that some depressed patients reported only somatic symptoms, with rates that varied from 45% in France, through 60% in Manchester to 95% in Turkey. There was a general increased tendency for patients in developing countries to present somatically, as there is for less educated patients in Western societies. Somatic symptoms in depressed patients occur across all bodily systems, with non-specific symptoms (headaches, lower back pain, weakness and tiredness) being particularly common. In general, the more physical symptoms patients complain of, especially when such symptoms remain medically unexplained, the more likely they are to be depressed. This also applies to hospital outpatient clinics.

Assessment

Patients may present complaining of some sort of mood or affective disturbance. However, as discussed earlier, it is important to maintain a high index of suspicion and consider whether risk factors for depression are present as many patients may present to their GP with a, perhaps minor, physical symptom which is not actually their main concern. The diagnosis is made on the basis of the history and mental state. A corroborative history from a family member is usually valuable.

Patients may equate 'mood' with anger or irritability and studies also indicate that many people cannot clearly distinguish between anxiety and depression in their descriptions of symptoms. Underlying these problems are issues of vocabulary as well as the way people conceptualize abnormal emotional states. Thus, it can be better to ask 'How have your spirits been recently?' It is a good practice to develop a 'checklist' of depressive

symptoms to run through with patients. This should comprise inquiry about mood, sleep, appetite, change in weight, loss of interest/enjoyment, energy/motivation, diurnal variation and concentration. One should always ask about suicidal ideation, and a sensitive way to commence this area of inquiry would be: 'Have things sometimes got so bad lately that you've felt life isn't worth living?' An affirmative response would lead to inquiries about possible suicidal plans and recent acts of self-harm (see Chapter 3). It is important to elucidate the timescale of the current symptoms and thus to know if these have endured long enough (usually 2 weeks) to make a diagnosis and whether they represent a change from usual functioning. If they do not, then consider dysthymia.

Other useful areas to consider in the history include

- Recent life events
- Difficult ongoing life circumstances
- More distant precipitants (e.g. childhood trauma, relationships with parents)
- Possible previous episodes of depression and their management
- Recent physical health
- Family history of psychiatric disorders and suicide
- Co-morbid psychiatric conditions (most notably anxiety and alcohol misuse)

Mental state examination includes observation for possible signs of self-neglect and for psychomotor changes in terms of retardation (unusual slowing of movement or speech) or agitation (fidgeting or restlessness). Tearfulness or irritability may be observed. If the patient's mood appears depressed, it is important to note whether this is consistent throughout the interview or if it is reactive to you and to the topics being discussed. To access possible negative cognitions, it can be helpful to ask: 'How do you see the future?' and 'How have you been feeling about yourself as a person?' In more severely depressed patients, one should routinely inquire about psychotic symptoms.

Differential diagnosis

When patients present with overt psychological symptoms at the mild end of the spectrum of severity, a stress reaction will be the common differential diagnosis. The brevity of symptoms and the clear relationship to stressful life events or circumstances will be important pointers. With more severe and enduring symptoms the psychiatric differential diagnosis will include anxiety disorders, dysthymia, alcohol misuse, dementia (in older patients) and eating disorders (notably in younger females). The more common medical disorders that can present with symptoms of depression symptoms are hypothyroidism and adrenal dysfunction. Among prescribed medication, steroids most often cause depression, with the other drugs listed in Table 6.1 being less commonly implicated.

Aetiological models of depression

Aetiological factors in mental illness are described in Chapter 2. To place management of depression in a logical context, three prominent models of causation are briefly reviewed.

Psychodynamic/Interpersonal

Psychodynamic theories of the aetiology of depression usually emphasize the importance of loss, especially when the person was ambivalent about the loss (e.g. early death of one's mother with whom there was a poor relationship). Loss of self-esteem coupled with negative expectations of relationships is deemed to be important. Disturbed emotions and relationships are considered to be central issues.

Cognitive

Rather than interpreting negative thoughts as the result of depression, cognitive models perceive them to be the primary cause. Underlying negative beliefs are thought to give rise to pessimistic expectations of the future, of relationships, of oneself and of the world in general. This pervasive pessimism leads to depression and withdrawal in a self-perpetuating pattern.

Neurotransmitters

Whether or not it is a primary phenomenon, there is a wealth of evidence to support the link between depression and disturbed neurotransmitter function. As just one example, depletion of tryptophan (a precursor of serotonin) gives rise to lowered mood in both depressed and non-depressed people. The picture is complex, but serotonin and noradrenalin are the neurotransmitters most closely linked to depression.

Management

General principles

Society's views of, and attitudes about, depression can be contradictory and are often reflected by patients and sometimes by doctors. It may be seen by some as a transient state of lowered mood out of which motivated people can ascend by strength of character and a positive state of mind. Allowing oneself to 'wallow in self-pity' can be seen as a self-indulgent weakness, rather than as an illness. Such perceptions will often delay or prevent presentation of depression to health services. For milder presentations, a doctor may not wish to overemphasize an 'illness model', since it is helpful to promote an attitude of 'active mastering' of problems. However, for more severely depressed patients it is often a relief to hear that a doctor considers them to have illness rather than weakness.

In managing depression, it is essential to beware of the fallacy of 'understandability'. As an example, one might form the view that 'The chap lost his job and then his wife left him, no wonder he's depressed' and then having 'understood' the situation a doctor may decide that treatment of depression is unnecessary. This is not too different from saying 'The chap had a bad head injury, no wonder he's got epilepsy' and thus deciding not to treat the epilepsy. If someone has symptoms of depression, these need to be treated, whether or not they are 'understandable'. Nowhere is this more important, arguably, than when people are depressed in the context of a physical illness. In the 'heart and soul' study of over 1000 patients with cardiovascular disease, quality of life did not correlate with any measure of cardiovascular incapacity but correlated strongly with depressive symptoms. Similarly, a study in Calgary found that, among patients with cerebral tumours, depressive symptoms were the most important predictor of quality of life.

Mild depression

If the onset of symptoms are recent and especially if they seem to have arisen in the context of an adverse (but not enduring) life event, the general practitioner is likely to engage in 'watchful waiting'. An initial sympathetic and supportive consultation may be therapeutic in itself. If symptoms have not resolved at a subsequent appointment 2 weeks or so later, then specific therapies would usually be discussed.

Psychological interventions will usually be the first option considered. When disturbed emotions and adverse life circumstances are prominent, counselling and/or problem-solving therapy may be most appropriate. When negative thinking is a predominant component, cognitive behavioural therapy (CBT) may be optimal. Self-help programmes, both through books and the Internet, have been used increasingly over recent years, and can be seen as a convenient, cost-effective addition or alternative to direct person to person therapy. Self-help interventions for depression (at the milder end of symptom severity) are of established efficacy, and effectiveness is associated with their incorporating CBT techniques and by ancillary contact with a therapist ('guided self-help'). Self-help programmes usually also contain educational components and attempt to assist with 'behavioural activation', through which patients address helpless inactivity.

National Institute for Health and Care Excellence (NICE) guidelines advise a structured exercise programme within the routine management of mild depression. Thirty minutes of aerobic exercise, three times a week would be a standard 'prescription'. Support and supervision are usually important since adherence, particularly among depressed patients, may well be a potential problem. The mechanism of the antidepressant effects of exercise is unproven, but the most plausible hypotheses relate to effects on central noradrenergic function, to enhanced feelings of 'self-efficacy' and as a form of behavioural activation.

Antidepressants are not usually indicated as a first-line treatment in milder cases. However, they should be considered when symptoms endure and have not responded to other interventions or if patients have previously responded well to antidepressants when moderately or severely depressed.

Moderate/severe depression

When patients have moderate or severe depression, the first choice of treatment will usually be an antidepressant. Selective serotonin reuptake inhibitors (SSRIs), such as citalopram, fluoxetine or sertraline, are preferred since (compared with the older tricyclic antidepressants) they cause fewer side-effects, treatment adherence is better and they are much less likely to cause death when taken in overdose. Perhaps self-evidently, the danger of overdose should be remembered with all depressed patients and realistic doctors appreciate that

they cannot predict which of their depressed patients might overdose with any satisfactory level of accuracy. Thus, safe prescribing and frequent reviews, in a context of 'suicide awareness', constitute appropriate care.

Patients should be alerted to some of the more common unwanted effects, as this can help compliance. For SSRIs, for example, these are nausea, headache and sweating, which are transient and will usually resolve within 1–3 weeks. A few patients may experience feelings of restlessness which they should be advised to report urgently (see Chapter 18). Although recent evidence suggests that a therapeutic effect within days is not uncommon, it is a standard practice to inform patients that they may well notice no improvement during the first 2 or 3 weeks. Again, this advice should enhance adherence. Once patients have recovered, the risk of relapse is high if therapy is stopped, and antidepressants should be continued for at least a further 6 months, and 12 months for more severe cases. Withdrawal effects, such as headache, anxiety and influenza-like symptoms, are quite common with SSRIs and the dose should be tapered when they are discontinued. Other pharmacological treatments will be mentioned in the section 'Secondary care'.

In moderate depression, psychological approaches (of the types described for mild depression) will often be deployed in tandem with antidepressants and it is helpful for patients to feel that they remain active participants in their own recoveries. CBT and interpersonal therapy are of established efficacy in moderate depression but are not effective for severely depressed patients.

Secondary care

General practitioners will generally refer a depressed patient to psychiatric services when

- Treatment has failed (usually this will comprise non-response to two treatments), or
- Treatment options (usually psychotherapeutic) are not available in primary care, or
- There is perceived to be a significant risk of suicide, or
- Psychotic symptoms are present, or
- Bipolar affective disorder is suspected

The vast majority of referred patients will be treated as outpatients and admission to hospital is generally reserved for people deemed to be at high risk of suicide or self-harm or those with psychotic symptoms.

The psychiatric management of depression is increasingly sophisticated and evidence-based. A full account is given in a review by Cleare et al. (2015), and what follows is an outline.

A detailed appraisal of biological, psychological and social factors will lead to a treatment plan that may well be multidisciplinary. Partially responsive patients will often be engaged in any of the various psychotherapies, in tandem with medication. Involving partners and families, in both clarifying background and symptoms and assisting with therapy, will often be appropriate. Patients referred to secondary care will usually have failed to respond to an SSRI and/or another of the newer antidepressants, in primary care. After attempting to ensure that this has been prescribed in an adequate dose and that non-response has not occurred due to poor treatment adherence, alternative antidepressants will include the following:

- Venlafaxine (a serotonin and noradrenalin reuptake inhibitor), or
- Escitalopram (an alternative SSRI), or
- Mirtazapine (a novel antidepressant with sedative and anxiolytic effects), or
- A tricyclic antidepressant (often clomipramine)

If patients do not respond to such changes, then pharmacological alternatives include the addition of lithium carbonate, as an augmenting agent, or the use of one of the monoamine oxidase inhibitors (MAOI) such as phenelzine.

Electroconvulsive therapy (ECT) is reserved for severely depressed patients, usually those who have failed to respond to several other treatments or when improvement is urgently necessary. In practice, this applies to people who are at risk either through suicide or through inability to eat and drink adequately. Psychotic symptoms and severe retardation are symptoms that may respond to ECT more readily than to other treatment approaches. ECT will be discussed at greater length in Chapter 18.

Recurrent depression

Depression is frequently a recurrent illness. Following a moderate depressive episode the lifetime risk of recurrence is approximately one in two. After two

episodes, the likelihood of recurrence is about 80% and rises to over 90% after three episodes. When several episodes occur over a short space of time, or when there have been many episodes over a longer period, long-term, prophylactic antidepressant treatment is usually advised. Patients with recurrent episodes should usually receive CBT since this has been found to have prophylactic efficacy.

Are doctors prescribing too many antidepressants?

Doctors are often criticized for 'medicalizing unhappiness' and then prescribing antidepressants for people who do not need them. However, many people in the community who are depressed and would benefit from antidepressants do not receive them; epidemiological studies confirm that unnecessary prescriptions are far outnumbered by the people who would indeed benefit from therapy (either pharmacological or psychological) that they are not receiving. Unfortunately, policymakers may be unduly influenced by simplistic public perceptions ('pills bad, talking therapies good') that stigmatize antidepressant therapy and prevent depressed people from presenting for assessment and accepting treatment. The NICE guidelines (2009), which are currently being updated, provide a useful overview of the different perspectives and may be useful further reading.

Bipolar affective disorders

As well as experiencing episodes of depression, some patients also experience episodes of mania (or the less severe hypomania), in which the principle features are elevated mood, increased energy and pressure of speech. A bipolar affective disorder is characterized by separate episodes of mood disturbance at the two 'poles' of depression and mania. In comparison with (unipolar) depression, it is less common and management tends to be initiated and supervised predominantly by psychiatrists. For these reasons, although it is important and fascinating, it will be accorded less space than depression.

History

The origins of the term 'mania' are obscure, but typical clinical pictures were described by Greek physicians as early as the fifth century BC. It was in the first century AD that Aretaeus explicitly linked mania with melancholia, but this link seems to have been lost until the latter half of the nineteenth century, when various terms including 'folie circulaire' and 'manic-depressive insanity' emerged. Manic-depressive illness became the established diagnostic terminology until quite recently, but now the terms bipolar affective disorder, or simply bipolar disorder, are preferred. Although it may be slowly diminishing, stigma persists, despite the knowledge that prominent people have functioned very well while suffering from a bipolar affective disorder (e.g. Winston Churchill). Bipolarity is linked to creativity and has contributed (among a long list) to the work of Van Gogh, Tchaikovsky, Shelley, Byron, Sylvia Plath and Spike Milligan.

Prevalence

Estimates of the prevalence of a bipolar affective disorder vary depending on the differentiation of illness from normality. Cyclothymia (sometimes referred to as cyclothymic personality) describes a picture of persistent periods of mild depression and mild elation. This 'disorder' (if indeed it is a disorder) sits between 'normality' and a bipolar affective disorder. The prevalence of bipolar affective disorders does not vary greatly between countries or between socio-economic groups, suggesting that the aetiology is more biological and less psychosocial.

The lifetime risk of tightly defined bipolar affective disorder with episodes of mania (bipolar I disorder) is slightly less than 1%. If one includes less-severe cases, with hypomania (bipolar II disorder) rather than mania, then the lifetime risk doubles to nearly 2%. For bipolar I disorder males and females are affected with equal frequency, but there is a slight preponderance of women among people with bipolar II disorder. The commonest age of onset (quite often identified retrospectively) is between 18 and 25 years. Younger age at onset may be associated with a higher genetic loading and there may also be a much smaller peak of onset in old age associated with organic brain disease. There is also a peak of first illnesses in women during the postnatal period (see below).

Associations and risk factors

It is difficult to disentangle whether some factors have a causal role, result from the disorder itself,

or are co-morbid due to a common aetiological link. For example, alcohol misuse can result from self-medication for mood swings but can also cause or exacerbate mood swings, or the two conditions could have a shared genetic predisposition.

Genetic predisposition is the most important risk factor. Monozygotic twins have a concordance rate of over 50% for bipolar affective disorder, and the risk among first-degree relatives is between 5% and 10%. The pattern within affected families fits with a model of multigenic inheritance rather than one of specific genes with major effects. Some studies have found overlaps in genetic susceptibility between bipolar disorder and schizophrenia (see Craddock and Sklar, 2013).

Abnormal brain function has been demonstrated in patients with bipolar disorder using functional neuroimaging techniques. There is evidence for abnormalities in some regions of grey matter, white matter connectivity and in some of the neural circuitry relating to emotional processing. Some of these abnormalities differ from those seen in patients with unipolar depression.

Table 6.2 lists several conditions through which intracerebral pathology can predispose to manic episodes. If there is a common anatomical site of importance, then this may well be the prefrontal cortex and other limbic structures.

The role of adverse life events in precipitating mania is not clear cut since these may not be independent of the illness itself: when someone is becoming manic they may be an active instigator of a life event rather than its passive victim.

The association between childbirth and affective disorders will be covered below, and the link with circadian rhythms will be mentioned under seasonal affective disorder (SAD).

Presentation

When people with bipolar affective disorder are not in a phase of normal mood (euthymia) they are less likely to be manic or hypomanic than they are to be depressed; spells of depression are usually more common and tend to last longer. The clinical features of depression are as described earlier and the features of mania/hypomania will now be described.

Clinical features

Summarized ICD-10 criteria for mania and hypomania are shown in Box 6.2. The distinction between mania and hypomania is made (somewhat arbitrarily) on the basis of duration and severity. When patients are manic they are clearly unwell but hypomania can be difficult to detect. Hypomanic patients will be cheerful, optimistic and energetic, and, especially if they are not known to you, can seem to be positively healthy and within the limits of normality. This possible blurring of the 'illness–wellness

Table 6.2 Bipolar affective disorder: risk factors and associations

Genetic Predisposition	**Medication**
Medical Conditions	Corticosteroids
Hypothyroidism	L-Dopa, dopamine agonists
Cushing's disease	Thyroid hormones
Multiple sclerosis	MAOI and tricyclic antidepressants sometimes precipitate mania
Epilepsy	**Miscellaneous**
Cerebrovascular disease	Circadian rhythm disruption
Cerebral tumours	Insomnia/sleep disruption
Head injuries	Travel across time zones
Substance Misuse	Shift work
Alcohol	Childbirth
Psychostimulants	Adverse life events
Cannabis	Summer months of the year
	Pathological gambling

Abbreviations: L-Dopa, L-3,4-dihydroxyphenylalanine; MAOI, monoamine oxidase inhibitor.

Box 6.2 Summarized ICD-10 criteria for mania and hypomania

Mania

1. Elated mood (occasionally predominant irritability)
2. Increased energy
3. Several of the following: decreased need for sleep, grandiosity, excessive optimism, pressure of speech, loss of social inhibitions, inability to sustain attention, impulsivity, extravagance, aggression
4. Episode of at least 1-week duration
5. Severely disrupts work and/or social activities
6. Psychotic symptoms may be present

Hypomania

1. Mild elevation of mood (or irritability)
2. Increased energy
3. Symptoms as in (3) in the preceding list, but *not* to the level of severe disruption in work or social contexts
4. Duration of 'at least several days on end'
5. No psychotic symptoms

boundary' should be borne in mind in the descriptions that follow.

Mood is persistently elevated, most usually with excessive cheerfulness and extraversion, less commonly with predominant irritability. Subjectively, patients characteristically report feeling extremely well and their good humour is often infectious at interview. Excessive drive and optimism, when thwarted, can lead readily to confrontation and aggression.

Energy levels are high and overactivity is often apparent. Characteristically, patients will rush around starting new tasks or projects, but tend not to bring these to satisfactory conclusions.

Patients report decreased sleep requirement without this causing fatigue. Manic patients may go for several days and nights without sleep.

Thinking is positive and optimistic, with patients considering new plans and ventures to be brilliant ideas despite their clear irrationality. At the severe end of the spectrum, plans and self-belief can reach the level of grandiose delusions, e.g. resolving the world's energy problems by harnessing the power of ants' colonies. Patients may come to believe that they have special powers (e.g. telepathy) and become frustrated and/or aggressive when their ideas are challenged.

VIDEO 6.6

"I'm raising money for the babies…": mania – https://vimeo.com/13268336

Impulsivity is characteristic, frequently becoming evident in overspending and/or in promiscuity. Risks tend to be taken more readily and, coupled with difficulties in sustaining attention, this makes manic patients particularly dangerous behind the wheel of a car.

At **interview**, dress may be bright and gaudy or inappropriate and idiosyncratic in other ways. Self-neglect may be apparent. Patients will usually talk loudly and quickly (pressure of speech). They may describe subjectively speeded thinking and may jump from topic to topic with only tenuous links between one topic and the next (flight of ideas). Elated mood, irritability, grandiosity and overactivity may all be observed. If delusions or hallucinations arise, these are consistent with elation and grandiosity (e.g. messages from God). Manic patients often lack insight into the fact that they are ill.

Mixed affective states

People with mania can often exhibit emotional lability, being moved quickly to tears or anger and then recovering with equal rapidity. In mixed affective states the depressive affect is more enduring but is combined with other manic symptoms such as increased energy and decreased sleep requirement. These mixed states are now being recognized more commonly, making up perhaps 10%–20% of presentations of mania/hypomania, and appear to be a time of increased risk of suicide. In relation to management, they should be treated in the same way as a manic/hypomanic state.

Neuropsychological dysfunction

Growing evidence shows that many patients with bipolar disorder exhibit neuropsychological impairment even during periods when they are well. These difficulties are mainly with attention, memory and executive function (problem-solving, decision-making). Their severity is associated with

more severe bipolar disorder in terms of numbers of manic episodes and hospitalizations.

Older adults

Bipolar affective disorder is rather less common among older adults, perhaps due to excess mortality among younger sufferers; it does not appear to 'burn itself out' in old age and episodes may become more frequent and more severe over time. As many as 10%–20% of older people with bipolar disorder can have a first episode of mania after the age of 50 years, although many will have a history of previous depressive episodes. Compared with patients who have an earlier age of onset, mania in old age is associated with a lower likelihood of a family history but increased rates of cerebral organic disorders and neurological co-morbidity. In old people, the possibility of 'secondary mania' should be strongly considered, noting particularly the physical and pharmacological risk factors listed in Table 6.2.

Course and outcome

Follow-up studies find that the 'average' person with bipolar disorder is well (euthymic) for 50% of the time, manic/hypomanic for 10% and depressed (to varying degrees of severity) for as much as 40% of their lives. The fact that (often mild) depressive symptoms are so common has only recently been recognized. This may contribute to indifferent social outcomes with high unemployment, reduced rates of marriage and high rates of divorce. Suicide occurs in as many as 10% of people with bipolar disorder. Factors associated with poorer outcomes include:

- Longer time spent depressed
- Psychotic symptoms
- Early onset
- Alcohol/drug misuse
- Poorer social support systems
- Low socio-economic status

Assessment

When patients are seen (in whatever setting) with a depressive episode, it is important to establish whether the pattern is of unipolar depression or of bipolar disorder, since the subsequent assessment and management may diverge significantly, although at the first depressive episode it may not be possible. Manic symptoms usually necessitate referral to specialist services. That said, a community prevalence of around 2% means that doctors in all specialties will encounter patients with bipolar disorder and thus need to be able to recognize the symptoms.

The interview

Interviewing manic patients can be difficult as their flight of ideas and pressure of speech can take the interview rapidly off course. Skill and judgement are required to retain appropriate control over the consultation. The patient needs to talk enough for the mental state and history to be clarified, and if one tries to exert excessive control then unnecessary confrontation can ensue (especially if their mood is labile and they are irritable). Given that a manic patient may well be grandiose and without insight, negotiation regarding treatment is rarely straightforward.

Different issues are presented by hypomanic patients in whom changes of mood and mental state are more subtle. Questions will focus on the symptoms listed in Box 6.2, with particular emphasis on mood ('too cheerful to be healthy?'), energy ('more active than usual without feeling tired?'), reduced sleep requirements and new ideas/interests. Hypomanic people with insight can often 'hold it together' for the duration of a medical consultation, leading to a diagnosis of 'happiness', and an informant should also be interviewed whenever possible and appropriate.

In the history, it is important to establish the temporal pattern of mood changes. The duration of the current episode is of clear importance as is the nature and frequency of any previous episodes of depression or elation. This also affords an opportunity to assess the possible effects of these mood swings on the life of the patient and on those around him or her, especially if a family member is present. Family history can often be further elucidated during an interview with a relative. A history of possible substance misuse should always be taken carefully. Remember also that abuse of alcohol and other substances may be secondary to the impulsivity and loss of normal social awareness that occurs in mania. A corroborative history can be crucial.

Differential diagnosis

In known patients with bipolar affective disorder, diagnosis of elated mood is seldom problematic, but it may not be so clear in a first episode of mania. 'Secondary'

mania, caused by factors listed in Table 6.2, should be considered and a history of physical symptoms and medications along with a physical examination should be conducted. A urine drug screen is the most important routine investigation.

The main psychiatric differential diagnoses in adults will be schizophrenia, schizoaffective disorder and substance misuse (see Chapters 9 and 10). The first rank symptoms of schizophrenia can occasionally occur in psychotic mania and, by definition, symptoms of both schizophrenia and affective disorder occur together in the same episode in schizoaffective disorder.

Management

The initial diagnosis and management of patients with bipolar disorder will be undertaken, almost always, by mental health teams. Once management strategies are established, care will be undertaken by, or shared with, primary care teams; ongoing shared care may be optimal, as patients' quality of life can be enhanced by skilled management and continuity of care.

Manic episodes

In acute mania, rapid symptom control is required to ameliorate behaviour, which can often be dangerous to the patient and very disruptive to others. Admission to the hospital will very often be necessary, with or without compulsory detention. Patients with mania are easily overstimulated, worsening their mental state, and it is helpful to attempt to redress this within as quiet and consistent an environment as possible.

In mania, a combined pharmacological approach with a mood stabilizer and an antipsychotic medication is usual. Lithium or valproate are the commonest mood stabilizers deployed in the acute situation. Second-generation antipsychotics (e.g. olanzapine, risperidone, quetiapine) are effective antimanic medications and are now the usual first-line treatments, although haloperidol may be considered. A high-potency benzodiazepine, such as lorazepam, can be helpful as an adjunctive sedative to promote behavioural control, and these are often used in the first few days of treatment. When intramuscular medication is required (in practice when the patient refuses oral medication or this is proving to be ineffective) lorazepam and/or haloperidol are often used.

In hypomania, the situation is much less acute and can often be managed on an outpatient or day treatment basis. A mood stabilizer alone, often the one on which the patient might continue prophylactically, usually suffices. In both mania and hypomania, it is helpful to re-establish a normal sleep–wake cycle, and the temporary prescription of a hypnotic can be therapeutic.

Depressive episodes

Management of depression in the context of bipolar disorder (often termed 'bipolar depression') differs from that of unipolar depression as there is a paucity of good clinical trial evidence for the prescription of antidepressants. Antidepressants are less effective and (particularly those with effects on noradrenaline [NA] as well as serotonin/5-hydroxytryptamine [5-HT] systems) can cause switching into mania. Mood stabilizers are generally more efficacious than antidepressants. For patients with bipolar depression who are not already on a mood stabilizer, or for whom this is not effective, quetiapine or a combination of fluoxetine with olanzapine may be effective.

Prophylactic medication

Although it may be ideal to instigate prophylactic treatment after one episode of mania, patients will rarely agree to long-term medication until they have experienced at least one recurrence. Lithium (see Chapter 18) has been used for over 50 years and is of known effectiveness, rather more so in the prevention of mania than depression. The emergence of side effects and the need for long-term monitoring and blood tests are factors that discourage patients from embarking on treatment, which is advised to be for an absolute minimum of 2 years. The anticonvulsant drug sodium valproate is also used extensively and there is a growing case for lamotrigine, which is more effective at preventing depressive than manic recurrences. Second-generation antipsychotics such as olanzapine, risperidone and quetiapine all have prophylactic efficacy, although these too are not without disadvantages, especially with long-term use (see Chapter 18). Not infrequently, mood stabilizers are used in combination if patients do not

respond adequately to one alone. Up to 30% of patients may not demonstrate a good response to treatment.

Psychological/psychosocial therapy

Psychological therapies have no place in the acute management of mania, but can have a role in the non-acute phases of a bipolar disorder, albeit a less central one than in depression. The approaches are, however, less well evidence-based.

Common to almost all psychological approaches in bipolar disorder is a strong component of psychoeducation, through which patients acquire knowledge about treatments and insight into their illnesses. This is often vital if patients are to adhere adequately with prophylactic medication. The identification and avoidance of factors such as work stress or sleep disturbance, which may precipitate a relapse, is part of this process. Patients should be encouraged to identify their own individual 'early warning signs' that are the first symptoms heralding a relapse (e.g. irritability, two consecutive sleepless nights) so that, hopefully, a possible episode of illness can be 'nipped in the bud' at an early stage.

Seasonal affective disorder

Seasonal fluctuations in health and mood have been described by doctors for literally thousands of years, but the term SAD has been in use only since the 1980s. The picture varies near the equator, but at temperate latitudes SAD is fairly synonymous with recurrent winter depression. Symptoms characteristically commence in the autumn and remit in spring and around one-third of people with SAD become mildly hypomanic during the spring and summer months. Many winter symptoms are those of non-seasonal depression but, in contrast to the picture of somatic syndrome, in SAD people usually experience hypersomnia (struggling to awaken in the morning), daytime somnolence, carbohydrate craving and weight gain. Since many of us experience these seasonal changes to some degree, the line between 'normality' and 'disorder' is necessarily somewhat arbitrary, but around 3% of the United Kingdom population suffers from clinically significant SAD. It is most common among women of reproductive age. Since light deprivation is the main cause, it is unsurprising that moving to live further away from the equator confers an increased risk.

Management of mild to moderate symptoms includes encouragement to continue exercise and daylight exposure during the winter months. Light therapy, most commonly with light boxes or with dawn-simulating alarm clocks, is the usual first choice of treatment when moderate to severe symptoms are present. In SAD there is circadian phase delay and circadian rhythms can be advanced by bright light, usually of about 30 minutes duration, around breakfast time. When patients prefer, or when light therapy is unavailable or only partially effective, non-sedative antidepressants (usually SSRIs) can be prescribed.

Childbirth and affective disorders

Mood changes in the post-partum period are common and well recognized. In order of increasing severity, these comprise maternity blues, postnatal depression and puerperal psychosis.

Maternity blues or 'post-partum blues' are so common as to be normal, and are reported by about two-thirds of women. Symptoms include weepiness, irritability and despondency. They peak on days 3–7 post-partum and are usually transient and self-limiting, although severe maternity blues sometimes merge with depression.

Postnatal depression is deemed to affect 10%–15% of women, although attention has recently been drawn to international differences in prevalence. Onset is usually within 6 weeks of delivery. The usual symptoms of depression occur, although mood can be more fluctuating, with irritability and impaired attention often being prominent. Aetiologically, the profound and rapid falls of circulating hormone levels (e.g. progesterone, oestrogen and prolactin) after delivery are considered important. Having a baby is also, of course, a significant and complex life event, giving rise to major family, relationship and occupational changes. Women very commonly experience post-partum physical complaints, including backache,

fatigue and perineal pain (which may be accompanied by sexual difficulties). Coping with a new baby is not straightforward, yielding a mix of emotional and social disruption, often exacerbated by sleep deprivation. Despite all this, new mothers are expected to be happy; this expectation may contribute to the depression itself and to the unfortunate finding that the majority of postnatal depression goes undetected. Detection and treatment are important, not only for the woman herself, because the children of untreated postnatally depressed mothers have been shown to experience developmental delay and the development of the child/mother relationship may be disrupted. Depressed mothers usually benefit from discussing their difficulties within a supportive therapeutic relationship, and more formal therapies such as CBT and interpersonal therapy are of established effectiveness. Antidepressants are prescribed for moderate to severe depression with due attention to possible breastfeeding; sertraline is one drug which is present in only minimal amounts in breast milk.

Puerperal psychosis occurs at a frequency of 1 or 2 per 1000 deliveries. The vast majority of these serious illnesses are affective psychoses, and women are about 20 times more likely to experience a manic illness in the first month post-partum than at any other time of their lives. Women with a history of bipolar affective disorder are at hugely increased risk, at around one in three, and there is a similar risk of recurrence after a previous puerperal psychosis. Early detection can be facilitated by acquiring this history. The onset is usually acute and within the first week after childbirth. Among the usual symptoms of affective psychosis, there is often also perplexity and confusion. Danger to the baby (because of psychosis which may lead the mother to believe the child would be 'safer' dead) and to the mother (largely through suicide) must be closely assessed. If possible, admission should be to a psychiatric mother and baby unit so that both can be cared for while mother–infant bonding is facilitated.

Pregnancy was traditionally regarded as a time of emotional well-being for women. There is now convincing evidence that this is not always the case. Around 10% of women will experience depression during pregnancy and as many as 7% will have an anxiety disorder. One study found that less than 6% of women with an antenatal psychiatric disorder received treatment. This is particularly unfortunate since antenatal disorder can proceed untreated into the postnatal period. Furthermore, independent of postnatal maternal well-being, it is now known that the children of mothers who are depressed or anxious in pregnancy do less well than other children; they are at higher risk of emotional and behavioural difficulties and they are prone to developmental delay. Psychological interventions (CBT, interpersonal therapy, group therapy) are usually the first options, given the wish to avoid medication in pregnancy if possible. However, in view of the possible long-term sequelae of maternal depression, and sometimes risk of suicide, antidepressant medications are sometimes required. There is limited data available regarding the safety of all antidepressants in pregnancy, but in general the SSRIs citalopram, escitalopram and sertraline are probably to be preferred. These three do not seem to be associated with any increase in birth defects while fluoxetine and paroxetine, particularly if used in early pregnancy, are associated with increased defects. There is a small increased risk of persistent pulmonary hypertension of the newborn with SSRI use in late pregnancy and there is also some evidence of a small increased risk of preterm birth. Fetal development during the first year of life does not appear to be affected.

Premenstrual depression

Changes in well-being across women's menstrual cycles have long been recognized, with cyclical, physiological and emotional symptoms being referred to as premenstrual syndrome, and this may include some features similar to depressive illness. When more severe psychiatric symptoms prevail, premenstrual depression can be diagnosed. Typical symptoms comprise low mood, anxiety/tension, anger/irritability, lethargy, poor concentration, and changes in sleep and/or appetite. However, it is worth noting that this diagnostic category is not free of controversy with some rejecting the concept as they feel that a woman's biology is being construed as a psychiatric disorder. However, in some cases the quality of life can be markedly improved with treatment.

Summary

The key points relating to mood disorders can be summarized as follows:

- Depressive disorders are very common.
- Depression occurs with increased frequency in a wide range of medical conditions.
- The detection and management of depression are core medical skills.
- About 50% of depression is not detected by doctors, often because patients present with somatic symptoms.
- Depression usually responds to antidepressants and/or psychological therapies.
- Very few other medical interventions rival the effects of treating depression on a patient's quality of life.
- Bipolar affective disorders occur in about 2% of the population.
- Depressed patients should be asked routinely about manic/hypomanic episodes.
- Especially in elderly people, mania can arise from other medical conditions or from prescribed medications.
- Untreated mania can pose a significant risk to the patient's and/or others' safety.
- Patients with bipolar disorders usually require long-term treatment with a mood stabilizer.

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CASE STUDY 6.1

You are a general practitioner consulted by a 26-year-old man with dyspepsia. You notice that he is glum and he has felt low since splitting up with his partner 6 months ago.

What symptoms would you ask about in relation to a possible diagnosis of depression?

The first task is to establish his symptoms of dyspepsia, and to treat these if this is appropriate.

You want to know the timescale of his low mood, and whether this is improving or worsening.

The specific symptoms to check include diurnal variation of mood, loss of interest, anhedonia, sleep, appetite/weight change, energy/motivation, concentration and suicidal ideation.

You conclude that he has symptoms of moderate depression. Which factors might lead you to advise initial cognitive behavioural therapy rather than antidepressants?

No urgent need for treatment (e.g. low suicide risk) as CBT will take longer to commence.

Patient preference.

Negative thinking and behaviour patterns are prominent.

Intelligent and psychologically minded patient.

No previous good response to antidepressant.

CASE STUDY 6.2

You are a general practitioner who is consulted by a middle aged lady with recurrent depression.

What would you be most keen to elicit from her?

You would want to be clear about how often these bouts have occurred, how long they endure and how much they impact on her functioning.

Are there intermittent precipitating factors, such as season of the year, or premenstrual syndrome?

Are there ongoing stress factors such as marital disharmony or work-related issues?

What management strategies might you suggest?

Counselling for stress factors would be considered.

Other precipitating factors could be treated e.g. light therapy for seasonal depression.

Antidepressants would be strongly considered if the episodes are severe and/or lengthy and/or numerous, and if they have helped during previous episodes.

Cognitive behavioural therapy might be advocated if the patient's thinking patterns were negative, and/or she did not wish to take antidepressants.

If these strategies proved ineffective, then referral to psychiatric services would be appropriate.

CASE STUDY 6.3

You are a general practitioner and a patient's wife has called to express anxiety that her husband is becoming hypomanic.

What would you ask her on the telephone to ascertain whether he is indeed manic/hypomanic?

If you did not know already, you would ask if he is prescribed prophylactic mood-stabilizing medication and whether he has continued taking this.

The symptoms you would ask about would include elevated mood/excessive cheerfulness, increased energy, reduced sleep requirement, irritability, impulsivity and grandiosity.

You see him and find him to be hypomanic, but he does not want to see a psychiatrist. Which factors would make you insist that he does?

These would include:

The severity of his symptoms.

Your assessment of risk, e.g. is he safe to drive (or willing to stop driving)? Is he able to make decisions rationally? Is he excessively impulsive (e.g. financially)? Is he irritable/aggressive, can he care for children if this is an issue?

His willingness to accept treatment and your willingness to provide it – this will range from his complete refusal to his being willing to restart a medication which has previously been rapidly effective.

The home situation in terms of support from his wife (plus other relatives/friends) and the presence of young children.

CHAPTER

7 Anxiety disorders

CIARAN MULHOLLAND

KEY CHAPTER FEATURES

- Clinical presentation of anxiety and 'medically unexplained' symptoms
- Important aspects of generalized anxiety disorder, panic disorder, obsessive–compulsive disorder, stress-related disorders, somatoform disorders and conversion disorders
- Treatment approaches for the above conditions

Introduction

This chapter describes psychiatric disorders in which anxiety is a key feature of the overall presentation, including anxiety disorders themselves, stress-related disorders, such as post-traumatic stress disorder (PTSD), and obsessive–compulsive disorder (OCD). Phobic anxiety disorders (agoraphobia, social phobia and specific phobias) are described in Chapter 8. This chapter also briefly describes two groups of disorders that present with medically unexplained symptoms: (1) the somatoform disorders (somatization disorder, hypochondriacal disorder, somatoform autonomic dysfunction, persistent somatoform pain disorder) and (2) the dissociative or conversion disorders. All of the disorders above are grouped together in the *International Classification of Diseases, 10th Revision* (ICD-10) classification system as 'neurotic, stress-related and somatoform disorders' (Box 7.1) as they have historically been seen as interrelated and are often associated with psychological stress.

Why is this topic relevant to you?

The anxiety disorders are very common. All doctors deal with patients with these conditions, especially

Box 7.1 Neurotic, stress-related and somatoform disorders (from ICD-10)

F40	Phobic anxiety disorder
F41	Other anxiety disorders
F42	OCD
F43	Reactions to severe stress and adjustment disorders
F44	Dissociative (conversion) disorders
F45	Somatoform disorders
F48	Other neurotic disorders

EXERCISE 7.1

Anxiety is probably easier to understand than some of the other conditions in this book as all of us have been anxious at some time. From your own experience of anxiety-provoking situations try to answer the questions given below:

What are the physical and psychological symptoms of anxiety?

When do you think anxiety is appropriate and when is it inappropriate?

When you are anxious how do you try to manage the anxiety?

Can you think of inappropriate or unhelpful techniques that some people adopt to try to overcome anxiety?

The answers to this exercise are summarized later after some of the above issues have been addressed.

VIDEO 7.1

Mary's story: anxiety – https://vimeo.com/12105992

general practitioners (GPs). Treating anxiety disorders is a major component of the workload of psychiatrists. A full understanding of the presenting symptoms and treatment of the anxiety disorders will help you in your future career.

Medically unexplained symptoms also present frequently to GPs, physicians, surgeons and other doctors. These symptoms are often puzzling and sometimes bizarre. There is a risk of over-investigation and overtreatment when patients present in this way and an awareness of the presentation of such symptoms and the treatment options available is thus necessary for all doctors.

What is anxiety and what is 'normal' anxiety?

Anxiety is a mental state that is characterized by physical and psychological symptoms as outlined in Box 7.2.

Box 7.2 Features of a state of anxiety

Affect	Feeling fearful
	Feeling apprehensive
Thoughts	Themes of misfortune
	Concerned will not be able to cope with stress
	Unrealistic ideas of danger
Arousal	Increased arousal/alertness
	Hypervigilance
	Restlessness
	Poor sleep (especially initial/middle insomnia)
	Poor concentration
	Exaggerated startle response
Behaviour	Reduced purposeful activity
	Increased purposeless activity
	Avoidance of some situations
Somatic	Palpitations
	Hyperventilation
	Light-headedness
	Numbness or tingling
	Retrosternal constriction
	Increased muscle tension
	Chest pain
	Nausea
	Diarrhoea
	Sweating
	Tremor
	Headaches
	Dry mouth
Associated symptoms	Depersonalization/derealization
	Irritability
	Low mood

Most of us can quickly grasp the nature of the anxiety disorders as we have all felt anxious when faced with a stressful situation. Anxiety in such situations is normal and adaptive (if we are not a little anxious prior to an important examination, for example, then we might not study hard enough). In summary, normal anxiety is a response to a known problem, usually a definable and external threat of short duration. Further examples of such threats include giving a speech, performing on a stage or facing a new situation or meeting new people.

When might anxiety be a problem?

Given that most of us experience anxiety at various times throughout our lives, it is easy to underestimate how those suffering from anxiety disorders can be compromised by their condition. Anxiety is considered abnormal (or pathological or clinical) if it is extreme and/or it is misplaced, and it thus prevents the individual from getting on with the tasks of daily living or interferes with life in other ways. By 'extreme' we mean when anxiety is much too exaggerated for whatever situation the person is faced with. By misplaced we mean when anxiety is not confined to recognized 'stressful' situations.

Some people have what is known as high-trait anxiety (or an anxious personality). These individuals are more anxious than the average person but do not meet criteria for an anxiety disorder as such. Anxiety is present without any stressful context and becomes almost the 'natural state of being' for an individual (anxious personality is discussed further in Chapter 16).

With pathological anxiety, there is a sense of fear which is not well defined. Any threat is not immediate (for example, an examination that is many months away) or there may be no threat whatsoever. The threat may be 'internal' to the person and the situation is often chronic. Reassurance is rarely effective and can exacerbate the situation as it confirms the original misperception that there is a reason to worry.

Pathological anxiety can occur as a secondary feature to other psychiatric illnesses, such as depression, dementia and psychotic disorders, and it can also occur secondarily to a range of physical conditions. Examples include a range of endocrine disorders like thyrotoxicosis, hypoparathyroidism, carcinoid syndrome, Cushing's disease, phaeochromocytoma, pituitary disorders and hypoglycaemia. Cardiovascular conditions (hypoxia, congestive heart failure, pulmonary embolism, hypertension, angina, myocardial infarction, arrhythmias, mitral valve prolapse [MVP]), neurological conditions (seizures) and respiratory conditions (chronic obstructive pulmonary disease, asthma) can also present with anxiety.

Drug intoxication (for example, with cocaine or amphetamines), medication side effects (theophylline, steroids, anticholinergics) or withdrawal states (especially from benzodiazepines and alcohol) commonly cause anxiety-type symptoms. Another potential cause of anxiety symptoms is excess use of caffeine or 'caffeinism' (Box 7.3). Stopping caffeine suddenly is not a good idea as withdrawal effects are likely. The best strategy is to gradually reduce caffeine intake, preferably over several weeks. Psychoeducation about the effects of caffeine may also be useful.

Finally, large numbers of individuals have primary anxiety disorders: generalized anxiety disorder (GAD) and panic disorder (covered in this chapter), and agoraphobia (with or without panic disorder), social phobia and specific phobias (covered in Chapter 8). The other conditions dealt with in this chapter have prominent anxiety symptoms as part of their clinical presentation.

EXERCISE 7.3

Summary of answers to exercise

The physical and psychological symptoms of anxiety are listed in Box 7.2.

Anxiety is appropriate in a stressful situation, so long as it is not more extreme than the stressful situation would normally provoke. Normal anxiety is a response to a known problem, usually a definable and external threat of short duration. Anxiety is inappropriate if it is too extreme in the circumstances (e.g. severe anxiety many months before an exam) and/or misplaced (e.g. anxiety is present all of the time for no apparent reason).

Individuals vary in their approach, but appropriate ways of managing anxiety include various relaxation techniques (for example, yoga) and taking exercise.

The most obvious examples of inappropriate or unhelpful techniques to overcome anxiety are smoking and the misuse of alcohol and benzodiazepines.

Box 7.3 Caffeinism

- Caffeinism occurs when an individual suffers from side effects of caffeine; the individual takes larger amounts over time and needs to keep drinking caffeine to function properly (resulting in a craving for caffeine).
- Caffeine of 250–500 mg per day is reasonable (equivalent to four cups of tea or coffee). Caffeinism occurs if one has an intake of above 600–750 mg of caffeine per day. Drinking more than 1000 mg per day is well into the toxic range and likely to produce symptoms of anxiety. The U.S. Olympic Committee considers caffeine a stimulant.
- Suddenly stopping taking caffeine can produce problems. Withdrawal from even moderate amounts of caffeine can produce headaches (52%), anxiety (10%), rebound drowsiness, fatigue and lethargy.

Explaining neurosis

In the past, the term 'neurosis' was used to describe many of the conditions now classified in Box 7.1 and the term remains in the ICD-10 title of this group of conditions. The word was coined in 1769 by the Scottish physician William Cullen, who recognized that some of his patients presented with what were at that time medically unexplained symptoms:

> *In a certain view, almost the whole of the diseases of the human body might be called nervous … in this place I propose to comprehend the title neurosis to all those preternatural affections of sense and motion which are without pyrexia as part of the primary disease and which do not depend on a topical affection of the organs but upon a more general affectation of the nervous system.*

In the eighteenth and nineteenth centuries, the term neurosis was used for a wide range of conditions which are now known to have an underlying physical pathology (for example, diabetes, epilepsy, chorea) or which have since been classified more accurately as specific psychiatric conditions (for example, depression).

Neuroses were traditionally seen as disorders characterized by unimpaired reality testing (that is, the person is in contact with reality compared with psychosis, where this contact is lost or impaired), with preservation of insight and an absence of any demonstrable organic basis to the condition. There was an implication that 'psychotics' were very ill but did not recognize it and that 'neurotics' were less ill but were very aware of it though this is often far from the truth. Indeed, the term the 'worried well' has been unfairly applied to so-called 'neurotics'. The ICD-10 classification system has little use for the term neurotic (it is more or less confined to the title of a chapter) and the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition* (DSM-5) classification systems does not use it at all, recognizing that the term has come to be used as a generalization for any situation in which anxiety or emotional symptoms are prominent and is often used pejoratively.

Aetiology of the anxiety disorders

The anxiety disorders are not a homogeneous group and do not have a single cause. Important aetiological factors include pre-existing personality traits, the impact of psychosocial stress and the degree of social support available to an individual. Genetic factors are less important for the anxiety disorders than for some other psychiatric conditions, but they are not unimportant. Older twin studies suggest that anxiety disorders, in general terms, show heritability. In more recent studies, panic disorder and agoraphobia demonstrate the greatest, most consistent genetic effects. In relatives of individuals with panic disorder, for example, there is a four–seven times increase in risk for panic disorder. There is no increased risk for GAD in these individuals. Relatives of individuals with GAD have up to a five times increased risk of GAD and also an increased risk of alcoholism, but do not have an increased risk of panic disorder or agoraphobia. These effects are generally reduced if milder cases are examined and are less marked in general population studies than in hospital clinic studies.

Prevalence of the anxiety disorders

Anxiety disorders are very common. The National Comorbidity Survey (NCS), in the United States, found that 25% of the population will meet criteria

for an anxiety disorder at some time in their lives. The lifetime prevalence of GAD was 5% in this study. The Epidemiologic Catchment Area (ECA) study, also in the United States, demonstrated a prevalence of 3.8% for GAD over a 6-month period and of 8.5% over a lifetime. Panic disorder had a 6-month prevalence of 0.8% and a lifetime prevalence of 1.6% in the same study. In a UK study (Office of Population Censuses and Surveys [OPCS] General Household Survey – United Kingdom, 1995), rates of GAD of 5% and 4% in females and males, respectively, were demonstrated. The corresponding figures for Panic disorder were 1% for both sexes, and for 'mixed anxiety and depression' (when both anxiety and depression are present and are equally important), they were 10% for females and 5% for males.

Clinical assessment of the anxiety disorders

When assessing an individual who presents with anxiety symptoms, it is of course necessary to take a full history and to complete a mental state examination in order to clarify the diagnosis. A physical health problem presenting with anxiety symptoms should be excluded (and physical tests may be necessary in order to fully exclude such a possibility). As anxiety symptoms may occur in other psychiatric conditions, such as depression and psychotic disorders, it is important to exclude these disorders. If a diagnosis of an anxiety disorder is indicated, it is important to carefully differentiate between panic disorder, phobic anxiety disorders and GAD. One particular diagnostic conundrum is when symptoms of anxiety and depression are both present, but neither is clearly predominant, and neither type of symptom is present to the extent that it justifies a diagnosis if considered separately. In these circumstances, a diagnosis of 'mixed anxiety and depression' is justified. In the OPCS General Household Survey, 'mixed anxiety and depression' was found in 10% of females and 5% of males.

However, when both anxiety and depressive symptoms are present and severe enough to justify individual diagnoses, both diagnoses should be recorded and the category of mixed anxiety and depression should not be used. Taking a careful history can help establish if anxiety has followed low mood or whether low mood followed on from anxiety.

Generalized anxiety disorder

Diagnosis

GAD is defined as persistent (occurring on more days than not for more than 6 months) 'free-floating' anxiety, with at least four associated features from the following list: palpitations, sweating, trembling, dry mouth, difficulty breathing, feelings of choking, chest pain, nausea, dizziness, derealization, depersonalization, fear of losing control, fear of dying, hot flushes or cold chills, numbness or tingling, feeling keyed up, difficulty swallowing, irritability, poor concentration, disturbed sleep (early or middle insomnia), increased muscle tension and restlessness.

'Free-floating' anxiety is generalized and persistent but not restricted to, or even strongly predominating in, any particular environmental circumstance. Fears that the patient or a relative will become seriously ill or have an accident are often expressed.

Onset is usually in early adulthood. In younger age groups it is twice as common in females, but there are only slightly more female patients in older age groups. For most sufferers GAD is mild, but the illness course is often chronic, in particular for females. GAD is distinct from panic disorder and is often comorbid with other psychiatric disorders (such as OCD, social phobia and dysthymia).

Management of GAD

The anxiety disorders often go unrecognized. Many individuals do not consult with their GP as they do not realize the meaning and significance of their symptoms. This is particularly true of men. When individuals do present, the GP might not ascertain the true meaning of symptoms, such as breathlessness or chest or stomach pain. Misdiagnosis and inappropriate or inadequate treatment may thus result. The overall effect is that there are greater levels of social disability as a result of the anxiety disorders than is necessary, with a resulting high economic cost. Long-standing problems are often also more difficult to treat, as maladaptive coping strategies may have been employed over many years.

There are several other important issues when considering the assessment and treatment of patients with anxiety disorders. As a general rule you should be reluctant to make a new diagnosis of an anxiety disorder in an older patient – they may actually be

troubled by an underlying medical condition. Be aware of the possibility of a patient 'self-medicating' with alcohol or benzodiazepines. Also, do not forget the importance of patient motivation if a successful treatment outcome is to be achieved.

Pharmacological interventions are rarely appropriate as the first-line intervention for mild forms of anxiety, which can usually be managed in primary care with basic psychological interventions. Psychological approaches range from simple reassurance, especially from the GP, through self-help, including guided self-help, counselling and problem solving. More sophisticated psychological approaches, applicable to those individuals who are referred on to mental health services, include cognitive behavioural therapy (CBT) and 'anxiety management' (often carried out in a group setting).

VIDEO 7.2

Charles' story: generalised anxiety disorder – https://vimeo.com/7083823

If the disorder is more severe or chronic, a referral to mental health services is indicated. Combined treatment, that is pharmacological and psychological treatments together, is often best. The selective serotonin reuptake inhibitor (SSRI) antidepressants are the first-line pharmacological approach, although they may cause exacerbation of symptoms initially (because of their activating effect) and patients should be warned about this. Tricyclic antidepressants are also effective, but they are used much less often because of their side-effect profile and their toxicity in overdose.

Benzodiazepines should in general be avoided (because of their addictive qualities and because they quickly induce increased tolerance) although they can occasionally have a useful short-term role. Buspirone, a non-benzodiazepine anxiolytic with negligible sedative side effects and which is a partial agonist at the 5-hydroxytryptamine$_{1A}$ (5-HT$_{1A}$) receptor, is probably less effective than the benzodiazepines but provides another therapeutic option. Beta blockers, such as propranolol, are useful for somatic anxiety symptoms, such as tachycardia, sweating and tremor but do not treat the core anxiety symptoms. They are, perhaps, more useful in social anxiety disorder.

Panic disorder

Diagnosis

The essential feature of panic disorder (also sometimes known as episodic paroxysmal anxiety) is recurrent attacks of severe anxiety (panic), which are not restricted to any particular situation or set of circumstances and are therefore unpredictable. The key symptoms of panic attacks are explained in Box 7.4 and include sudden onset palpitations, chest pain, choking sensations, dizziness and feelings of unreality (depersonalization or derealization). There is often also a secondary fear of dying, losing control or going mad. The patient fears further attacks (phobophobia) and often changes his or her behaviour in relation to attacks.

The onset of panic disorder is usually in adolescence or in the mid-30s. New onsets after the age of 45 are rare. The illness course may be of varying severity. In younger age groups, there are twice as many female as male cases but this difference equalizes with age.

It has been established that there is an increased incidence (30%–50%) of mitral valve prolapse (MVP) in people with panic disorder (compared with perhaps 5%–15% in the general population). The reason for this association is not clear. There is not a simple relationship: MVP does not cause panic disorder and persists after panic disorder is treated. It is possible that MVP acts as an autonomic precipitant or that

Box 7.4 Panic attacks are characterized by acute development of several of the following symptoms, reaching peak severity within 10 minutes

- Escalating subjective tension
- Sweating, chills
- Chest pain/discomfort
- Palpitations, tachycardia
- Tremor
- Nausea
- 'Butterflies' in the tummy
- Dizziness/feeling faint
- Depersonalization/realization
- Dread, i.e. fear of loss of control or dying
- Paresthesiae (pins and needles)

both MVP and panic disorder are part of a syndrome of autonomic dysfunction.

Assessment and treatment

Panic may occur as part of other psychiatric conditions. For example, panic disorder should not be given as the main diagnosis if a patient has a depressive disorder at the time the attacks start – in these circumstances the panic attacks are probably secondary to depression. It is also important to exclude epilepsy and intoxication with or withdrawal from drugs or alcohol when assessing a patient who presents in such a way.

In general, assessment is essentially the same as for GAD. Again, combined pharmacological and psychological treatment is usually best. The SSRI antidepressants are recommended as a first-line treatment, although the tricyclic antidepressants have also been shown to be effective. Fast-acting benzodiazepines, such as alprazolam, may be used in the short term, but in general it is best to avoid these medications in panic disorder. If a patient is seen when he or she is actually experiencing a panic attack, a better strategy is to ask them to rebreathe their own air by the simple expedient of breathing into a paper bag. However, many patients (especially in such a state) may dislike the sensation of something over their mouth and should instead be encouraged to focus on emptying the lungs on expiration as hyperventilation is associated with rapid shallow breaths that do not allow adequate outbreaths.

Of the relevant psychological approaches, CBT has the best evidence for sustained improvement. Exposure therapy, relaxation techniques and self-help are also effective.

VIDEO 7.3

Tracy's story: panic disorder – https://vimeo.com/12051972

Obsessive–compulsive disorder

Diagnosis

The essential features of OCD are recurrent obsessional thoughts or compulsive acts or both together. Obsessional thoughts (or ruminations) are ideas, images or impulses that enter the patient's mind again and again in a stereotyped form. They are almost invariably distressing and the patient often tries, unsuccessfully, to resist them. They are recognized as his or her own thoughts, even though they are involuntary and often repugnant (common themes include fears of the person themselves acquiring disease or coming to harm in some way or of the person causing physical or sexual harm to others). Sometimes the dominant feature is an indecisive, endless consideration of alternatives, often associated with an inability to make trivial, but necessary decisions in day-to-day living. The result may be 'obsessional slowness'.

Compulsive acts or rituals are stereotyped behaviours that are repeated again and again. They are not inherently enjoyable and they do not result in the completion of inherently useful tasks. Their function is to prevent some objectively unlikely event, often involving harm to or caused by the patient, which he or she fears might otherwise occur. A patient might, for example, count up to a certain number repeatedly to ensure that his or her child is not involved in a car accident. Usually, compulsive acts are recognized by a patient as pointless or ineffectual and repeated attempts are made to resist them, but often to little avail. Family members can often become embroiled in the obsessional behaviour as initially this may appear to be helpful, but in the longer term this is rarely the case as the irrational nature of the person's obsessions becomes apparent to those around him. Anxiety is almost invariably present and if compulsive acts are resisted, the anxiety gets worse.

The majority of compulsive acts are concerned with cleaning (particularly handwashing), repeated checking to ensure that a potentially dangerous situation has not been allowed to develop (for example, returning repeatedly to the kitchen at night to ensure that all electrical appliances have been turned off) and orderliness and tidiness.

The presentation of OCD can vary considerably. Sometimes obsessional thoughts are to the fore, for example, upsetting and violent sexual thoughts regarding family members. Sometimes compulsions are to the fore, for example, endlessly checking the kitchen cupboards to ensure that the labels on every jar and tin face in the same direction.

Prevalence

For many years, OCD was considered to be a relatively rare condition, but it has become apparent that it may affect between 1% and 3% of the population

over their lifetime. However, many individuals do not present to services and view the behaviours as an integral part of who they are. Approximately half of those with OCD have another comorbid anxiety disorder. Rates in males and females differ in childhood with more males affected, but in adulthood rates are about equal. Age of onset is earlier in boys and is more often associated with physical tics. The course of OCD tends to be chronic and males have a worse prognosis in general.

Aetiology

The aetiology of OCD is complicated, but it is clear that it is at least in part a genetic condition and there is increasing evidence for underlying brain abnormalities. First-degree relatives of individuals with OCD have higher rates of anankastic/obsessional personality (explained in the next section, 'Assessment'), depression and OCD (the rate in first-degree relatives is 10%) than the general population. The OCD concordance rate between monozygotic twins is 50%–80%, whereas between dizygotic twins it is 25%.

Abnormalities of the serotoninergic system appear to play a role in OCD – studies have demonstrated hypersensitivity of postsynaptic serotonin receptors in some patients. OCD symptoms are particularly common in Gilles de la Tourette syndrome, a neuropsychiatric disorder characterized by multiple motor and vocal tics and sometimes coprolalia (the shouting out of swear words). Up to 80% of individuals with Tourette's have obsessional symptoms. Abnormalities of the basal ganglia may well play an important role in both OCD and Tourette syndrome: brain imaging has demonstrated abnormalities of brain circuits linking prefrontal cortex (especially the orbitofrontal region) and the striatum and thalamus. Overactivity of thalamocortical pathways may be the important mechanism.

Assessment

A thorough assessment is necessary to exclude various possible differential diagnoses. It should be remembered that obsessional symptoms often occur in childhood and sometimes in adulthood too, but this does not necessarily mean that a person has OCD. In research studies it has been demonstrated that up to 14% of the population have obsessions or compulsions.

Obsessional symptoms may occur in other psychiatric conditions, especially in schizophrenia and early dementia and other organic brain conditions. The relationship between obsessional ruminations and depression is particularly close and a diagnosis of OCD should be preferred only if ruminations arise or persist in the absence of a depressive episode. Some obsessional behaviours start as a way of managing anxiety but then become habitual, so that by the time patients present to services the picture may be quite confused.

Some people have premorbid anankastic or obsessional traits and can be considered to have an anankastic personality disorder. Such traits are characterized by doubt, perfectionism, excessive conscientiousness, checking, preoccupation with details, stubbornness, caution and rigidity. There may be insistent and unwelcome thoughts or impulses that do not attain the severity of OCD.

A diagnosis of OCD is appropriate if an individual has obsessional thoughts or compulsive acts for most days over a period of 2 weeks. The obsessions and acts need to have a number of features: they are recognized by the patient as arising from their own mind, they are repetitive and unpleasant, there is a degree of resistance to them, the thought or the act is not a pleasurable experience, the condition leads to some form of disability (with effects on relationships, work or family) and the symptoms are not related to a psychotic illness or a mood disorder.

Management

Serotoninergic antidepressants – the SSRIs and clomipramine – have proven anti-obsessional effects. Dopamine receptor antagonists may also be useful. In very extreme cases neurosurgery (anterior capsulotomy or subcaudate tractotomy) may be useful. More recently, a new technique called deep brain stimulation has been subject to clinical trials.

Various psychological interventions have a clear role to play in the treatment of OCD, including response prevention and graded exposure (for rituals), thought stopping (for obsessional thoughts) and CBT. In exposure and response prevention a therapist asks the patient to expose themselves to a particular situation that brings on anxiety and/or a compulsive act. The patient is asked to resist from engaging in that act for as long as possible and eventually the drive to act and the resulting anxiety decreases.

Reactions to severe stress and adjustment disorders

These disorders are identified not just by the presence of certain symptoms and the course of illness, as are the majority of psychiatric disorders, but also by a consideration of the aetiology or cause of the presenting problems. The presumed causes are either acute or chronic psychological stressors. One of the stressful events such as witnessing a violent death, or continuing stressful and unpleasant life circumstances, is considered to be the primary and overriding causal factor. The disorders in this section would not have occurred without the impact of stress and can thus be regarded as maladaptive responses to severe or continued stress, in that they interfere with successful coping mechanisms and therefore lead to problems with social functioning (that is, the ability to cope with day-to-day life).

Acute stress reactions

Diagnosis

The acute stress reactions are transient disorders that develop in an individual without any other apparent mental disorder in response to exceptional physical and mental stress, for example witnessing a murder, and which usually subside within hours or days.

An individual's vulnerability to stress and ability to cope play very important roles in the occurrence and severity of acute stress reactions. The symptoms typically show a mixed and changing picture and include an initial state of 'daze' with some constriction of the field of consciousness and narrowing of attention, an inability to comprehend stimuli and disorientation. This state may be followed either by further withdrawal from the surrounding situation (to the extent of a dissociative stupor, see Box 7.5 in the section 'Dissociative (conversion) disorders') or by agitation and overactivity (a flight reaction). Autonomic signs of anxiety (tachycardia, sweating, flushing) are commonly present.

The symptoms usually appear within minutes of the stressful event and disappear within 2–3 days (often within hours). Partial or complete amnesia for the episode may be present. If the symptoms persist, a change in diagnosis should be considered.

Acute stress reactions have been shown to occur in around 15% of the population at some time in their lives. The rate for individuals who have suffered a severe traumatic event is approximately 30%–40% (that is, the majority of individuals who have been exposed to such trauma do not experience an acute stress reaction).

Management

It is important to explore the event that precipitated the acute stress reaction. Most often no intervention is required or indicated as the disorder is self-limiting. Simple explanation and support may be helpful. Brief CBT may be helpful when an acute stress disorder is characterized by dissociation and PTSD-type symptoms. For some years, there was a trend to offer 'post-traumatic counselling' as soon as possible after an individual experienced trauma, in the hope that this would prevent the later development of problems such as PTSD. However, it is now recognized that this can be unhelpful. It is very important to allow individuals the time to make sense of events for themselves.

Post-traumatic stress disorder

Diagnosis

PTSD has been a diagnosis available to psychiatrists since the early 1980s. While the concept has only been widely accepted in recent times, it was becoming increasingly apparent over a long period of time that some individuals develop long-standing symptoms after a traumatic incident.

In the nineteenth century, 'railway spine' was often diagnosed when passengers were involved in railway accidents and presented with symptoms which we would now recognize as consistent with PTSD. Physicians of the time thought that such symptoms were due to the 'excessive speeds' of the trains. Wartime has a way of concentrating medical minds and often leads directly to advances in our understanding. It became increasingly apparent that soldiers were adversely affected in a psychological sense by the experience of conflict. In the American Civil War the preferred diagnosis was 'irritable heart', in the First World War 'shell shock' and in the Second World War 'combat neurosis'. Finally, largely thanks to the efforts of groups of ex-soldiers and innovative psychiatrists, and drawing on the experience of the Vietnam War, PTSD became established.

PTSD arises as a delayed or protracted response to a stressful event or situation (of either brief or long duration) of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive

distress in almost anyone. Predisposing factors, such as personality traits or previous history of psychiatric illness, may lower the threshold for the development of the syndrome or aggravate its course, but they are neither necessary nor sufficient to explain its occurrence.

Typical features include episodes of repeated reliving of the trauma in intrusive memories (flashbacks), dreams or nightmares occurring against a persisting background of a sense of 'numbness' and emotional blunting, detachment from other people, unresponsiveness to surroundings, anhedonia and avoidance of activities and situations reminiscent of the trauma. There is usually a state of autonomic hyperarousal with hypervigilance, an enhanced startle reaction and insomnia.

Anxiety and depressive symptoms are commonly associated with these symptoms and signs, and suicidal ideation is common. The onset follows the trauma with a latency period that may range from a few weeks to months. The course is fluctuating but recovery can be expected in the majority of cases. In a small proportion of cases the condition may follow a chronic course over many years, with eventual transition to an enduring personality change.

The lifetime prevalence for PTSD has been estimated in various studies as low as 1% and as high as 10%. For obvious reasons, certain groups are more likely to develop PTSD, such as soldiers, those who have been exposed to natural and man-made disasters, assault and rape victims and victims of torture. Between 30% and 40% of women who have been sexually assaulted will develop PTSD, as did 20% of American Vietnam War veterans. Around one-third of individuals with PTSD suffer from a severe form of the condition.

Three factors that help to predict whether a person develops PTSD are the scale of the trauma, the patient's previous experience of trauma and the level of social support available to the person. There may also be some genetic predisposition to PTSD. The hippocampus (responsible for memory) and the amygdala (the fear centre of the brain) may be oversensitive in people who develop PTSD. Magnetic resonance imaging scans have shown decreased hippocampal size in patients who develop PTSD.

Management

An initial period of engagement and the establishment of trust are important. Psychological treatment in the form of either trauma-focused CBT or what is known as 'eye movement desensitization and reprocessing' (EMDR) are recommended as the first-line treatments by the National Institute for Health and Clinical Excellence (NICE). EMDR is a somewhat controversial treatment that involves generating rapid and rhythmical eye movements while holding traumatic images in the mind.

The key component of trauma-focused psychological therapies is to expose the patient to the traumatic event repeatedly. This is often accomplished by getting them to write a detailed account of the traumatic event and recording it (a 'testimony'). The idea is that repeating the trauma will reduce symptoms as an individual becomes used to the experience. Although various non-specific psychological and social treatments, such as stress management and family and support groups can be helpful, trauma-focused treatments are more effective.

Pharmacological treatment, with a focus on the use of SSRI antidepressants, is the recognized second-line treatment. There is no role for routine prescription of benzodiazepines. Pharmacological treatments can of course be combined with psychological treatments, and often are.

Any comorbid conditions, including physical health problems, should be treated. Prognosis is good if the individual concerned had a healthy premorbid function, the trauma was brief and of limited severity, if there is no past psychiatric history, no family history of psychiatric illness and the individual has good social support. Approximately 65% of patients recover within 18 months.

Adjustment disorders

Adjustment disorders are states of subjective distress and emotional disturbance, usually interfering with social functioning and performance, arising in the period of adaptation to a significant life change or a stressful life event. The stressor may affect the integrity of an individual's social network (for example, bereavement or separation experiences) or the wider system of social supports and values (such as the experience of migration or becoming a refugee) or represent a major developmental transition or crisis (going to school, becoming a parent, failure to attain a cherished personal goal, retirement).

Individual predisposition plays an important role in the risk of occurrence and the shaping of the manifestations of adjustment disorders, but it is nevertheless assumed that the condition would not have arisen without the stressor.

The manifestations vary and include low mood, anxiety or worry (or a mixture of these), a feeling of inability to cope, plan ahead or continue in the present situation, as well as some degree of disability in the performance of daily routine. The predominant feature may be a brief or prolonged depressive reaction or a disturbance of other emotions or conduct. The prevalence of adjustment disorder is not known although it is clearly a very common condition.

Management of adjustment disorders

There is generally no need to intervene. If an individual is very distressed or is thought to be at risk, it is best to treat individuals for whatever symptoms dominate: PTSD treatments for PTSD symptoms or anxiety and depression treatments for individuals with predominant anxiety and depressive symptoms.

Dissociative (conversion) disorders

Individuals with dissociative disorders (also sometimes known as 'conversion hysteria' or simply 'hysteria' – older words which are still in use) present with a wide array of physical symptoms that are presumed to be psychological in origin. The more dramatic manifestations of these disorders are not seen very often, but when they are, they are seldom forgotten as the patient's presentation may be startling or even bizarre.

The dissociative disorders are presumed to be psychological in origin because of the frequently observed association between the symptoms and the timing of

Box 7.5 The main dissociative disorders and their presentation

F44.0 Dissociative amnesia

Loss of memory usually for important recent events is not due to organic condition. This is usually partial or selective and centred on traumatic events

F44.1 Dissociative fugue

This has all the features of dissociative amnesia plus purposeful travel beyond the usual everyday range. For example, a patient may turn up at a hospital many miles from where they live, apparently unaware of their origin. The patient's behaviour during this time may appear completely normal to independent observers

F44.2 Dissociative stupor

Profound diminution or absence of voluntary movement and normal responsiveness to external stimuli, such as light, noise and touch when examination and investigation reveal no evidence of a physical cause

F44.3 Trance and possession disorders

These are disorders in which there is a temporary loss of one's sense of personal identity but full awareness of one's surroundings. The diagnosis should only be made when such trance states are involuntary and should not be applied to trances which are religious or culturally appropriate

F44.4 Dissociative motor disorders

In the commonest varieties there is loss of ability to move the whole or a part of a limb or limbs. There may be close resemblance to almost any variety of ataxia, apraxia, akinesia, aphonia, dysarthria, dyskinesia, seizures or paralysis

F44.5 Dissociative convulsions

May mimic epileptic seizures very closely in terms of movements, but tongue-biting, bruising due to falling and incontinence of urine are rare, and consciousness is maintained or replaced by a state of stupor or trance

F44.6 Dissociative anaesthesia and sensory loss

Loss of sensation to an area of skin. The area of skin involved often has boundaries that make no anatomical sense and it is clear that they are associated with the patient's ideas about bodily functions. Loss of vision and hearing can also occur but are rarely total

F44.8 Dissociative identity disorder

Patients may complain of experiencing multiple different personalities – otherwise known as 'multiple personality disorder'. This is a somewhat controversial diagnosis though there is good evidence for its validity. There is a very strong association with childhood trauma

traumatic events, insoluble and intolerable problems or disturbed relationships. There is often evidence that the loss of function experienced is an expression of emotional conflicts or needs. The symptoms often appear suddenly and represent the patient's concept of how a physical illness would present rather than the actuality of how such an illness would present. The patient can often be rather indifferent to the problem presented and appear disassociated from the presentation. Medical examination and investigation do not reveal the presence of any known physical or neurological disorder.

Some dissociative disorders remit after a few weeks or months, particularly if the onset is associated with a traumatic life event. More chronic disorders, particularly paralyses and anaesthesias, may develop if the onset is associated with insoluble problems or interpersonal difficulties.

Only disorders of physical functions normally under voluntary control and loss of sensations are included in the dissociative disorders category. Disorders involving pain and other complex physical sensations mediated by the autonomic nervous system are classified under somatization disorder. Some of the many varieties of dissociative disorder are outlined briefly in Box 7.5.

Prevalence

The exact prevalence of the dissociative disorders is not known. We do know that people with PTSD, substance misuse, eating disorders and those who have been abused in childhood (sexually, physically or emotionally) have a high frequency of dissociative experiences or symptoms and many will meet ICD-10 criteria for dissociative disorders.

Risk factors

The aetiology of the dissociative disorders is not clear but it presumably is due to a psychological mechanism. There is no evidence of any genetic contribution. There is clear evidence that psychological trauma precipitates dissociative states.

Management

Initially, it is important to establish that there is in fact no underlying physical disorder that explains the patient's presentation. Patients will often arrive at the psychiatric clinic after months of thorough assessment at medical or surgical clinics or indeed after months or years of pointless medical or surgical treatment and by then any unusual physical illness will have been excluded. Long-term follow-up studies have demonstrated that even bizarre presentations may sometimes turn out to have an actual underlying pathology, however, and the possibility of the later appearance of serious physical or psychiatric disorders should always be kept in mind.

It is sometimes possible to ascertain the actual underlying psychological cause. If such a cause is identified, therapeutic efforts can be focused on this but even if no such direct cause can be pinpointed, psychological therapy, usually in the form of long-term psychotherapy, often helps.

Somatoform disorders

Somatization refers to 'physical symptoms suggesting physical disorder for which there are no demonstrable organic findings or known physiological mechanism and for which there is positive evidence, or a strong presumption, that the symptoms are linked to psychological factors or conflicts'. Somatoform disorders are at the extreme end of the somatization spectrum.

The main feature of the somatoform disorders is repeated presentation of physical symptoms together with persistent requests for medical investigations, in spite of repeated negative findings and reassurance by doctors that the symptoms have no physical basis. If any physical disorders are present, they do not explain the nature and extent of the symptoms or the distress and preoccupation of the patient.

A differential diagnosis for these conditions, and for the conversion disorders, is simple malingering, where an individual is feigning symptoms to avoid an adverse consequence, for example, loss of a relationship or criminal proceedings.

Somatization disorder

The main features of somatization disorder are multiple, recurrent and frequently changing physical symptoms of at least 2 years' duration. Most patients have a long and complicated history of contact with both primary and specialist medical care services, during which many negative investigations or fruitless exploratory operations may have been carried out. Symptoms may be referred to any part or system of the body. The course of the disorder is chronic and fluctuating and is often associated with disruption of social, interpersonal and family behaviour.

Hypochondriacal disorder

The essential feature is a persistent preoccupation with the possibility of having one or more serious and progressive physical disorders. Patients manifest persistent somatic complaints or a persistent preoccupation with their physical appearance. Normal or commonplace sensations and appearances are often interpreted by patients as abnormal and distressing, and attention is usually focused upon only one or two organs or systems of the body. Marked depression and anxiety are often present and may justify additional diagnoses.

Somatoform autonomic dysfunction

Symptoms are presented by the patient as if they were due to a physical disorder of a system or organ that is largely or completely under autonomic innervation and control, that is the cardiovascular, gastrointestinal, respiratory and urogenital systems.

The symptoms are usually of two types, neither of which indicates a physical disorder of the organ or system concerned. First, there are complaints based upon objective signs of autonomic arousal, such as palpitations, sweating, flushing, tremor and expression of fear and distress about the possibility of a physical disorder. Second, there are subjective complaints of a non-specific or changing nature, such as fleeting aches and pains, sensations of burning, heaviness, tightness and feelings of being bloated or distended, which are referred by the patient to a specific organ or system.

Persistent somatoform pain disorder

The predominant complaint is of persistent, severe and distressing pain, which cannot be explained fully by a physiological process or a physical disorder, and which occurs in association with emotional conflict or psychosocial problems that are sufficient to allow the conclusion that they are the main causative influences. The result is usually a marked increase in support and attention, either personal or medical.

Prevalence

Somatization is common among the general population – in one study in the United States, 4.4% of a community sample complained of significant unexplained somatic symptoms. The prevalence of somatoform disorders, however, is much lower, perhaps as low as 0.03%. In general, somatoform disorders are more common in women.

Risk factors

Genetic studies in this area have not provided any clear answers. Childhood experience does, however, appear to play an aetiological role. A number of studies suggest that adults who somatize have mothers who somatize or have parents who in fact are or were ill. The process of continuously seeking medical reassurance may reinforce the associated behaviour and thus perpetuate the disorder. It is important to note that patients with depression may present with somatic symptoms as their primary and perhaps the only symptom. This is not uncommon in patients from South Asia.

Assessment

Full assessment is vital in these conditions to exclude any physical problem, but it is not helpful to continue investigation when such investigations are no longer clinically indicated.

Management

The starting point is making the link between physical symptoms and psychological distress. Treatment is then psychological rather than pharmacological. Psychological intervention is more likely to be successful if a patient accepts that psychosocial factors are making an important contribution to their clinical problem. Early treatment is also more likely to be successful (within 2 years of the onset of symptoms).

Summary

Anxiety disorders are very common – perhaps a quarter of us will suffer from such a disorder at some time in our lives. However, many of these conditions remain undiagnosed and untreated. Increased awareness among all medical practitioners will help to alleviate unnecessary suffering as the anxiety disorders are amenable to both pharmacological and psychological treatments.

The other disorders described in this chapter can broadly be categorized as disorders that present with unexplained medical symptoms, often with anxiety as a prominent clinical feature. Patients with these

disorders usually present to GPs, physicians and surgeons before they present to psychiatrists. When such patients are referred to psychiatrists they may be reluctant to accept that their symptoms are psychological in origin. If a patient comes to accept that this may be the case, then psychological intervention may well help to resolve their symptoms.

CASE STUDY 7.1

A 30-year-old woman describes episodes of severe anxiety that occur unpredictably, but frequently and regularly. During attacks she experiences acute breathlessness, chest pain and palpitations. She frequently attends casualty departments as she fears that she is having a heart attack.

What is the most likely diagnosis? Would you carry out physical investigations? What treatment would you initiate?

The most likely diagnosis is panic disorder. If the anxiety symptoms were suffered more or less continuously then the diagnosis would be GAD.

While patients such as the one described here often fear a heart attack, it is usually relatively easy to exclude such a possibility on clinical grounds. Often, however, some limited physical investigation (carrying out an electrocardiogram [ECG], for example) may be helpful when a patient first presents, both to reassure the patient and to absolutely exclude physical pathology.

Simple explanation and reassurance are the starting point – such measures can have a dramatic and immediate effect for a patient who has come to casualty fearing the worst. Whether a referral is made to mental health services depends on the severity and chronicity of symptoms. An SSRI antidepressant is the most common pharmacological treatment, either given alone or combined with a psychological treatment. It is recommended that a psychological treatment, often CBT, is tried first in the majority of cases.

Further reading

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CASE STUDY 7.2

A 45-year-old man presents to mental health services stating that he cannot see. He has been extensively assessed by neurological specialists who state that there is no evidence of any underlying physical pathology. When questioned closely he reveals that he had been in some trouble over a sum of missing money at his workplace just before the onset of his difficulties.

What is the most likely diagnosis? What is the most appropriate approach – an SSRI antidepressant or long-term psychotherapy?

This is a case of dissociative or conversion disorder. Some clinicians would still use older terminology and would call this 'hysterical blindness'. As in this case, patients with conversion disorders often spend many months, or even years, being investigated by various specialists before it becomes clear that there is no underlying physical pathology.

An antidepressant will not help with the underlying problem, though it is possible that depression may develop in such an individual as a secondary feature and may require such treatment. Long-term psychotherapy is much more likely to prove fruitful. In psychotherapy the therapist explores the underlying causes of a patient's symptoms and hopes to resolve the symptoms by helping the patient to understand more about themselves.

CHAPTER 8

Phobias

RICHARD DAY

KEY CHAPTER FEATURES

- Definition of phobic anxiety
- Phobic disorders, risk, epidemiology, assessment and management
- Useful questions to elicit phobic symptoms
- How to assess severity of phobic symptoms
- How to differentiate phobias from other causes of anxiety

Introduction

This chapter begins by defining phobias before considering the risk of developing phobias, their epidemiology and assessment. Some attention is then paid on how to identify symptoms and their severity and differentiate phobias from other anxiety disorders.

There are times when it is very appropriate and useful to be anxious – if a hungry and angry tiger burst into the room as you are reading this chapter, it would not be helpful to your long-term survival to calmly place a bookmark in this page and then survey the room to consider your options for escape (perhaps you could offer to make it some tea?). Instead, you would feel fear; your sympathetic nervous system would be activated in the 'fight or flight' response and you would quickly respond to the dangerous situation with prompt activity (probably this would involve running away fast but that may depend on your previous experience of facing such threats!). We would consider this a normal response to a threatening situation.

What, however, if you repeatedly showed the same response, and the same degree of response, to the sight of a small, harmless spider on the other side of the desk? Or to travelling over a bridge (an entirely safe and structurally secure bridge)? Or to finding yourself in a busy supermarket? Or to eating in public? This situation, where a person experiences a degree of anxiety or panic that is predictable (i.e. happens in recognizable circumstances), is far in excess of what is necessary (i.e. disproportionate) and this causes a significant emotional distress to the individual is what is meant by the term phobia.

EXERCISE 8.1

Have you ever used the word 'phobia' or 'phobic' to describe yourself or have you heard others use the word? What did you (or they) mean by the word?

What behaviour or feeling was being described by the use of the word 'phobia'?

Aside from a description of an illness, can you think of other medical or non-medical uses of the suffix-phobic, e.g. xenophobic?

How does your own use and general use of the word 'phobia' relate to its use as a description of a particular mental health problem?

We can all be excessively anxious about different things and it can be difficult to distinguish between this 'normal' anxiety and phobic anxiety. Does this lack of clear-cut distinction bother you? Are you more comfortable with the definition of hypertension? In what ways do the definitions of hypertension or obesity have similar limitations to that of anxiety? It may be useful to revisit Chapter 1 in which we discussed the problems that may arise when the line between what is a disorder and what is not are unclear.

Types of phobias

Phobic situations are divided into three different types depending on the nature of the feared situation: agoraphobia, social phobia and specific phobia.

1. In agoraphobia, the anxiety is caused by being in a busy situation or an open area where there is no immediate possibility of escape to a 'safe' area (the 'agora' in ancient Greece was an open area where crowds would gather together and was also used as the busy marketplace).
2. For social phobia, the anxiety is a 'fear of scrutiny' by others, associated with being the focus of attention and being embarrassed or humiliated. Typically relatively small social settings are feared, such as eating with others, speaking in a small group setting or even simply social interaction with another person who is not well known.
3. Specific phobias are many and varied but all involve a very specific situation that is the cause of fear: a particular type of animal, the sight of blood or injury, darkness, flying, etc. Most specific phobias usually fall into one of the following six groups: fear of animals (usually relatively small animals and 'creepy-crawlies'), heights, water, enclosed spaces (claustrophobia), dental treatment and blood/injury/injection.

Prevalence

In terms of lifetime prevalence in the general population, agoraphobia as a primary diagnosis is less common (1%–8%) than social phobia and specific phobias which are the two most common anxiety disorders (both about 2%–14%).

Agoraphobia tends to present in young adults (50% presented by the age of 20 years and 75% by the early thirties). In contrast, social and specific phobias present in childhood or soon afterwards – 75% by early adolescence and 90% by the age of 23 years.

Risk factors

It is not very well understood why some people develop phobias and others do not. There is evidence for phobias having a genetic element in that rates are increased in family members of someone affected (a relative risk of about 2:3) and monozygotic twins (genetically identical) are more likely to be either both affected or both unaffected when compared to dizygotic twins (sharing only 50% of genetic material).

Several social factors are associated with phobias – those who are married have lower rates of phobia than the unmarried, divorced or widowed; being unemployed, having a low income and low educational levels are associated with higher risk of phobia. It is not clear though which is the cause and which is the outcome, that is having a phobia may make an individual less able to form close and sustained relationships, less able to do 'normal social' things and adversely affect their education and employability.

Agoraphobia

Agoraphobia is often related to panic disorder. A person can begin to experience unpredictable panic attacks and, as a result of this, subsequently develop agoraphobic symptoms whereby they fear having a panic attack while out, and therefore avoid going out. However, it is possible to develop agoraphobia without previously

having panic attacks. It is not clear why some people with panic disorder develop agoraphobia and others do not – but those who do develop agoraphobia tend to have higher levels of premorbid anxiety as children, depression and more dependent or avoidant personality traits. They also tend to have lower perceptions of self-efficacy and self-dependence. It is about three times as common in women as in men.

Social phobia

Social phobia has previously been an overlooked and underestimated diagnosis. In many people it goes unnoticed and untreated and they are merely thought of as being 'shy'. Although people who are very shy are more likely to have a social phobia than people who are not shy, very shy people may also not have a social phobia but instead have other psychiatric diagnoses. Also, people may develop a social phobia without the presence of shyness in their premorbid personality. People who are shy are unlikely to have the presence of symptoms of anxiety or panic about social contexts in general (although they may be nervous about specific issues such as meeting new people or having to speak out) and less likely to have anticipatory anxiety. In shyness the degree of anxiety can vary as with any other personality traits. People with social phobias are also more likely to perceive other people as potentially negative and critical.

Previous prevalence rates of 2%–3% have now been increased to 12%–13% as a result of more careful evaluation. It tends to present at a very early age (i.e. at school) with a slight female preponderance. It is a risk factor in children for poor school performance and school refusal and in adults for poor work performance, unemployment and low socio-economic status. There is some evidence that there are higher rates in children who have experienced either parental rejection or overprotection but no clear relationship to overall childhood adversity.

More specifically, social phobia has been linked to a heritable personality trait known as behavioural inhibition. This is a tendency to react to novel situations by avoidance and withdrawal to safety (i.e. the caregiver) with the expression of distress (crying or fretting). It is evident early in childhood (toddlers and preschool) and has been associated with the later development of anxiety disorders in general and social phobia in particular.

Functional neuroimaging indicates increased bilateral activation of the amygdala and increased regional cerebral blood flow to the amygdala and related limbic areas that normalizes on successful treatment (whether selective serotonin reuptake inhibitor [SSRI] or cognitive behavioural therapy [CBT]).

Specific phobia

For specific phobias, explanations of aetiology now generally use learning theory or evolutionary explanations rather than assuming unconscious and traumatic psychodynamic factors related to early childhood.

It has been hypothesized that a person may develop a specific phobia after exposure to a particular stimulus or situation that has been associated (by classical conditioning) with trauma and anxiety. This situation is then feared and avoided. If the person subsequently is exposed to the same feared situation they begin to feel anxious again and escape from the situation. This leads to a reduction in their level of anxiety, which thus serves to reinforce the fear of that specific situation. Similarly, if they are faced with the prospect of the feared situation they begin to feel anxious and if they manage to then avoid the situation they again experience a reduction in anxiety which reinforces their avoidance behaviour.

This may explain some specific phobias, but many people with a phobia cannot remember any earlier traumatic experiences and it does not explain why we tend to develop specific phobias to only a relatively small range of situations.

Evolutionary theories are based on the observation that most of the common specific phobias are focused on situations that could in the past hundreds of thousands of years have posed a significant threat and that therefore the phobia is some kind of evolutionary adaptation to increase survival. However, this theory is difficult to prove and does not explain why we have evolved a tendency to be phobic of small 'creepy-crawlies' such as spiders but not of larger predators that could be just as dangerous and threatening to personal survival (nor were there many dentists around 100,000 years ago).

There may well be multiple explanations for how people develop specific phobias and there is also some evidence that some people might be more prone to develop phobias by, for example, being more aware

of bodily sensations and therefore more likely to link the somatic manifestations of anxiety with particular circumstances. Children's fears differ in nature across different ethnic groups. Culturally mediated beliefs, values and traditions may play a role in their expression and what is identified as a phobia or not.

Presentation

It is helpful to consider the effects of phobic anxiety in the three areas of cognitive (psychological) symptoms, behavioural responses and somatic (physical) symptoms. Phobias are associated with autonomic (sympathetic) nervous system activation with a large number of consequent physical symptoms (see Table 8.1).

Cognitive symptoms

The cognitive symptoms, as well as the experience of anxiety itself, often involve fear of losing control, fainting or even dying. The person may experience derealization or depersonalization. In derealization a person experiences their external environment as in some way unreal, for example people and objects may appear to be like 'cardboard cut-outs' or like actors being watched on a TV screen. It is as though they have stepped out of their environment. In depersonalization, it is the individual themselves who feel that they are in some way distanced or remote from everyone else around them, as if they were 'not really there' or a passive observer of what is happening rather than being actively involved in the proceedings – they can feel this even while, in reality, they are actively involved in what is happening. In this experience, it is as though they have stepped outside of themselves. It is important not to confuse these presentations with features of psychosis.

Behavioural symptoms

The major behavioural symptoms are either to hurriedly exit the feared situation (e.g. to rush out of a busy supermarket leaving the shopping trolley in an aisle with the shopping in it) or marked avoidance of the situation itself (so avoidance of the supermarket altogether). Thus, a person with agoraphobia may get a relative or friend to do their shopping in busy places, go themselves at very quiet times, often only if accompanied by a trusted other person, or happily use online shopping; a person with a specific phobia of bridges may take a very convoluted route in order to avoid going over a bridge. The impact of the coping strategies, for example avoidance behaviour, may be what leads the patient and/or family to seek help.

In severe forms, agoraphobia can result in a person becoming virtually housebound or a person with social phobia may avoid all contact with people beyond their immediate social circle. Avoidance of the feared situation can also lead to avoidance of other things that might possibly lead to exposure to the

Table 8.1 Presentation of the somatic, behavioural and cognitive features of phobias

Somatic/physical	Behavioural
Dry mouth	Escape from the feared situation
Feeling of choking	Avoidance of the feared situation
Difficulty in breathing	
Chest pain/chest discomfort	**Cognitive**
Palpitations/pounding heart/accelerated heart rate	Feeling that objects are unreal (derealization) or that self is distant or 'not really here' (depersonalization)
Nausea/abdominal distress, e.g. churning	Fear of losing control, 'going crazy' or passing out
Sweating	Fear of dying
Trembling/shaking	
Hot flushes/cold chills	
Numbness/tingling sensations	
Feeling dizzy, unsteady, faint or lightheaded	

feared situation; thus, someone with a phobia of spiders may go to such great lengths to avoid spiders that they find themselves unable to search in the corner of a dark cupboard because there *might* be a spider there. The avoidance could also mean that they are unable to watch a video of a spider, look at a picture of a spider or even talk about spiders.

Typically, the fear and associated symptoms can also be felt even at the *thought* of being exposed to the phobic situation. This is called anticipatory anxiety. Thus, a person with social phobia may become very anxious at the prospect of having to give a small group presentation in several weeks' time. Someone with agoraphobia becomes anxious even if they talk about perhaps going out sometime in the future and the ensuing distress may lead them to opt out of treatment.

A characteristic of phobic anxiety is that the person affected recognizes that their fear and the associated avoidance are excessive and unreasonable but is nevertheless unable to overcome them. Someone with a bridge phobia will be able to acknowledge that a bridge is safe and that there is minimal risk in walking over it. They will be able to agree that other people are perfectly correct to walk over it without giving it a thought; but still, if they approach the bridge themselves, they become so overwhelmed with anxiety that they are unable to cross over it.

Phobic anxiety is also characterized as being manifest only, or mainly, in the feared situation or when thinking about the feared situation. That is, there is not the more pervasive anxiety about lots of different things as in generalized anxiety disorder nor do the bouts of fear occur unpredictably, such as in panic disorder.

The emotional distress felt by the person can be directly as a result of the symptoms themselves or a consequence of the avoidance of the feared situation. In fact, the consequences of phobic avoidance are often the biggest disruption to a person's life. They may place significant limitations on what someone is able to do because of what has to be avoided. If a person has a severe agoraphobia it will be almost impossible for them to work unless they have a job where they can virtually work from home. Think of yourself as a medical student – would it be easy for you to complete your studies if you had a social phobia such that you found it almost impossible to give group presentations, to perform an aspect of history taking or physical examination in front of a group of your peers?

Somatic symptoms

These are as for anxiety and shown in Table 8.1.

Agoraphobia

Agoraphobia is relatively rare on its own and much more commonly develops in association with panic disorder. Typically, agoraphobia develops as a result of repeated unpredictable panic attacks such that the affected person begins to avoid going outside for fear that they might have a panic attack in public. There is often focus on the physical symptoms experienced and their feared effects. The focus of anxiety is a fear that they will occur while the person is out in an area from which there is not easy escape rather than the fear being focused on a particular location or setting. There is typically fear or avoidance of crowds and public places, travelling alone or travelling away from home.

Co-morbid rates of depression are very high, as are substance misuse (including alcohol and benzodiazepine dependence) and other anxiety disorders.

Social phobia

In social phobia, the fear is of being the focus of attention from other people and fear of behaving in such a way as to be socially embarrassed or humiliated. This is associated with avoidance of situations in which the person might be the focus of attention. Social phobia can be limited to specific forms of social scrutiny such as eating in public, speaking in public, meeting known people or entering small groups such as parties, meetings or classrooms. It can also be a more generalized fear of all or most of these different social situations.

Someone with social phobia will exhibit some of the somatic and cognitive symptoms of anxiety described earlier, including, at least one of the following:

- Blushing or shaking
- Fear of vomiting
- Urgency or fear of micturition or defaecation

Co-morbidity with other psychiatric diagnoses is common, particularly with other phobic disorders and panic disorder, but also with depression and alcohol misuse (about 20% risk).

Specific phobias

A major consideration with specific phobias is that although a very large number of people (up to 60%) may have a degree of phobia about a specific situation, in that they exhibit an exaggerated fear that they recognize as such, the vast majority of these do not cause significant distress or disruption to ordinary life and so can be considered to be subclinical. However, a minority of phobias do interfere with everyday life to such an extent that they require treatment. This may be either because of the severity of the distress and avoidance or because of the frequency with which the feared situation would ordinarily be encountered. For example, a severe blood/needle phobia may be much more problematic (and therefore require treatment) for a medical student than a student studying accountancy.

In particular, exaggerated anxiety in certain situations is very common in children, but in the majority of cases this phobic anxiety spontaneously reduces with age and does not cause a problem in adulthood. It would be unusual though for a specific phobia to emerge in adulthood if it had not been present as a child or adolescent.

As with the other phobias, there is commonly co-morbidity with other anxiety disorders in specific phobias but less commonly with depression or substance misuse.

Assessment

The key aspects of assessment in phobias are: to establish whether phobic anxiety is present; if so, then to identify the nature of the feared situations; to look for any other associated psychiatric symptoms; and to consider the variety of treatment options (including beginning to identify the underlying beliefs and assumptions that may be targeted in any cognitive therapy).

To establish the phobic nature of the anxiety it is necessary to confirm that the anxiety is episodic and occurs relatively predictably upon exposure (or the thought of exposure) to a small number of situations. Or alternatively, that those situations are so well and habitually avoided that very little anxiety is actually experienced.

Enquiry about what situations are feared and avoided and what it is about those situations that is feared should help to distinguish between agoraphobia, social phobia and specific phobias. For example, three people might say that they are fearful of meeting their friend in the local shopping centre but for different reasons. When asked what it is they fear about going to the shopping centre, the first may say that it is because it is very busy there and they fear that they will have a panic attack or faint (agoraphobia); the second may say that it is because they will have to go with their friend to one of the cafes for a coffee and they get very anxious eating or drinking when there are other people around (social phobia); the third may say that it is because the only way to get into the shopping centre is to use one of the lifts and they always get very panicky in lifts (specific phobia).

Phobic anxiety may also present secondary to a number of other psychiatric diagnoses. Enquiry about the time course of the phobic anxiety in relation to other symptoms will help to clarify whether the phobia is a primary problem or merely a manifestation of the other diagnosis.

Assessment of co-morbidity

A depressive illness can result in the development of phobic anxiety. This may happen without any history of previous phobic anxiety or it can occur when a relatively minor degree of phobic anxiety is exacerbated to the extent that it causes a significant amount of impairment. In this case, there will be evidence of depressive symptoms that predate the phobic anxiety. There may be a history of previous episodes of phobic anxiety (associated with previous depressive episodes) but there should not be a lifelong history of clinically significant phobic anxiety. Alternatively, depression can occur in someone who has phobic anxiety and in this circumstance the long-term and early-onset nature of phobic symptoms should be evident together with more recent development of depressive symptoms.

It is also commonly co-morbid with obsessive compulsive disorder and substance misuse problems. As with depression, it can be difficult to distinguish which has come first.

It is also important to avoid confusion with other disorders in which patients may demonstrate avoidant behaviour because of psychotic symptoms or long-standing personality traits, for example in patients with schizophrenia and paranoid delusions or in those with schizoid personalities.

Other key features to assess

One consideration in the assessment of phobias is the degree of distress and loss of function caused by the symptoms. Thus, someone in Britain who has a specific phobia of spiders may experience much more distress and interference with everyday life than someone with a similarly severe (in terms of symptoms) specific phobia of scorpions.

Another consideration is how motivated a person is to engage with and persevere with the various treatment options. The degree of anxiety may be so great that a person is unable or reluctant to engage with treatment because they expect that the very nature of that treatment (which will involve thinking about and ultimately encountering the feared situation) will be unbearable and too stressful.

Careful questioning will help to identify some of the false assumptions that are operating in that person's thoughts. Asking questions such as 'What is it you most fear happening if you …? and 'What would that mean?' may help the person to verbalize their automatic thoughts that will need to be challenged in subsequent cognitive therapy. So, someone with a social phobia may describe that they fear embarrassing themselves socially, that they have an image of how they ought to perform but are convinced that they will fail to reach that degree of performance and that others are judging them in similarly negative ways.

General principles for the management of phobias

There are some general principles of treatment for phobias. For the reasons already stated, a person may be reluctant to engage with treatment if they are too fearful of confronting their feared situation. Careful assessment with empathy can be necessary to elicit these concerns, discuss treatment options and highlight the options that may be least unacceptable, given a patient's concerns. If the overall assessment has identified other associated problems (such as depression), then this should be specifically treated as well and it may well be beneficial to treat depression before embarking on trying to treat the person's phobia.

Treatment for phobias can be psychological, pharmacological or a combination of both. The selection of treatment will depend on a combination of factors such as patient choice (an individual may have a strong preference for a particular treatment); availability of treatment (e.g. a 9-month waiting list for CBT); safety (there may be absolute or relative contraindications to particular drug treatments); psychological mindedness (some people struggle to make sense of what they are expected to do in psychological treatments and so are less able to make good use of them).

Psychological treatments

Useful psychological treatments are CBT and behavioural therapy. A number of behavioural techniques can be used and these generally involve the patient being exposed to the feared situations together with help to control their anxiety and relax. The exposure can be in real life (actually going to a shop, eating under the scrutiny of others, crossing a bridge) or can use imagery (simply thinking about or visualizing the feared situation while sitting in the therapist's office). It can involve exposure to the most feared situation in the first instance or can use a hierarchy of increasingly feared things to build up gradually to the most feared situation. Table 8.2 shows the different psychological approaches to treating phobias.

Thus, someone with agoraphobia may be exposed to a busy supermarket on a Saturday afternoon directly (an example of flooding) or may gradually gain confidence in less anxiety-provoking situations first: standing at the front door; going to the gate of their property; walking a short distance along the street; walking round the block; visiting the corner shop; going into town early on a quiet weekday morning, etc. (gradual exposure). Similarly, someone with a specific phobia of spiders may start with a small, simple drawing of a spider; then progress to a photograph; then to a video recording; then to a dead spider; then a very small live spider; then a larger one.

At each stage, the person continues in the situation until their anxieties have reduced. This can happen either as a natural process of extinction (i.e. fear

Table 8.2 Cognitive and behavioural therapy approaches to treating phobias

Technique	Form of exposure	Rate of exposure	Relaxation management
Flooding	Real or imagery	Sudden	No
Graded exposure	Real	Gradual	Yes
Systematic desensitization	Imagery	Gradual	Yes
Cognitively delivered exposure	Real or imagined real	Sudden or gradual	Yes

tends to subside over time as no harm comes to the person by being in the feared situation) or by the use of relaxation techniques and anxiety management such as progressive muscular relaxation and breathing retraining. Cognitively based exposure techniques (cognitive remodelling) additionally involve getting the patient to enter the feared situation and test their feared predictions. Exposure can be done by the patient on their own, as 'homework', or accompanied by the therapist.

Pharmacological treatments

Pharmacological treatments include the four main groups of antidepressants – SSRIs, SNRIs, tricyclics and monoamine oxidase inhibitors. There can be problems with initial exacerbation of anxiety with the start of drug treatment and also, given that drug treatment will need to be long term, adverse effects such as weight gain and sexual dysfunction can limit tolerability. Subsequent reduction or discontinuation of drugs may result in a relapse of the anxiety, and so any reduction should be done gradually with careful monitoring of the re-emergence of symptoms.

If antidepressants are used, simple gradual self-exposure can be encouraged for whatever behaviours do not spontaneously improve. If psychological and pharmacological treatments are combined, then one way of doing this is to commence medications first of all, to reduce intensity of the phobia, and then use psychological treatments to further improve symptoms.

Agoraphobia

Pharmacological and psychological therapies are both reasonable options in agoraphobia and they can be used effectively together. SSRIs tend to be the medication of choice. Medication will be particularly appropriate if there is co-morbid depression.

Psychological treatments may well use elements of graded exposure and systematic desensitization but would typically include a significant cognitive element that seeks to identify, challenge and remodel unhelpful, automatic thoughts.

Social phobia

As with agoraphobia, there is the option of using antidepressants but pharmacological treatment usually needs to be long term (with relapse common if medicines are stopped). It can take 2–6 weeks for benefits to become apparent, with further improvement over 12–24 weeks. Again, antidepressants can be very helpful to treat any co-morbid depression or to improve symptoms to a level where psychological treatments are easier.

Psychological therapies are usually cognitively based rather than simply being behavioural and incorporate elements of progressive muscular relaxation, imagined and real-life exposure, cognitive restructuring and also social skills training. People with social phobia often display cognitive biases. They tend to interpret ambiguous information on their performance negatively, to focus their attention on finding and attending to negative information, to be unable to recall positive information about their performance and to react catastrophically to mild negative feedback.

People who have relatively mild social phobia can sometimes gain benefit from beta blockers (which reduce the somatic manifestations of anxiety without really affecting the cognitive feelings), which can be taken before feared situations on an 'as required' basis. Similarly, a benzodiazepine can be used as an anxiolytic on an occasional basis with minimal potential for tolerance and the development of dependence.

Specific phobia

The treatment of specific phobia is almost exclusively psychological with behavioural methods being well established as effective treatments. Depending on the severity of the phobia, behavioural methods can even be effective in a single session. It is also possible to use cognitive techniques to get patients to test out their erroneous and fearful beliefs in the avoided situation.

Summary

Phobias are relatively common but they usually only present for treatment when they interfere with a person's life. Phobias can have varying impacts on the lives of individuals and often individuals and their families have learnt to work around the phobia. Phobias may present with physical and/or psychological symptoms. Phobias, especially specific phobias, are amenable to treatment but require patients to be motivated sufficiently well to follow through treatment programmes.

Further reading

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CASE STUDY 8.1

Susan is a 24-year-old legal secretary. She presents with a 3-month history of variable low mood and tiredness. She is doing less in her evenings and weekends because she is too tired and 'cannot be bothered'. Despite her tiredness her sleep has also deteriorated and she now has very unsettled nights.

She is very stressed at work because she is increasingly being expected to attend dinners and luncheons. She says that she just can't bear eating in front of other people and has so far always made excuses for not attending and once or twice has even stayed off 'sick' during the day in order to avoid going to the dinner at night. She is worried that her boss is dissatisfied with her performance because of this.

In the course of enquiry about other anxieties, she also mentions that she cannot bear the sight of spiders and if she sees one she has to catch it with a glass and throw it outside as soon as possible.

What would you include in the list of differential diagnoses for Susan?

Depressive episode: she describes a number of symptoms of depression with onset over the past 3 months.

Social phobia: the fear of and avoidance of eating in public together with the recognition that this is impacting her effectiveness at work raise the possibility of a social phobia diagnosis.

Specific phobia: the dread of spiders may indicate a specific spider phobia.

Generalized anxiety disorder needs to be considered.

Hypothyroidism: the tiredness and apathy warrants this in the list although it is unlikely.

What questions would you ask to try to determine if she has a social phobia about eating in company?

- Is the anxiety only specific to such situations?
- Does she feel generally anxious at other times or get fearful at unexpected times?
- When she eats on her own does she feel all right?
- Can she attend meetings where she does not have to eat? Does she avoid eating in public situations?

(*Continued*)

CASE STUDY 8.1 (*Continued*)

- Ask what specific situations she is fearful of and what she fears will happen in these situations.
- Does she recognize her fears as irrational?
- Are her fears associated with any physical symptoms (e.g. blushing or shaking, fear of vomiting, urgency or fear of micturition or defaecation)?
- How long has she had these fears – have they developed in recent months or been present for years (e.g. since adolescence)?

She subsequently gives a 10-year history of social phobia for eating but has never had problems with depression before now. She says that she 'can't go on feeling like this' especially as she has an annual appraisal in 6 weeks' time. She had never realized that there was anything that could be done to help with her fears regarding eating but is keen to 'try anything' otherwise, she thinks she may lose her job.

What information would you need to elicit in her history to work out how her apparently recent depressive illness relates to her probable social phobia?

What are the timelines of both sets of symptoms? Have her fears of eating in public been present for a long time and merely worsened since the development of depressive symptoms? Did they worsen first, lead to worsened social and occupational function or stress, prior to the development of depressive symptoms?

How would you decide if she also has a specific phobia of spiders?

There is clearly a fear of spiders but does it lead to avoidance or anticipatory fear? It sounds as though she is able to effectively deal with any spiders that she sees – is there significant distress or inability to function associated with her fears?

What will you tell her about treatment options?

Explain that there are both psychological treatments and medicines available to help; that psychological treatments require effort on her part, in between appointments, to put into practice what is discussed; and that medications may be useful to help initially with her depressive symptoms but that there may be side effects with them. She should know that it is important for her to be involved in the decisions made about what treatments to proceed with.

If you suggest that you can prescribe drugs, what will you tell her about possible side effects and the time course?

She needs to know that the effect of any drugs used will have to be assessed over a matter of months rather than having a dramatic and sudden effect on her symptoms and that it is important to persevere with treatment over this time period. If you suggest an selective serotonin reuptake inhibitor (SSRI) then she needs to know about the common side effects, such as nausea and headaches, that she may experience when starting the drug but which will often improve over the first 7–10 days. She should also know about other side effects such as vivid nightmares and sexual dysfunction.

How would you explain the possible psychological treatments and what they involve?

Explain that her fears are related to how she views the threatening situations and that treatment aims to help her alter the way she thinks and the unhelpful, unrealistic expectations she has. It helps her see the situation in alternative ways that do not lead to the same fear. Treatment will involve her looking at the automatic assumptions that she makes and help her intentionally relax in threatening situations. She will then be encouraged to, probably gradually, enter progressively more threatening situations, using the new skills she has learnt.

CHAPTER

9 Schizophrenia

BRIAN LUNN

KEY CHAPTER FEATURES

- Introduction to schizophrenia
- Epidemiology and presentation
- Aetiology of schizophrenia
- Management and assessment of acute episodes and chronic disorder

Introduction

If a member of the general public was asked to describe their archetypal 'madman', the chances are that their reference point would be an individual with schizophrenia. Asking this archetypal layman for terms they associate with schizophrenia the odds are that 'split personality' and 'violence' are liable to be mentioned. Both terms are highly stigmatizing and in the case of the former, simply wrong, and in the case of the latter, inaccurate. In this chapter, the aim is to introduce the reader to a more scientific understanding of the disorder starting with a historical perspective.

Schizophrenia is a major health problem both in the United Kingdom and worldwide. It is a relatively common condition in which the first acute symptoms usually occur in early adult life but it is often misrepresented and poorly understood by the general public. The acute illness is typically followed by a gradual deteriorating course. The symptoms and signs of the illness cause severe distress to patients and their relatives. Extensive resources are required for the management of the acute illness and long-term support in the community. Across a patient's lifetime, the costs add up to make it the most expensive individual illness treated in the National Health Service (NHS).

Why is this relevant to you?

Although only around 4% of UK medical students will go on to become psychiatrists, it is inevitable that doctors in every field of medicine will come

across patients with schizophrenia. This complex disorder, unfortunately, is also associated with poorer physical health including higher rates of heart disease, cancer, diabetes, etc. An understanding of schizophrenia and psychosis in general is therefore essential for these patients to receive the care they need.

It is perhaps worthwhile to first consider what 'psychosis' means.

Psychosis

Psychotic symptoms can occur in the context of a wide variety of different clinical situations. The presence of psychotic symptoms will frequently suggest that a patient may suffer from schizophrenia, and this illness will be the main focus of this chapter. However, there are other so-called 'functional' psychoses such as schizoaffective disorder and delusional disorder that share some features with schizophrenia. (As discussed in Chapter 2, the term 'functional psychosis' was coined in the days when nothing was known about the evidence for biological factors in the cause of psychotic illness. The term was used to distinguish presentation of symptoms that clearly indicated disturbance of brain function but which were in the absence of what could, at that time, readily be perceived as organic brain disease, such as tertiary syphilis, a brain tumour or post-encephalitic state.) Psychotic symptoms may also present in the context of severe mood disorders, abuse of alcohol and a variety of other drugs, organic brain disease (e.g. Alzheimer's disease), and poorly controlled complex partial seizures, in delirium, in conditions such as porphyria and occasionally as an adverse effect of usual doses of prescription drugs.

When we say a person is 'psychotic' we imply that they have, to some degree, lost touch with reality. Usually the person will have no insight or only limited insight into this. The most common and obvious features will be the presence of delusional ideas and/or hallucinations, which are usually driving some significant disturbance of behaviour. It is often the behavioural disturbance that results in the person being presented to mental health services. These individuals are also usually said to be 'paranoid', and paranoid thinking is one of the most ubiquitous manifestations of psychosis. However, it is important to understand what we mean by the term 'paranoid'. Someone who is paranoid tends to see everything as relating to themselves in some way. This may be in the sense that they feel others are out to harm them or that they feel others are talking about them. However, it does not just imply people or events relating to them in a potentially detrimental way, although this is often the way the term is used in more loose conversation. It may be that they see themselves as responsible for, or linked to, real events that they are not in reality connected to, leading perhaps to a grandiose view of themselves. Or they may believe another person is in love with them, for example, leading to delusional ideas about a romance.

Recognizing psychosis is not a diagnosis in itself but the first step towards making a diagnosis. The ready availability of laboratory tests and brain imaging techniques usually makes it relatively easy to exclude obvious organic brain disease in the majority of cases where such may be suspected. However, it can often be extremely difficult in the early stages to distinguish between the other potential causes. For example, schizophrenia and schizoaffective disorder merge across a continuum of symptoms; some drug-induced psychoses can be slow to resolve and the related social dysfunction may create a presentation akin to schizophrenia. The final diagnosis often depends on the acute symptoms and also the response to treatment and/or evolution of the illness over a period of weeks or months.

What does schizophrenia mean?

A brief history of schizophrenia is presented to convey how our understanding of this has changed over time. The diagnosis of schizophrenia, as for the majority of psychiatric disorders, is not on the basis of pathology or one individual symptom but on the occurrence of clusters of symptoms, which allows consistency in the diagnosis of the disorder. Schizophrenia was first recognized as a discrete syndrome in the middle of the nineteenth century, although the term schizophrenia was not used until 1911, by Bleuler. There are, however, descriptions of what sound like similar psychotic disorders in Vedic and ancient Egyptian texts from several thousand years earlier as well as descriptions in Arabic medical writings from the Middle Ages.

The first modern phenomenological description of what we now term schizophrenia was proposed by Morel in 1853. He referred to *Demence Precoce* characterized by bizarre behaviour and mental function, withdrawal and self-neglect, starting in adolescence. Fifteen years later Kahlbaum identified a cluster of symptoms, including stereotyped movements, outbursts of excitement and stupor that he called *Katatonie*. The description of *Hebephrenia* in 1871 by Hecker was very similar to Morel's *Demence Precoce*. The most important step forward in identifying a phenomenologically clear disorder was made by Kraepelin in 1893, when under the term *Dementia praecox* he grouped together all of the previously described disorders with the addition of what he called *dementia paranoides*. Kraepelin considered hallucinations, delusions, thought disorder, negativism and emotional blunting to be characteristics of *dementia praecox*. He also observed that the onset was usually in early adult life and often progressed to a 'demented' end stage. Kraepelin did, however, realize that the breakdown was not intellectual, the onset was not necessarily in adolescence and the prognosis was not always poor. He did not use the term dementia in the way that we currently do, but rather to convey a deteriorating picture of global functioning that included psychotic symptoms.

When using the term 'the schizophrenias', Bleuler described four groups of symptoms that he thought were characteristics. These four A's were:

- **A**mbivalence
- **A**utism (Bleuler didn't use this term in the modern sense, but instead meant an extreme withdrawal of oneself from the fabric of social life into a vivid internal fantasy life)
- Flattened **a**ffect
- Loosening of **a**ssociations

From the description of *dementia praecox* by Kraepelin, up until the 1950s, the focus of interest in schizophrenia was on cognition. Schneider, in 1939, described his 'first rank symptoms' (Box 9.1), which he proposed were diagnostic of schizophrenia in the absence of overt brain disease. The discovery of chlorpromazine at the end of 1950 and its clinical use in the following years led to a shift from the focus on cognition to a therapeutically driven emphasis on tranquillization. As a result, there needed to be clear diagnostic criteria to support therapeutic trials, with an inevitable focus on psychotic symptoms. Diagnosis began to be framed around Schneider's first rank symptoms.

Box 9.1 Schneider's first rank symptoms

1. **Auditory hallucinations of a specific type**
   Audible thoughts: A voice anticipating or repeating the patient's thoughts aloud, two or more voices discussing the patient in the third person, voices commenting on the patient's behaviour.
2. **Thought alienation**
   Thought insertion
   Thought withdrawal
   Thought broadcasting
3. **Passivity phenomena**
   Experiences of bodily influence
   Made acts/impulses/affects: Experiences which are imposed on the individual or influenced by others
4. **Delusional perceptions (a two-stage process)**
   First, a normal object is perceived; second, there is a sudden intense delusional insight into the object's meaning for the patient, e.g. 'The traffic light is green; therefore, I am the King'.

VIDEO 9.1

Auditory hallucinations – https://vimeo.com/13209836

VIDEO 9.2

Delusional perception – https://vimeo.com/13209518

Although the majority of patients with Schneider's symptoms will probably have schizophrenia, the symptoms are not specific for schizophrenia (8% of psychotic patients with these symptoms do not have schizophrenia) and are most typical of the acute phase of the illness. A minority of patients with first rank symptoms may be suffering from delusions or hallucinations in the context of a severe mood disorder or an organic disorder. Twenty per cent of patients with chronic schizophrenia never have a first rank symptom.

ICD and *DSM* (as outlined in Chapter 1) classification systems allow the diagnosis of schizophrenia to be made in both acute and chronic phases of the illness and reduce the likelihood of the misdiagnosis of schizophrenia in those with other primary disorders such as mood disorders and organic syndromes such as toxic confusional states, drug-induced psychoses and epilepsy.

Epidemiology

The usually cited figure of lifetime incidence of schizophrenia is 1%. A 2002 systematic review stated that the figure might well be lower, suggesting a figure of 0.55%. In the United Kingdom, prevalence is roughly 200 per 100,000. The older literature suggested that there is no significant difference in the incidence of the disorder between males and females, but onset is typically earlier in males. More recent first-episode studies have suggested that there may actually be a higher incidence in males. Peak onset for males is between the ages of 20 and 28 years, whereas for females the peak range is between 26 and 32 years. Schizophrenia can occur in childhood and in middle to old age but incidence in these age groups is much lower. In adolescents, presentation of schizophrenia may be slightly different from the standard pattern seen in later life in that there may be vague, non-specific symptoms that are only recognized as the onset of the disorder in retrospect. Another interesting observation is that there is no difference in social class of parents. The clustering of patients in lower socio-economic classes would therefore seem to be a result of the illness, suggesting downwards social drift.

A World Health Organization study originally seemed to suggest that there was little difference in prevalence between different countries. However, closer examination of this coupled with data from more recent studies suggest that there may be significant differences between countries. There are two important difficulties in trying to elucidate this issue: (1) it is often difficult to ascertain all cases, as a proportion of people with psychosis (perhaps 20%) will not present to doctors; (2) it can take some years of follow-up to determine a final diagnosis with reasonable certainty. There is clustering of cases in urban areas. One explanatory theory for this is 'urban drift' in that resources for managing patients with schizophrenia tend to be found more commonly in areas with higher population densities, but some studies have suggested that the urban environment itself may have a role to play in the aetiology of the disorder. So-called 'urban drift' was first demonstrated 60 years ago by Faris and Dunham who showed that although the geographical distribution of residence of the parents of patients (between upper-class suburbs, intermediate areas and inner-city areas) was the same as the overall population, the patients were much more likely to be found living in the inner-city areas. Patients also drift downwards in terms of socio-economic status – the university student who drops out and becomes a part-time shop worker, the factory worker who loses motivation and becomes unemployed. However, there is also some evidence to support the proposition that some of the preponderance in lower socio-economic groups is because of a slightly higher incidence of the illness in urban areas and among such groups.

Presentation

Symptoms of schizophrenia are typically divided into 'positive' and 'negative' subtypes. The former are typified by psychotic symptoms such as identified by Schneider (Box 9.1), i.e. delusions, hallucinations, thought disorder, etc. By contrast, negative symptoms can be characterized as losses of normal functioning, e.g. blunting of affect, social withdrawal, poverty of thought, etc. It is important to realize that not all such symptoms may be related to schizophrenia but may represent other disorders such as depression or be related to recreational substance use. A useful way to think about signs and symptoms might be to use the components of a mental state examination as in Box 9.2

VIDEO 9.3

"I just knew…": schizophrenia – https://vimeo.com/21423496

Prior to the onset of acute symptoms (and not due to mood disturbance or substance abuse) and following an acute episode many patients exhibit less dramatic symptoms:

- Marked social isolation and withdrawal
- Impairment in social role, e.g. as a wage earner or student

Box 9.2 The features of mental state examination in possible schizophrenia

Appearance and behaviour

This can be completely normal but classical presentations include perplexity, social awkwardness, withdrawal and odd behaviours such as smiling in response to no apparent stimuli. Impulsivity along with over-aroused behaviours can occur as can aggression, but this is less common than a pattern of withdrawal.

Speech

In the acutely ill patient this can be difficult to follow reflecting thought disorder. Just as common is poverty of speech, not just in relation to quantity but also poverty of ideas and vocabulary. Classically described are neologisms (made up words; sometimes a condensation of more than one word).

Mood

Mood changes are common in schizophrenia, something often forgotten by students. These changes can be divided into three main types. These are:

- Blunting of affect where emotional responsiveness is flattened or absent leading to the patient appearing indifferent to events
- Mood changes such as depression (relatively common in the acute phase) and euphoria/elation
- Incongruous mood where the emotional response is not in keeping with the trigger, e.g. smiling when describing sad or upsetting events

Thought form

Disorders of form of thought are already mentioned in the section on speech but it is worth emphasizing here. The train of a patient's thoughts may be difficult to follow, the ideas expressed may be 'concrete' reflecting impaired abstract thought or the links between ideas expressed may be tenuous or incomprehensible (loosening of associations). Other observed abnormalities may include thought block where the patient suddenly stops speaking mid-sentence and has the subjective experience of their thought coming to an abrupt halt.

Thought content

Delusions are common. Although primary delusions are classical they are not common. Much more frequently seen are secondary delusions. These are commonly persecutory but may be grandiose, delusions of reference (where external events have a direct and special meaning for them), delusions of external control (may be called passivity phenomena) or delusions of thought alienation. The latter covers thought broadcasting, thought withdrawal and thought insertion. More detail is provided in Chapter 3.

Perceptions

The most common hallucinations are auditory, with those set out in Box 9.1 having particular significance. Hallucinations can occur in any sensory modality. Delusional interpretation of hallucinations is common.

- Peculiar eccentric behaviour
- Poor personal hygiene
- Blunted or inappropriate affect
- Vague speech
- Odd beliefs
- Unusual perceptual experiences
- Lack of initiative or energy

Subtypes of schizophrenia

A number of subtypes of schizophrenia have been described, based on particular patterns of clinical symptoms. However, these subtypes are not entirely distinct from each other and do not have clearly different aetiology from each other. Thus, they have a limited value in everyday clinical practice:

- **Simple schizophrenia:** Negative symptoms predominate.
- **Hebephrenic schizophrenia:** Mood is inappropriate with giggling and shallowness, behaviour is irresponsible. Delusions and hallucinations are fragmented. Thoughts are disorganized. Onset is typically in the age range of 15–25 years.
- **Paranoid schizophrenia:** Complex delusions and hallucinations. Delusions may be persecutory, grandiose or religious.

Box 9.3 Differential diagnosis

Organic disorders:

Autoimmune encephalitis

Complex partial seizures

Psychosis arising from use of psychoactive substances

Functional disorders

Depression

Mania

Delusional disorders

Personality disorders (the early stages of schizophrenia may appear as though a personality disorder)

VIDEO 9.4

Jo's story – "I'm disgusting…": schizophrenia – https://vimeo.com/9350256

VIDEO 9.5

Catatonia – https://vimeo.com/19840546

- **Catatonic schizophrenia:** Psychomotor disturbance varying from stupor to sudden outbursts of activity, waxy flexibility, automatic obedience and negativism.

Box 9.3 highlights the list of other diagnosis that need to be considered before a diagnosis of schizophrenia can be made.

Aetiology

Theories include the dopamine hypothesis, neurological dysfunction, and environmental and genetic theories.

The aetiology of schizophrenia is a complex and unresolved subject. The contribution of any individual factor (e.g. obstetric complications) to the total incidence of schizophrenia in the population is usually modest, but may be important for an individual case. Overall, however, it would seem that aetiology at a population level is multifactorial and may also often be so for individual patients. Most often, for individual cases it is impossible to identify a cause, although families will frequently ask this question.

Over the years there have been varying emphases applied to the multiplicity of factors that play a role in the aetiology of schizophrenia. The traditional approach has been to look for predisposing genes and environmental or other intrinsic risk factors. In the end, there has been consistent failure to identify a single extrinsic cause. The recent debate has centred on a 'stress-vulnerability' model where the interaction of genetic and environmental factors (molecular epigenetics) can work as a model to understand this complexity.

Original work emphasized possible neurological causes as at that time the emphasis was on the neurological nature of the disorder. As drug treatments became available this shifted to neurotransmitters, particularly dopamine. In the backlash against the overuse of medication there was an emphasis on psychological causes, but speculation about particular styles of child rearing as an aetiological factor has not been supported by evidence and only served to fuel feelings of guilt in some parents. Current theories blend many of the elements of previous work in a more coherent but still partial model. There has been a rekindling of interest in the neurological model particularly around the role of inflammation and the immune system.

Genetic

The risk for individuals with an affected relative is significantly greater than for the general population and can be seen most strongly in twin studies where dizygotic twins have a risk not much different from non-twin siblings, but the risk for monozygotic twins is about four times higher. Further reinforcement has come from adoption studies where the risk for a child adopted away

Box 9.4 Epigenetics

Epigenetics refers to how changes in gene expression occur as a consequence of either intrinsic or external factors. External factors can include age, environment, stress etc.

from a parent with schizophrenia remains high. This has been covered in more detail in Chapter 2.

Environmental

The role of environmental factors remains unclear. As already mentioned, there is an increased incidence of schizophrenia in urban populations, and it has been suggested that pollution may play a role. Other factors include obstetric complications and winter births (in males). Heavy cannabis use, particularly before the age of 16 years, can contribute to the risk, although this may be mediated by genetic factors.

Environmental factors such as stressful life events can have a role in precipitating episodes of illness, and highly charged emotional environments (high expressed emotion) may well have a maintaining role and increase risk of relapse.

These factors may work through altering the expression of genes that in turn predispose individuals to schizophrenia epigenetic factors.

Neurological dysfunction

Symptoms similar to schizophrenia are seen in complex partial seizures where left temporal lobe limbic structures are involved, e.g. temporal lobe epilepsy. It has also been observed that birth complications are associated with an increased risk of schizophrenia. Along with the observations of structural brain abnormalities seen in brain imaging studies, an association with an increased prevalence of childhood developmental problems and the presence of soft neurological signs (e.g. minor abnormalities in coordination), it has been argued that these observations suggest neurological insults at an early age (and stage of brain maturation) may play a role in the development of schizophrenia in some individuals.

The role of the immune system

It has been argued that genetically predisposed individuals if exposed to maternal viral infection in utero developed schizophrenia as a consequence of subsequent interference in brain development. More recently theories have shifted as animal studies suggested that it was the subsequent immune response that determined the risk. There are issues with research in this area as there are many confounders that influence immune responses. It has been shown that antipsychotic medication has a significant effect on the immune system.

The autoimmune hypothesis

There is an increased incidence of schizophrenia in families with a history of autoimmune disorders, perhaps by 29% (examples include coeliac disease, autoimmune thyroiditis, psoriasis).

Autoimmune encephalitis, also known as limbic encephalitis, was a term first generated in the late 1960s and referred to a syndrome where there were hallucinations, memory disturbance (often leading to secondary delusions) and seizures. Investigation showed inflammation of medial temporal lobes. It is only in the last few years, as assays allowing identification of relevant autoantibodies have been developed, that it has been discovered that such an autoimmune disorder can present with clinical pictures indistinguishable from an acute psychosis. A number of autoantibodies responsible for such a clinical picture have been identified. These include *N*-methyl-D-aspartate (NMDA) receptor and voltage-gated potassium channel antigens.

This is an important discovery for two reasons: a recent study has suggested that up to 10% of first onset psychosis are a result of autoimmune encephalitis, which can be treated with a variety of strategies such as immunosuppression and intravenous immunoglobulins. Presenting as psychosis this important high morbidity neurological disorder can be missed if there is not awareness of how it may present.

Stress

The immune system's response to stress is via the glococorticoid hormone, cortisol. Responsible for mediating fight or flight when peril is perceived, cortisol also acts to supress the immune response. Therefore when stress is chronic there are long-term effects on the immune system. Stress has long been implicated in both the aeitology and prognosis of schizophrenia through stressful life events, living environment (e.g. high expressed emotion, poverty, overcrowding), and we now have evidence that people with schizophrenia are more vulnerable to stress than the general population. Since the autonomic nervous system and the hypothalamic pituitary adrenal axis are involved in

processing stress reponses it may be that trauma has a role in the aetiology of psychoses:

Neurochemical hypotheses

The first neurochemical hypothesis for the causation of schizophrenia, for which there was a consistent body of evidence, implicated dopamine pathways (see Table 2.5) and evolved from around 1970. (Previous neurochemical hypotheses all lacked consistent and replicable evidence.) The initial evidence in favor was that dopamine agonists such as amphetamines and levodopa (L-dopa) could cause schizophrenia-like symptoms in some people, and that all drugs effective for schizophrenia were dopamine receptor antagonists. Indeed, drugs with geometric isomers are only effective when the isomer given is a dopamine receptor antagonist. It should be remembered, however, that the therapeutic effects of antipsychotic drugs on psychotic symptoms are not restricted to schizophrenia. Studies of post-mortem brain tissue suggested possible changes in dopamine receptors but subsequent in vivo brain imaging studies in patients found conflicting data. More recently, a considerable body of evidence suggests that there may be primary disturbances in glutamate pathways and that these may then have knock-on effects on gamma-aminobutyric acid (GABA) pathways. Dysfunction of dopamine pathways are likely to be secondary to such changes or at least manipulation of dopamine pathways may ameliorate some of the effects of these possible primary disturbances. Interesting observations are that the neurochemical changes observed are consistent with some of the neuropsychological changes found in people with schizophrenia and with some of the loss of brain grey matter found in magnetic resonance imaging (MRI) brain imaging studies.

Nevertheless, the only currently available treatments for the 'positive' symptoms are drugs which are dopamine (DA) receptor antagonists. Disturbance of glutamatergic systems have been found and may be related to some of the underlying neuropathological changes seen. Research into the 5-hydroxytryptamine (5-HT) system has not suggested this as primarily important, but manipulation of this may be relevant to certain symptoms, such as low mood, and to the presence of some of the effects of the second-generation antipsychotic drugs. Accepting that neurotransmitters play a role in schizophrenia, it is unclear whether the neurochemical findings in research are indicative of primary or secondary pathological processes, or are the result of compensatory mechanisms or environmental influences.

The role of recreational drug use in schizophrenia is complex. In clinical practice, it is difficult to identify whether an individual's drug use has unmasked an underlying illness, has caused the illness in a vulnerable individual or may be the consequence of the illness in an individual with a subclinical picture. The answer like almost everything in psychiatry is liable to be complex. Cannabis use, for example, increases the risk of illness but this is most significant in individuals with an underlying genetic vulnerability and has a strong correlation with early (prior to the age of 15) cannabis use.

Management

Management of schizophrenia can be best thought of as assessment followed by management of the acute and then chronic stages.

Assessment

Although the initial assessment of the acute illness has often taken place in hospital, depending on local services, it is increasingly carried out in community settings either by community mental health teams or crisis/home treatment teams. Specialist teams (which may be named Early Intervention Teams, First Episode Psychosis Teams or variants of these) are increasingly common in the United Kingdom and other clinical locales. These have significantly decreased the need to admit patients. Where the assessment takes place will depend on how the individual presents (for example, if acting upon delusions or hallucinations the police may be involved), severity, risk and/or level of support available. It is unusual for an individual to 'self-present', more typically it is the concerns of others that result in involvement of mental health services. An assessment should include:

- A full history and mental state examination, in particular to identify delusions, hallucinations, thought disorder
- A check for clouding of consciousness

- An interview with third-party informants as relevant to that individual
- A physical examination and appropriate investigations depending on age and mode of presentation, e.g. urine drug screen to exclude a drug-induced psychosis, electroencephalogram (EEG), brain imaging, neuropsychological assessment
- An assessment of the patient's global physical health due to high risk of vascular disease and/or diabetes mellitus
- A social assessment: housing, work, etc.

The aim is that by the end of the assessment:

- Information supporting the diagnosis should have been collected.
- Particular risks the patient may present to themselves and others should have been identified.
- Vulnerability factors such as a family history of schizophrenia should have been considered.
- Possible precipitants to the acute illness should have been identified (the 'why now?' question).
- Factors that may maintain the illness or make relapse more likely should have been explored, such as high critical expressed emotion within the family or stresses at work.
- Whether the patient is prepared to accept treatment should have been decided.
- A differential or coexisting disorder should have been considered.

Management of the acute episode

In the acute stage of schizophrenia the focus is typically on management of acute psychotic symptoms and risk. The mainstay of treatment is pharmacotherapy with antipsychotic drugs combined with various types of social, practical, emotional and psycho-educational supports, as appropriate to the person's stage of illness. Second-generation antipsychotic drugs should be tried first, but it may sometimes be appropriate to try a first generation drug if one or two second-generation drugs are not effective. The pharmacology of these drugs is described in Chapter 18.

As a first-line treatment oral medication is used, with some drugs giving the option of an 'oro-dispersible' preparation where compliance is in doubt. In more difficult cases, depot preparations administered every 2–4 weeks allow monitoring of patient compliance, as they are administered intramuscularly. This can also address variability in first-pass metabolism and provide more predictable plasma levels in individuals who often have chaotic lives.

In the acute phase, antipsychotics provide sedation, which helps with excitement, irritability and insomnia. More recent analyses of clinical trial data suggest that improvements may be seen in the first 1–2 weeks, and if definite improvements are not being seen within 2–4 weeks that change in the initial antipsychotic medication should be considered. Although antipsychotics help manage the acute psychotic positive symptoms such as hallucinations and delusions, they are less effective at managing the negative symptoms. Indeed, many of the claimed benefits from the trials of newer drugs in this respect may only reflect differing side-effect profiles or issues with study design.

In patients with more extreme behavioural disturbance, rapid or acute tranquillization may be required, usually with short-acting benzodiazepines either on their own or in addition to an antipsychotic. This is covered in more detail in Chapter 19. As many patients when acutely well lose insight and lack capacity as a direct result of their illness, mental health legislation is sometimes needed to ensure that they are treated. Compulsory treatment is dealt with in Chapter 5.

Prognosis following the first episode varies considerably. Patients whose first presentation is with an episode of acute psychosis, but in whom the nature of persistence of symptoms does not meet the diagnostic criteria for schizophrenia, can have a very good outcome with perhaps 20% never experiencing a further episode of illness. However, the majority of those who meet the diagnostic criteria for schizophrenia will have some persistence of symptoms. This may range from a mild level of negative symptoms to persistent and distressing delusions and/or hallucinations accompanied by significant social disability. Age at onset and mode of onset of symptoms are the two most reliable prognostic indicators. Younger age of onset predicts poorer outcome. Males have earlier onset than females and thus, on average, also less good outcomes. More gradual insidious onset also predicts

poorer outcome. A sudden onset, over a few days, especially if prodromal features are absent (e.g. a prolonged period of social withdrawal) will usually have a good outcome. Patients whose illnesses appear (usually in retrospect) to have come on over many months do less well. Insidious mode of onset also predicts a longer duration of the period of untreated psychosis (i.e. before the condition is recognized), which itself predicts poor outcome. Heavy premorbid cannabis use may be another factor in poor outcome but persistent use after diagnosis is clearly detrimental.

Management of the post-acute phase

In patients who recover fully from the acute phase, there is a need to consider strategies to prevent further episodes. The management involves pharmacological and psychosocial components. The more acute episodes a patient has in their life the worse the prognosis, so there is every reason to try and prevent relapses.

A common approach is to use long-term antipsychotics. This has to be balanced against the deleterious effects of possible side effects on a patient's quality of life, not least because these side effects may have a negative influence on compliance. Often clinicians will try and use lower medication doses in the maintenance phase in contrast to the treatment phase.

Consideration must be given to psychosocial interventions. It may be necessary to consider what levels of support activities of daily living require. Adequate housing and meaningful activities can also contribute. Family interventions can target behaviours, such as high expressed emotion (see above), that can contribute to relapses and help families to understand and cope better with the 'negative' type symptoms.

Where a patient remains symptom free there will come a time when it is reasonable to attempt to stop antipsychotic medication. This needs to be preceded by a discussion regarding the possible risk of relapse balanced against the potential negative consequences of long-term antipsychotic use. The period before such a withdrawal is attempted is open for debate with no hard evidence, but in England and Wales the National Institute for Health and Care Excellence (NICE) highlights the high risk if medication is discontinued in the first year or two and recommends close monitoring for at least 2 years following medication withdrawal.

Where there is incomplete response to treatment then treatment resistance, covered below, needs to be considered, but when the residual symptoms are predominantly negative medication change is unlikely to prove effective. In all these patients there is a recommendation for psychological interventions. Already mentioned are family interventions. Some studies have shown utility for some form of cognitive behavioural therapy (CBT), which aims to aid the patient in challenging hallucinations and delusions and therefore allow them to cope with greater ease. Psychodynamic approaches may help in understanding the patient's problems and formulating responses and is more commonly used now in family work.

In all cases, ensuring that patients have adequate housing, are not in financial difficulty and are able to care for themselves or are supported in this is essential.

Box 9.5 Who might be involved in providing post-acute care

- Community or rehabilitation psychiatrist
- Primary care doctor
- Community psychiatric nurse
- Social worker
- Support worker (health, social care)
- Voluntary organizations
- Housing providers (council, voluntary organizations, private providers); housing may be group care, supported or independent
- Occupational therapist
- Psychologist
- Primary care practice nurse
- Education services

Treatment resistance

A proportion of patients with schizophrenia do not respond to standard treatment approaches. In all cases where patients do not appear to respond to treatment, a reasoned, questioning approach needs to be followed. In the first case, adherence to treatment needs to be questioned. Failure to follow a medication regime may not be wilful but nonetheless needs to be considered and, if suspected, addressed. For cases where the negative symptoms and/or chaotic lifestyle of the patient lead to neglectful non-compliance, the use of a depot/long-acting injectable medication, or other strategies to ensure medication is taken, warrant consideration.

Other causes also need consideration, such as use of alcohol and/or other recreational drugs, comorbid illnesses such as depression or significant ongoing stresses. These then need to be managed appropriately.

Only when these issues have been addressed and adherence to treatment is assured, with evidence that a patient has had an adequate trial of at least two antipsychotic drugs (one of which must have been a second-generation drug) at an adequate dose for a sufficient period of time (at least 6 weeks), can there be said to be treatment resistance.

In cases of treatment resistance the next approach should be a trial of clozapine. Owing to the risk of agranulocytosis, patients require regular haematological monitoring. This starts off as weekly but falls in time to monthly. In some cases there will be failure to respond to clozapine. Here, as covered above, psychological interventions may help, but there remains the option of augmentation with other antipsychotics (e.g. amisulpiride or aripiprazole).

Physical health

Patients with schizophrenia are at greater risk of physical health problems, such as cardiovascular disease, diabetes and cancers. The causes of this include lifestyle, side effects of medication and what appears to be a higher inherent risk.

Ideally, all patients starting on antipsychotics should be first assessed physically, including an assessment of cardiac risk. If the patient is too disturbed to enable a physical examination at initial presentation, this should be done once the patient settles. The assessment should include:

- A history of smoking, diet, exercise and alcohol and recreational drug use
- Calculation of body mass index and measures of truncal obesity such as waist-to-hip ratio
- Recording of blood pressure

Investigations include electrocardiogram (ECG), full blood count (FBC), urea and electrolytes (U and Es), liver function tests (LFTs), glucose and lipid profiles. As several of the drugs used may cause hyperprolactinaemia, prolactin should be assessed and added to the list of measures that require regular review.

Active management of problems identified at baseline or subsequently on repeat measures is essential. Psychoeducation programmes looking at healthy lifestyles should augment this. It is worth remembering that a patient who is stable and has given up smoking may need their medication reviewed as cessation of smoking leads to an increase in drug bioavailability.

Schizoaffective disorder

This is not a subtype of schizophrenia but a separate disorder. It is common in differential diagnoses, particularly by students, but should only be used in very specific circumstances. It is important to realize that mood symptoms are quite common in schizophrenia, particularly following early acute episodes of illness. It is important therefore that the diagnosis of schizoaffective disorder should be limited to cases where diagnostic criteria for both schizophrenia and a mood disorder occur during the same episode. Otherwise, the diagnosis is of the predominant syndrome.

Summary

Schizophrenia is a relatively common mental illness and can have significant impact on those who have the disorder and their families. It is often poorly understood by the general public and non-mental health professionals, which means that patients can experience discrimination. Early recognition and reduction of relapses improves prognosis. Most present with acute symptomatology, although a significant minority never develop Schneider's first rank symptoms. Antipsychotic medications are extremely effective for many but maintenance doses can present some challenges.

Further reading

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CASE STUDY 9.1

A 19-year-old man is brought into accident and emergency (A and E) by the police after he had been found wandering in the street in the small hours of the morning. They describe him as sitting mumbling to himself and occasionally looking to the side and talking as though replying to someone sitting next to him.

When you go to see him, he is sitting looking relaxed with a distracted smile on his face. On trying to engage him in conversation, you find his train of thought impossible to follow. He appears distracted and will break off conversation or not attend to what you are saying to look to a point just behind your right shoulder when he will smile as though in response to something that has attracted his attention.

What actions would you take to assess this young man?

An assessment of his mental state should focus on:

- Observing his behaviour
- Assessment of his speech (particularly looking for breakdown of syntax and pointers to disorders of form of thought)
- Assessment of his mood
- Exploration of his thoughts with a view to identify any abnormally held ideas such as delusions and/or overvalued ideas, thought alienation (thought insertion, withdrawal or broadcasting)
- Assessment of whether there are abnormal perceptions (hallucinations or illusions/misperceptions)
- Cognitive assessment with particular reference to his conscious state
- Assessment of history – this is most likely to be of value when a corroborative history can be obtained, e.g. from a family member or close friend. Looking for a pattern of change over time, a history of drug use etc. would be of particular value.
- Investigations in the immediate period might include urine or blood screening for recreational drug use.

What differential diagnoses would you consider?

- Schizophrenia
- Other schizophreniform illnesses
- Intoxication with psychoactive substances
- Drug-induced psychosis
- Complex partial seizures

CHAPTER

10 Substance misuse

ILANA CROME

KEY CHAPTER FEATURES

- Frequency of substance misuse
- Drug and drug addiction
- Susceptibility to substance misuse and impact of substance misuse
- Assessing and treating substance misuse

Introduction

There are many myths about addiction, such as patients do not get better, there are no treatments, treatments do not work, 'once an alcoholic always an alcoholic', older people cannot be drug users, and addicted parents are bad parents. Hopefully this chapter demonstrates that this is exactly what they are – myths. Substance use, misuse and dependence are commonplace in the community, as well as in clinical settings. There are many different psychological and pharmacological treatments available, patients can and *do* improve, substance misusing parents are not necessarily poor parents, and older people *are* at risk of developing substance problems. Medical students and junior doctors are very important partners in raising the profile of substance misuse issues, as well as collaborating with multidisciplinary teams in its identification and treatment. Not all addiction services are provided directly by the National Health Service (NHS) and not all of these employ psychiatrists.

Why is this relevant to you?

No major specialty or subspecialty in the whole of medical practice can *avoid* substance misusers. Even if the patient presents with a substance problem, this may not be his or her major problem. Conversely, patients may present with numerous conditions resulting from substance use. Emergency

departments (previously called accident and emergency units) and departments of cardiology, dermatology, gastroenterology, neurology, infectious diseases, oncology, respiratory medicine, obstetrics, geriatrics and surgery will generate their share of patients with substance-related problems, as will trauma and orthopaedic departments. This is because people may present with acute symptoms due to the effects of intoxication and withdrawal syndromes and as a consequence of the chronic use of substances. Convulsions, acute disturbance (psychosis, panic, confusion and accidents), cancer and cardiovascular and respiratory conditions are some of the conditions with which patients may present. Psychiatric specialties such as adult, child and adolescent and old age, forensic and liaison psychiatry in hospital, community and criminal justice settings are increasingly contributing to the treatment of substance misusers. General practitioners too will regularly be managing the impact of substance misuse on the individual, family and community, because it is very common. In addition, medical services often have to work with social care services, e.g. education, housing, child protection and the criminal justice system, to manage this group of patients.

How common is substance misuse?

Boxes 10.1 and 10.2 show the impact of alcohol use and misuse in England and the advice given regarding alcohol use, respectively.

Alcohol problems make up 4% of the global burden of disease. Around 25% of the population drink above the safe recommended limits, per capita alcohol consumption has doubled over the last 50 years and is still rising (Prime Minister's Strategy Unit, 2004). There are 7 million hazardous drinkers in the United Kingdom. Hospital admissions for conditions related to alcohol consumption have doubled in the last 10 years, as have death rates over the last 15 years. Alcohol misuse costs the country £20 billion per annum and there are about 22,000 premature deaths from alcohol misuse every year. The mortality associated with alcohol and drugs can be up to 16 times higher than in the general population. Twenty-five per cent of young people (16–24 years) drink over the recommended weekly limits for low-risk drinking in adults (21 units for men and 14 units for women) and 9% of young males and 6% of young females drink over 50 units per week, which would be high-risk drinking behaviour in adults.

Box 10.1 The impact of alcohol use and misuse in England

- Alcohol costs the NHS £2.7 billion per year.
- There are over 1 million attendances at emergency departments each year as a result of excessive alcohol consumption.
- Alcohol intoxication and withdrawal are common in emergency departments and prompt treatment is required to reduce mortality and morbidity.
- 40% patients attend emergency departments in the day but 70% attend it at night.
- 17% of road traffic accidents are related to illegal blood alcohol levels.
- 3%–6% adults in England have alcohol dependence.
- 18% of adults binge drink.

Box 10.2 Advice about alcohol consumption

Department of Health advice

- Men should not drink more than 3–4 units a day regularly.
- Women should not drink more than 2–3 units per day regularly.
- Regularly means most days of the week.
- If episodes of heavier drinking occur, people should have 48-hour alcohol free periods.

Royal College of Physicians' advice

- Men should not drink more than 21 units and women 14 units a week, provided the total amount is not drunk in one or two bouts and that there are 2–3 alcohol free days a week.

Illicit drugs

It is estimated in the British Crime Survey 2014/15 (https://www.gov.uk/government/statistics/drug-misuse-findings-from-the-2014-to-2015-csew)

that about 2.8 million adults used illicit drugs during 2014–15. Among 16–59-year-olds, reported use is as follows: cannabis 6.7%; powder cocaine 2.3%; ecstasy 1.7%.

Proportions, by age, reporting use of illicit drugs in the last year is the following:

- 16–39 years ($n = 91,360$): 13%
- 40–59 years ($n = 10,416$): 3.6%
- 16–59 years ($n = 19,732$): 8.6%

Proportions reporting misuse of prescription drugs are the following:

- Men 16–24 years ($n = 1,060$): 8.8%
- Women 16–24 years ($n = 1,274$): 5.6%
- Men 25–59 years ($n = 7,942$): 5.8%
- Women 25–59 years ($n = 9,715$): 4.1%

279,000 adults used a new psychoactive substance in the last year with use concentrated among young adults aged 16–24 years.

Drug misuse costs the country £15 billion per annum at a conservative estimate). There are 2,000–3,000 deaths from drug misuse every year.

Smoking

Smoking causes more preventable deaths than any other substance. There are about 10 million smokers in the United Kingdom. 22% of adult males and 17% of adult females smoke. Smoking rates have halved since 1974 from 45%, and are highest in the 24–34 years age group (25%) and lowest in the over 60s age group (11%). Two-thirds of smokers start before the age of 18. There are approximately 100,000 premature deaths from cigarette smoking each year as half of all smokers will be killed by their addiction. There is also an impact on smokers' families each year as UK hospitals see around 9,500 admissions of children with illnesses caused by second-hand smoke. Rates are higher in economically deprived groups.

Box 10.3 Classes of illicit drugs (Misuse of Drugs Act, 1971)

- Class A: Ecstasy, lysergic acid diethylamide (LSD), heroin, cocaine, crack, magic mushrooms and amphetamines (if prepared for injection)
- Class B: Amphetamines, cannabis, methylphenidate, pholcodine
- Class C: All benzodiazepines and the 'z' hypnotics, some painkillers, gamma-hydroxybutyrate (GHB), ketamine

What is a drug and what is drug addiction?

What is a drug or substance?

The term 'drug' covers licit/legal (e.g. tobacco and alcohol) and illicit substances (Box 10.3), e.g. central nervous system depressants such as opiates and opioids (e.g. heroin and methadone), stimulants (e.g. cocaine, crack, amphetamine and ecstasy), and LSD, khat and magic mushrooms. Street use and non-compliant use of prescription and over-the-counter drugs, such as benzodiazepines and codeine-based products (e.g. cough medicines, decongestants) are also included. People may use a combination of all these substances, which is known as polypharmacy or polydrug misuse. Patients may buy, borrow and share and may use out-of-date drugs. They may knowingly or unwittingly not report all use and they may forget or may not realize that a substance is psychoactive. They may be deliberately misleading, sometimes because they think that they will be denied treatment.

Concepts of harmful use and dependence (addiction)

There are specific criteria for the diagnosis of substance problems and dependence, outlined in Boxes 10.4 and 10.5 respectively, in the *International Classification of Diseases*, 10th edition (ICD-10) and the *Diagnostic and Statistical Manual*, Fifth edition (DSM-V) of the American Psychiatric Association. While ICD-10 continues to define 'harmful use' of a substance separately from 'dependence' on a substance, the DSM-V criteria have now linked these two concepts together as a single disorder measured on a continuum from mild to severe. For the purposes of treatment management, it is essential to distinguish non-dependent substance

misuse from dependent or addictive use. The more serious the substance problem, the more likely there are to be associated with it psychological, physical and social problems, which require a more intensive level of treatment intervention. A diagnosis of dependence can be made if three of the criteria listed have been present during the preceding 12 months. The criteria are as follows:

- Compulsion or craving, i.e. a strong desire to take the substance
- Tolerance, i.e. needing more to get the same effect
- Difficulties in controlling the use of substances
- A withdrawal syndrome when substance use is reduced or stopped
- Relief of withdrawal by substance use
- Persistent use, despite evidence of harmful consequences
- Neglect of interests and an increased amount of time taken to obtain the substance or to recover from its effects

Every substance has its own withdrawal syndrome. For example, the withdrawal syndrome for alcohol consists of tremor, sweating, craving, agitation, nausea and vomiting, whereas for opiates the features are runny eyes and nose, muscle aches and pains, diarrhoea and nausea (Table 10.1).

Can people suffer from addiction at all ages?

Substance misuse problems may commence in adolescence and continue into old age. Childhood

Box 10.4 ICD-10 (World Health Organization, 1992) harmful use and dependence syndrome criteria

1. **Harmful use**
   - A pattern of psychoactive substance use that is causing damage to health. The damage may be physical or mental.
   - The diagnosis requires that actual damage should have been caused to the mental or physical health of the user.
   - Harmful patterns of use are often criticized by others and frequently associated with adverse social consequences of various kinds. The fact that a pattern of use or a particular substance is disapproved of by another person or by the culture, or may have led to socially negative consequences such as arrest or marital arguments, is not in itself evidence of harmful use.
   - Acute intoxication or 'hangover' is not itself sufficient evidence of the damage to health required for coding harmful use.
2. **Dependence syndrome**

   A definite diagnosis of dependence should usually only be made if three or more of the following have been present together at some time during the last year:
   - A strong desire or sense of compulsion to take the substance
   - Difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use
   - Physiological withdrawal state when substance use has ceased or been reduced, as evidenced by either of the following: the characteristic withdrawal syndrome for the substance, or use of the same (or closely related) substance with the intention of relieving or avoiding withdrawal symptoms
   - Evidence of tolerance, such that increased doses of the psychoactive substance are required to achieve effects originally produced by lower doses
   - Progressive neglect of alternative pleasures or interests because of psychoactive substance use and increased amount of time necessary to obtain or take the substance or to recover from its effects
   - Persisting with substance use despite clear evidence of overly harmful consequences (physical or mental)

Box 10.5 Criteria for substance use disorder in DSM-V (American Psychiatric Association, 2013)

1. A problematic pattern of use of an intoxicating substance leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within 12 months.
2. The severity of the disorder is defined as: mild (the presence of two or three symptoms); moderate (the presence of four or five symptoms); severe (the presence of six or more symptoms).
   a. Substance is often taken in larger amounts or over a longer period than was intended.
   b. There is a persistent desire or unsuccessful efforts to cut down or control substance use.
   c. A great deal of time is spent in activities necessary to obtain substances, use substances or recover from its effects.
   d. Craving or a strong desire or urge to use substances. Persistent desire or repeated unsuccessful efforts to cut down or control substance use.
   e. Recurrent substance use resulting in failure to fulfil major role obligations at work, school or home.
   f. Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use.
   g. Important social, occupational or recreational activities are given up or reduced because of the substance.
   h. Recurrent substance use in situations in which it is physically hazardous.
   i. Use of the substance is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been cause or exacerbated by the substance.
   j. Tolerance, as defined by either of the following:
      i. A need for markedly increased amounts of the substance to achieve intoxication or desired effect
      ii. A markedly diminished effect with continued use of the same amount of the substance
   c. Withdrawal, as manifested by either of the following:
      i. The characteristic withdrawal syndrome for that substance
      ii. The substance (or a closely related substance) is taken to relieve or avoid withdrawal symptoms

maltreatment contributes to the prevalence of co-morbid personality disorder in addiction populations.

Substance misuse may result in miscarriage, stillbirth, fetal distress, placental abruption, eclampsia, early labour and sudden death. Babies born to pregnant, substance misusing women may suffer from the effects of withdrawal, and neonates and infants may experience delayed development, which can have an impact in adolescence and young adulthood.

Young people are using substances more frequently, mainly alcohol, cannabis and nicotine, and the age of use is earlier now than previously. Children as young as 10 years may present with substance misuse issues. However, most presentations are during adolescence. Many young people who present to treatment units have numerous associated psychosocial problems (for example, self-harm, unstable accommodation, poor education, involvement in criminal activity and difficult family relationships) as well as mental and physical health problems. Their coping and adaptive strategies may be poorly developed or absent. The overall mortality rate of adolescent addicts is 16 times that of the general adolescent population.

Women who use substances in particular are more likely than a control group to have been exposed to sexual, physical and emotional abuse as children and are more likely to experience emotional distress than

Table 10.1 Symptoms of intoxication and withdrawal

Substance	Intoxication	Withdrawal
Alcohol	Disinhibition Argumentativeness Aggression Interference with personal functioning Labile mood Impaired judgement and attention Unsteady gait and difficulty in standing Slurred speech Nystagmus Decreased level of consciousness Flushed face Conjunctival injection	Tremor (tongue, eyelids, hands), agitation, insomnia, malaise, convulsions Visual, auditory, tactile illusions or hallucinations
Opiates	Apathy Sedation, drowsiness, slurred speech Disinhibition Psychomotor retardation Impaired attention and judgement Pupillary constriction Decreased level of consciousness Interference with personal functioning	Craving Sneezing, yawning, runny eyes, muscle aches, abdominal pains, nausea, vomiting, diarrhoea, pupillary dilatation Goose flesh, recurrent chills, restless sleep
Cannabis	Euphoria and disinhibition Anxiety and agitation Suspiciousness and paranoid ideation Impaired reaction time, judgement and attention Hallucinations with preserved orientation Depersonalisation and derealization Increased appetite Dry mouth Conjunctival injection Tachycardia	Anxiety Irritability Tremor Sweating Muscle aches
Nicotine	Craving Malaise or weakness Anxiety, irritability, moodiness Insomnia Increased appetite Increased cough and mouth ulceration Difficulty in concentrating Tachycardia and cardiac arrhythmias	Insomnia Bizarre dreams Fluctuating mood Derealization Interference with personal functioning Nausea Sweating
Stimulants	Euphoria and increased energy Hypervigilance Repetitive stereotyped behaviours Grandiose beliefs and actions Paranoid ideation Abusiveness, aggression and argumentativeness Auditory, tactile and visual hallucinations Sweats, chills, muscular weakness Nausea or vomiting, weight loss Pupillary dilatation, convulsions Tachycardia, arrhythmias, chest pain, hypertension Agitation	Lethargy Psychomotor retardation or agitation Craving Increased appetite Insomnia or hypersomnia Bizarre and unpleasant dreams

a control group of female substance misusers who do not have that background.

Older people suffer from physical and psychiatric co-morbidity. They may be taking prescribed and over-the-counter medications for their ailments, as well as licit and illicit drugs. They may start using substances at an early age or when they are older. The number of older people is projected to increase, so that people over the age of 65 will constitute 23% of the population by 2020.

Older people are at risk of substance misuse for many reasons, including the development of multiple,

chronic physical and psychiatric illnesses, for which they receive prescription medications that they may or may not take compliantly and that may interact with physical and psychological illness and illicit and licit substance use. Some users who began their substance misuse in the 1960s continue it into old age, whereas some initiate substance misuse in older age due to isolation, losses and illness and others use substances to cope with disability, pain, anxiety and insomnia.

Thus, every single patient you come across requires a substance misuse assessment, because it may have a bearing on diagnosis and treatment. Age is no barrier to the development of substance-related problems, as Case Study 10.2, at the end of the chapter, demonstrates.

Who is susceptible?

People who have psychological (e.g. anxiety, depression and psychosis) and physical (e.g. pain) problems are particularly susceptible to substance misuse through many mechanisms:

- A primary psychiatric and/or physical illness may precipitate or lead to a substance problem
- Substance misuse may worsen or alter the course of a psychiatric and/or physical illness
- Intoxication and/or substance dependence may lead to psychological and physical symptoms
- Substance misuse and/or withdrawal may lead to psychiatric or physical symptoms or illnesses
- Primary psychiatric disorder may precipitate substance use, which may lead to psychiatric and/or physical symptoms or syndromes

In studies that have examined the association between substance misuse and psychiatric illness, roughly 30%–50% of people with psychiatric disorder have been found to use substances. Between 30% and 50% of substance misusers have psychiatric illnesses. Substance misuse may be associated with psychotic illnesses, such as schizophrenia, bipolar disorder, schizoaffective disorder, anxiety, depression, post-traumatic stress disorder, eating disorders, attention deficit hyperactivity disorder, personality disorder and dementia.

In an extensive review of risk factors in young drug users, the most consistent evidence revolved around family interaction. Biological factors (i.e. those which cannot be changed) include genetic predisposition, age and ethnicity. Psychological factors such as self-esteem, early behavioural disorders, impulsive and sensation-seeking behaviour, antisocial behaviour and mental disorder (as described earlier) may be difficult to change. Behavioural or attitudinal factors, which may be susceptible to change, include family attitudes to substance misuse, substance use by family members, religious affinity and attendance, conduct disorder and educational aspiration and attainment. Interpersonal relationships with family and friends are potentially open to modification, with regard to parental supervision and control, familial bonding and harmony and childhood trauma. Environmental and economic factors are outside the individual's control and include socio-economic status, neighbourhood unrest and the availability of local amenities, which require political involvement.

A number of these factors may be operative at any one time and they may be mitigated by protective factors such as family support and monitoring, good relationships with parents, religious affiliation, later age of initiation and academic achievement. The age at which exposure to substances takes place and the setting also have an influence on substance use later. Social problems such as family problems can also be a result of the substance misuse.

What is the link between substance misuse and violent behaviour?

The pharmacological effect of the drug itself, especially (but not only) if taken in high doses, may be disinhibiting and lead to impulsivity, aggression, abusiveness and argumentativeness, agitation and grandiosity (e.g. amphetamine or cocaine). Some substances may exacerbate a psychotic episode. Chronic misuse of substances may lead to a dependence syndrome so that withdrawal causes unpleasant symptomatology, including a confusional state (benzodiazepine withdrawal) during which paranoid ideation may occur. The distress and discomfort arising from cravings, including anxiety and depression, may lead to disturbed behaviour. Accidents, injuries and violent acts may occur during intoxication, in part as a result

of the individual's decreased ability to make sound judgements due to impaired attention and concentration, paranoia and even the presence of delusions and hallucinations. The combination of psychosis and co-morbid use of drugs results in a higher rate and severity of violence than in a population with psychosis but no substance use. Non-adherence to medication for a psychiatric disorder may also lead to violent behaviour. If patients are substance misusers and non-compliant with medication, violence is twice as likely in these patients compared with those with either one of the problems. The prevalence of violent behaviour generally increases with the number of psychotic symptoms, except in those with the most psychotic symptoms where violence risk is relatively low. This can be explained in terms of social isolation and a degree of impairment so severe that it incapacitates the person from carrying out violent acts.

Thus, drug-related violent crime could be classified both as violence arising from the effects of substances and violence associated with the interaction of a psychiatric illness with drug use. Remember that violence can also be connected with acquisition of drugs and linked to disputes between drug users, drug dealers and drug gangs.

Sex-related crimes such as prostitution and 'date rape' sexual assault are examples of illegal activity. Driving under the influence of drugs and alcohol may lead to injury, as well as criminal convictions. Crime and violence are associated with victimization, social instability and marginalization, including homelessness and economic deprivation. These often stem from childhood and adolescent trauma and family problems, as well as educational and social skill deficits.

What is the impact of substance misuse on individuals, families and communities?

Substances do not have to be regularly used or even misused to have a significant impact on the lives of individuals and families. You do not have to be addicted to experience serious effects from a substance. For example, a person may be intoxicated from alcohol abuse, become involved in a road traffic accident and suffer severe injuries. However, the consequences of substance misuse may be related to the degree of dependence, the quantity and the quality (purity, adulterants) of the substance and the route of administration (e.g. smoking or injecting).

Serious physical illness is, of course, an additional 'co-morbidity' and one that is, perhaps, overlooked, underrated and undertreated. Physical problems such as pain, infection, injury and cancer may lead to mental illness and may result from substance misuse. If not adequately treated, these conditions not only add to the suffering of individuals, but also undermine treatment provided for substance misuse.

Substance use may be life threatening. Intoxication from alcohol is characterized by impaired judgement, reduced consciousness, lack of coordination and ataxia, disinhibition and slurred speech. Coma, associated with hypoglycaemia and hypothermia, and even death may ensue. Accidents, injuries, falls, burns and choking are other consequences, along with convulsions as a result of head injury. Stimulant intoxication may lead to increased aggression, and paranoia and anxiety or irritability due to craving. Volatile substances are dangerous, since not only does use result in unsteadiness and lack of coordination but also in cardiac arrhythmias, which may prove fatal. Drugs such as opiates may depress respiration to the extent that coma results.

Withdrawal from substances can also be a medical emergency necessitating rapid response. Alcohol withdrawal is unpleasant since tremulousness, nausea, vomiting, sweating, general malaise and insomnia result. Benzodiazepine withdrawal presents in a similar fashion. Severe alcohol withdrawal results in delirium tremens (discussed further in Chapters 12 and 19), which includes severe agitation, confusion, hypertension, hyperthermia, delusions and hallucinations. This condition can be fatal but, if intervention is early, recovery is good.

Withdrawal from opiates is not as risky, but it is very uncomfortable. General aches and pains, diarrhoea and vomiting, runny nose and eyes, and difficulty sleeping may be part of the withdrawal state. As combinations of substances may be used, withdrawal symptoms from several drugs may occur. Stimulant withdrawal may lead to inertia, depression, increased appetite and retardation; suicidal ideas may develop.

Chronicity of use is another factor in the extent to which substances may impact on health. Alcohol and nicotine contribute to the development of cardiovascular and respiratory disorders and are associated with heart disease and stroke. They may also cause cancer:

tobacco use is associated with cancer of the lung, lips, tongue, throat, larynx, oesophagus, kidney, pancreas and bladder, and alcohol use is associated with cancer of the liver, bowel, throat, mouth, larynx, breast and oesophagus. In addition, fertility may be reduced.

Substance use by injecting the drug carries the risk of infection, e.g. blood-borne viruses such as HIV, hepatitis B and hepatitis C as a result of contamination from the blood or body fluids of infected users. Septicaemia, localized infections, such as abscesses in the heart, skin and central nervous system, and emboli may result. Repeated injecting may damage the veins, causing ulcers, abscesses and thrombophlebitis. Deep vein thrombosis from injecting large vessels in, for example, the groin is of special concern because of the possibility of emboli.

Substance misuse is a strong predictor of self-harm and suicide, and previous history of self-harm is a strong predictor of completed suicide. This too is a psychiatric emergency, since poisoning with substances is common among patients presenting to emergency departments with self-harm. Craving for substances, specific adverse effects or distress may provoke an overdose. Whether accidental or deliberate, overdose with depressants such as alcohol, sedatives and opiates may lead to coma and death, whereas with stimulants may lead to cardiac arrest. Reduced tolerance may develop during a period of abstinence, for example if the patient has been in prison, which may lead to an unintentional overdose.

As a result of these complications, 'co-morbid' patients present to primary care, secondary care and general medical, surgical and mental health services.

VIDEO 10.1

"I think I'm drinking too much…": alcohol dependence – https://vimeo.com/29137043

What are the components of a good assessment?

The key to appropriate management is acquiring a thorough history as discussed in Chapter 3. It is helpful to consider what specific information you need to elicit to ensure a comprehensive substance misuse history. An understanding of the current social circumstances, e.g. living arrangements, family constellation and education or employment activities, is important as these may be precipitating and perpetuating factors. They may also influence potential interventions and the likelihood of their success. Details about all current licit and illicit substance use, as well as prescribed and over-the-counter medication should follow. Information about the history of use, e.g. age at first use, onset of weekly and daily use, route of use (e.g. oral, injecting, smoking), and pattern of use are core parts of the assessment. Whether withdrawal symptoms for any substance have developed, the cost of use, the maximum ever used, periods of abstinence and triggers to relapse provide a picture of the extent of the problem. Previous treatment for substance misuse, mental and physical illness and the effectiveness of treatment is another strand. The nature of substance misuse and mental illness in the patients and their family provides vital background information, as do details of physical illness associated with substance use. Life events, such as bereavement or divorce, as well as the nature of family relationships (e.g. conflict or separation) can provide some understanding regarding the historical precipitants of initiation, continuation or cessation of substance misuse. The history of criminal activity and resulting charges and convictions, including level of debt, is also valuable. The degree of social and community support and details of other agencies involved should be discussed so as to plan a comprehensive response. Special groups, e.g. teenagers, older people, homeless people, parents with young children and immigrants, have particular needs, in terms of both assessment (e.g. consent and confidentiality) and treatment (for further details please see the 'Further readings' and 'Useful websites' sections). Child protection may be an important issue to consider when assessing parents who have substance misuse problems. After these basic details have been completed, the information obtained can be utilized in the general protocol that follows. It is adapted from one developed for nicotine dependence and is a useful way to formulate the assessment process, because it translates into specific management plans. These are all the things you as a medical student can and should consider with every patient you see.

Phase 1: Ask

- Ask all patients about alcohol and other substance misuse, including prescribed and over-the-counter medications.
- Differentiate between substance use, harmful use and dependence.
- Assessment is ongoing and the information should be recorded.
- Be aware of, and sensitive to, the ambivalence substance misusing patients may feel.
- Be non-judgmental and act in a non-confrontational way, as this can be a powerful determinant of engagement with treatment.

Phase 2: Assess

- Educate patients about the effects of substances, for example inform them about withdrawal symptoms.
- Assess the degree of dependence.
- Assess the level of motivation or 'stage of change'.
- In this context, aim to provide advice or suggestions as to what the 'goals' (e.g. abstinence or harm reduction) are.
- Negotiate appropriate treatment options (e.g. pharmacological interventions, the need for admission to specialist services).
- Clinical manifestations of the condition may impair the history-taking process (e.g. dementia, confusion, poor concentration).
- Follow an assessment schedule.

Phase 3: Advise

- Continue the assessment within a brief 5- to 10-minute 'motivational interviewing' framework.
- Provide the patient with the opportunity to express their anxieties and concerns.
- Offer personalized feedback about clinical findings, including physical examination and biochemical and haematological tests.
- Discuss and outline the personal benefits and risks of continued substance misuse.
- Provide self-help materials (e.g. manuals).

Phase 4: Assist

- Provide support and positive expectations of success.
- Acknowledge that loss of confidence and self-esteem might have resulted from failed previous attempts.
- Suggest that, if the goal is abstinence, a 'quit date' is set, so that the patient can plan accordingly (e.g. get rid of any alcohol in the house) and quit safely (is it safe to stop drinking abruptly or not?).
- Work through a range of alternative coping strategies, e.g. identification of cues that might help to distract the patient.

Phase 5: Arrange

Be prepared to refer or organize admission to a specialist or the appropriate unit if the patient:

- Is in, or is likely to develop, severe withdrawal, including delirium tremens
- Is experiencing unstable social circumstances
- Is severely dependent
- Has a severe co-morbid physical or psychiatric illness, including suicidal ideation
- Is using multiple substances
- Has a history of frequent relapse

During all phases, close attention should be paid to the appropriateness of various options for the particular individual – 'tailor-made' where possible. Case studies 10.1, 10.5 and 10.6 illustrate some of these issues.

What treatments are available and do they work?

The National Institute for Health and Clinical Excellence (NICE) and similar bodies (Scotland has an equivalent in the Scottish Intercollegiate Guidelines Network [SIGN]) have produced a series of reviews

of the treatment of opiate dependence, smoking and alcohol (see www.nice.org.uk/ and www.sign.ac.uk/). In Northern Ireland there is no equivalent organisation but NICE guidelines are usually adopted following consultation with relevant local clinicians and others. Psychological treatments are an important part of the spectrum of available interventions. For 'treatment' to be successful, aftercare and long-term support with social issues, especially employment and accommodation, are essential components. It is also about treating the combination of substance misuse disorders, rather than treating them separately. We have become increasingly aware that addiction is not a 'stand-alone' disorder – in fact co-morbidity is the norm within the family and community, with local or regional and even national and international ramifications, since substances and substance misusers have a habit of crossing national boundaries.

Psychological approaches

Substance misusers vary in their suitability for psychological treatments and interventions may be more or less appropriate in individual cases due to age, cognitive ability or dysfunction, education, willingness and capability or capacity to view problems as psychological. However, psychological treatments are nonetheless perceived as being pivotal to treatment effectiveness, even when pharmacological treatments are administered. Standardization of approaches and outcome measures is complex. Treatment philosophies, environments and settings may differ greatly (e.g. primary care, emergency departments, prisons). Additional resources for treatment (e.g. support from other agencies such as housing, education, probation) may vary. Some groups may be discriminated against across a variety of services because of general stigma around substance misuse, poorly trained staff and lack of resources, or because of old age, female sex or ethnic minority status. It has been noted that these are the emotional and socio-economic issues that present the major challenge to recovery.

Stages of change

The process by which change occurs has been formulated as a series of stages: preconception, contemplation, preparation, action and maintenance. This theory has been influential in treatment and research. There is considerable evidence that provision of information in itself may be of some help. Information needs to be accurate and up to date and must provide advice not only on the negative effects of substance use, but also acknowledge any potential benefits. Responses to situations in which overdose might occur, physical consequences and psychological problems are useful baselines to start from.

Counselling may be appropriate. However, behavioural therapies have become the mainstay of treatment over the past decade and encompass social skills and self-control training, motivational counselling (which includes motivational interviewing and motivational enhancement therapies), marital therapy, stress management, contingency management, community reinforcement and cognitive therapies. Cognitive behavioural therapy, 12-step approaches and motivational approaches, as well as behavioural self-control training, coping skills and marital/family therapy, have been demonstrated to be beneficial for drinkers in a variety of studies.

Brief interventions have become extremely popular and are especially useful in primary care, but are also helpful in medical and psychiatric care and, to some extent, in emergency departments. Most work has been undertaken in drinkers and smokers. The key components are that treatment is delivered by a non-specialist, such as a general practitioner, opportunistically, in a non-confrontational style with a focus on the individual's preferences. Brief interventions provide the opportunity for all health and even allied professionals to raise and manage the initial intervention for people with substance problems.

The key characteristics of brief interventions are best described by the acronym FRAMES which are enumerated as follows:

- Personalized **F**eedback or assessment results detailing the target behaviour and associated effects and consequences on the individual
- Emphasizing the individual's personal **R**esponsibility for change
- Giving **A**dvice on how to change
- Providing a **M**enu of options for change

- Expressing **E**mpathy through behaviours conveying caring, understanding and warmth
- Emphasizing **S**elf-efficacy for change and instilling hope that change is not only possible, but also within reach

Pharmacotherapies are available to treat a variety of situations such as the following:

- Emergencies, e.g. overdose, fits, dehydration, hypothermia and acute confusional state, including delirium tremens
- Detoxification and withdrawal syndromes, e.g. lofexidine, methadone, buprenorphine, chlordiazepoxide
- Substitution, e.g. methadone, buprenorphine, nicotine replacement therapy
- Relapse prevention, e.g. naltrexone, acamprosate, disulfiram
- Treatment of vitamin deficiency
- Co-morbid psychiatric disorders, e.g. depression, anxiety and psychosis
- Co-morbid physical disorders, e.g. HIV, hepatitis C, diabetes and hypertension

More and more evidence is accumulating about the effectiveness of treatment. Several major studies have been undertaken in the United Kingdom and others in the United States, Australia and Scandinavia.

Who has responsibility for treating patients with substance problems? What are the challenges?

Given that any doctor may come across individuals with substance misuse issues in their work, we all have a responsibility in ensuring that these patients are properly assessed and managed. There may be a need to work closely with other doctors and health care staff. In Case Study 10.6, a general practitioner, addiction psychiatrist, physiotherapist and pain specialist were involved in the patient's care.

There is an evolving diversity of models and guidance for best practice and best treatment and there are many pointers to what the components of a less risky and more comprehensive approach should be. This has to take into account the following needs of some patients:

- Need for a holistic approach
- Complexity: this sometimes involves the 'long haul' and practitioners need to be aware that it is not a 'quick fix'
- Continuity: continuity and constant review and reappraisal of the changing situation are both reassuring and beneficial
- Unpredictability: patients may present in crisis suddenly
- Particular people have particular risks and vulnerabilities, e.g. the homeless, teenagers, older people
- Need for support and knowledge about how to access available services
- Need for supervision by experienced practitioners
- Severity: the severe nature of medical, psychological, psychiatric and substance misuse problems with which patients, who are sometimes very young, present and the need for trained medical staff. It is not just a matter of prescribing, which must be undertaken by trained, experienced staff in the context of many other medical and social problems
- Multidisciplinary team interaction within a service and good interagency cooperation
- Relationships between inpatient statutory services and how they are used regularly, yet are very difficult to access
- The dilemmas of disengagement in this vulnerable group

The Case Studies demonstrate that, in order to provide the best care for patients with addiction problems, colleagues in general medicine and general practice, general surgery, pain clinics, and emergency and psychiatry departments must be involved and to do so need to be appropriately trained. Training reduces stigma and increases confidence in management by improved identification and intervention. Treatment has been shown to be cost-effective in that every pound spent saves many more.

Summary

Hopefully this chapter has dispelled some of the myths and highlighted the developments over the last two decades and has also addressed the realities of substance misuse. Substance use, misuse and dependence are as predictable in clinical settings as they are unsurprising in our communities. There are many different treatments available and patients *do* get better, as long as the treatments are made accessible. Addicted parents are not necessarily poor parents, although they may need considerable support. Older people, like everyone else, are at risk of developing substance problems. However, they too can be treated and will improve substantially if offered the opportunity. Practitioners and services, patients and carers, policymakers and communities need to work together to erase the impact of the socio-economic challenges that undermine the potential for treatment to work. The treatment provided may be suboptimal because of resource constraints. Small, gradual improvements or prevention of further harm are also important steps in the route to achieving abstinence and should not be denigrated. It is sometimes very difficult for people to achieve a drug-free condition on the first occasion they attempt to reduce or stop substance use.

Medical students and junior doctors are essential allies in this quest to support the individual, families and communities. At the present time a national undergraduate addiction curriculum is being rolled out, so that all medical schools can train students in the basic management of addiction patients.

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Useful websites

Alcohol and Drug Abuse Institute: http://depts.washington.edu/adai/

Alcohol Concern: www.alcoholconcern.org.uk/

Australian Drug Information Network: http://www.adin.com.au/

Daily Dose: www.dailydose.net/

Department of Health publications: www.dh.gov.uk/en/Publicationsandstatistics/index.htm

DrinkandDrugs.net: www.drinkanddrugs.net/

Home Office: www.homeoffice.gov.uk/

National Institute for Health and Clinical Excellence: www.nice.org.uk/

National Institute on Drug Abuse (USA): www.nida.nih.gov/

National Treatment Agency for Substance Misuse: www.nta.nhs.uk/

NHS Information Centre: www.ic.nhs.uk/

National Institute on Alcohol Abuse and Alcoholism (NIAAA) (USA): www.niaaa.nih.gov/

Royal College of General Practitioners: www.rcgp.org.uk/
Royal College of Psychiatrists: www.rcpsych.ac.uk/

Scottish Intercollegiate Guidelines Network: www.sign.ac.uk/

Society for the Study of Addiction: www.addiction-ssa.org. Factsheets especially aimed at medical students

Substance Abuse and Mental Health Services Administration (USA): www.samhsa.gov/

CASE STUDY 10.1

A 13-year-old girl was referred from school due to alcohol use affecting her school work and attendance. She was assessed by a substance misuse worker at school, as this was felt to be the most appropriate environment due to her age and she could be supported by people she knew. It also ensured minimal disruption to her education. The alcohol use was affecting all aspects of her life and was a result of home dynamics as well as an older boyfriend who was causing concerns. The worker continued to see her at a regular time in her timetable and brought in the support of the family therapist, who explored her relationships with others and accessed extra support for her education, as she had fallen behind. This work aimed to prevent exclusion from school and explore the reasons for her substance misuse to be able to address the underlying issues.

CASE STUDY 10.2

A 60-year-old man was found wandering around his local area picking up cigarette stubs and begging for money to buy alcohol. He was very dishevelled. Neighbours said that he had lost his job several months previously, because he had been drinking heavily. He had had chest pains and asthma and had been noted to have hypertension. He had not taken the treatment for his cardiovascular problems for several months. He was not eating properly and was neglecting himself. He had had a period of heavy drinking years ago, but had managed to cut down. His wife died suddenly and his social network seemed to have contracted to such an extent that he was isolated and bored, especially at the weekends. He had therefore taken to drinking regularly. On admission to a geriatric unit, he was diagnosed as having alcohol-induced dementia.

CASE STUDY 10.3

A 42-year-old man presented to the outpatient clinic for detoxification. He had a longstanding history of substance misuse. He had first tasted alcohol at the age of 8 years and started drinking regularly at 13 years. He had tried almost every substance, including solvents, amphetamines, ecstasy, magic mushrooms and heroin. He had been in custody at three different periods because of theft, burglary and shoplifting offences, which were committed to fund the purchase of drugs or to maintain basic living needs. At the time of presentation, he was drinking alcohol, using benzodiazepines and topping up his methadone prescription with street opiates. He lived with his partner and two young children, in whose care he had a central role.

CASE STUDY 10.4

A specialist registrar was called to see a 49-year-old man in the surgical ward who was extremely agitated and distressed. He was attending assessment for inpatient admission. Although he had drunk 7 pints of cheap cider that morning, he was tremulous, sweaty and confused. He did not know the date and had difficulty walking. He was depressed and said that he wanted to die. He complained of failure and guilt in relation to his children and felt hopeless about the future. He was unemployed and living alone. His blood pressure was 168/102 and his pulse rate was 100. After several hours, it became clearer that he was in severe alcohol withdrawal and so he was admitted immediately for detoxification and assessment of his mental state.

CASE STUDY 10.5

A 25-year-old woman presented to the emergency department with cocaine misuse. Her parents, who were proud and respectable people, attended with her. They were devastated. She complained that people were staring at her. Her appetite and sleep were poor. She was a bright student who had started a college course, but had to drop out temporarily because of her drug use, though the college was prepared to keep her place. Her parents virtually placed her under house arrest. For 3 weeks it appeared that she was managing without drugs, but then she started to use and developed a full-blown amphetamine dependence, injecting several times a day. Her appearance deteriorated badly: she appeared gaunt, pale and unhappy. She said that she did not mind if she died after a binge of amphetamines. She was referred to the specialist addiction service. She usually attended when arranged and continued to make good progress. Her parents thought that they could manage and refused family therapy.

CASE STUDY 10.6

A 16-year-old pregnant user was referred by her general practitioner, who had begun prescribing her buprenorphine. She appeared to be a nice friendly girl, who presented no particular problems. She lived with her boyfriend, who was an older, established drug user. Her supportive family did not know about her drug use. The client had been attending the service for 18 months and, while she appeared relatively stable on methadone, in fact she never produced a negative urine screen and her methadone gradually crept up. She attended the clinic fairly reliably. Since her partner was a user, she was tempted to ‘use on top’ just a little each day and was always on the verge of feeling that she might need an increased dose of methadone. Her partner was fast-tracked for assessment and treatment and, once he was treated with a high dose of methadone, she improved.

Eating disorders

CHAPTER 11

JOHN EAGLES

KEY CHAPTER FEATURES

- Anorexia nervosa: prevalence, risk factors, assessment and management
- Comorbidity of anorexia
- Anorexia nervosa in children and males
- Bulimia nervosa: prevalence, risk factors, assessment and management
- Binge eating disorders

Introduction

Eating disorders have become more common during the last 50 years. Their aetiology comprises a complex mix of biological, psychological and social factors. Their management is eclectic and challenging. Broadly, eating disorders comprise restricting intake or binging and purging type disorders. Both are more common in females and this may be linked to societal expectations of the ideal female body shape.

Why is this relevant for you?

General practitioners, other doctors and dentists need to be well informed and vigilant regarding the possibility of eating disorders, as early detection is associated with a better prognosis. Anorexia nervosa is the most common cause of unexplained weight loss among adolescent girls and sensitive exploration of this, and other potentially suspicious physical symptoms and signs with which eating disorders may present, may lead to diagnosis. Women may present with amenorrhoea and/or infertility to obstetricians and endocrinologists. All young females are a high-risk group but athletes, dancers and models are at very high risk. There is no substitute for a clinical consultation in reaching the diagnosis, though screening of high-risk groups with instruments such as the Sick, Control, One stone, Fat, Food (SCOFF) questionnaire (see Box 11.1), may be helpful.

Box 11.1 SCOFF screening questions

1. Do you make yourself **s**ick because you feel uncomfortably full?
2. Do you worry you have lost **c**ontrol over how much you eat?
3. Have you recently lost more than **o**ne stone in 3 months?
4. Do you believe yourself to be **f**at when others say you are too thin?
5. Would you say that **f**ood dominates your life?

Doctors may also have a role to play in prevention, which can be viewed from broad political and educational perspectives, focusing on widely held dysfunctional attitudes arising from society's preoccupation with a thin body ideal. More general public education in healthy nutrition would be helpful. Educational programmes have been used to promote realism with regard to body shape ideals, to normalize peripubertal physical changes, to address low self-esteem and to inform about healthy eating patterns.

Classification and definition

The two common eating disorders are anorexia nervosa and bulimia nervosa. Although there are some commonalities, there are differences in presentation, management and prognosis. The typical features of both conditions will be described. For the sake of clarity they will be regarded as neatly definable and separate illnesses, though in practice, there may be considerable overlap. Binge eating disorder will be discussed briefly. Although obesity is very common and often has psychological consequences such as low self-esteem and depression, as it is not regarded as a primary psychiatric disorder, it will not be discussed.

Anorexia nervosa

History and prevalence

William Gull is credited with the first description of anorexia nervosa in 1868, to which he ascribed a psychogenic aetiology. However, psychiatrists seldom saw the condition in the nineteenth century. It is probable that the symptoms of emaciation and amenorrhoea were interpreted as reflecting somatic illnesses and therefore patients were seen by physicians. Nevertheless, despite under-diagnosis, anorexia nervosa probably remained relatively uncommon in the first half of the twentieth century before its prevalence in western societies rose from the 1960s through to the 1980s, in tandem with increasing emphasis on the importance of an idealized slim female body shape. The death of the American singer Karen Carpenter from anorexia nervosa, in 1983, is often credited with raising awareness of eating disorders. Others in the public eye (for example, Diana, Princess of Wales) subsequently acknowledged their own eating disorders, and the fact that these illnesses were thus admitted to and detected more readily complicated studies of changing rates across time, though there has probably not been further increase in the last 25 years.

The tendency of sufferers to conceal their symptoms makes it difficult to estimate its true prevalence. In western societies, the current prevalence of anorexia nervosa is estimated to be 0.3% among young females. Rates among adolescents may be as high as 1%.

Risk factors

Sociodemographic factors

Females are around 10 times more likely than males to develop an eating disorder. The reasons for this are unclear and links have been made with evolutionary, biological, psychological and social theories of aetiology. One hypothesis was that the illness might constitute a way of avoiding adolescent changes, as it may prevent development of secondary sexual characteristics.

It was originally thought that eating disorders occurred more commonly among Caucasian girls in higher socio-economic groups, and this was linked to the alleged causal factor of 'striving middle-class parents'. However, this probably reflected detection biases based on cohorts referred to specialist treatment centres. Ethnic and socio-economic factors have not been confirmed as important aetiological factors in recent studies.

Theories of family pathology as causes of anorexia nervosa achieved prominence in the 1970s and 1980s. These included the ideas that anorexics were 'enmeshed' in inappropriately close relationships with parents and that they were 'triangulated' between parents who had a poor relationship, the conflicts of which were expressed in their child's illness. In these

family pathology models, the child is seen as stuck and unable to exercise appropriate autonomy. However, it is problematic to regard these clinical observations of 'abnormal' families as straightforward aetiological factors, since patterns of family interactions may be results of, rather than causes of, illness in a child.

Biological factors

Genetic factors are important. A girl who has a first-degree relative with anorexia nervosa is at least 10 times more likely to develop it than a girl who does not. For bulimia nervosa, genetic risks are less specific. Having a first-degree relative with an eating disorder increases the risk only fourfold and there are also links to the heritability of impulsivity, mood variability and substance misuse. Molecular genetic studies relating to the serotonergic system had looked promising, but no specific genes have yet been identified.

For anorexia nervosa there is some evidence for two very early biological risk factors, both similar to findings for schizophrenia. Future sufferers have experienced increased rates of pregnancy and perinatal complications and are more likely to be born in the spring and early summer, suggesting a contribution from seasonal intrauterine factors.

Despite above-average academic achievements, girls with anorexia nervosa often exhibit impaired visual memory and visuospatial skills, these deficits being independent of malnutrition. Brain imaging studies have detected abnormalities in the cingulate, frontal and temporal areas. There are inconsistencies in current findings and it is often difficult to distinguish between the causes and the effects of eating disorders.

Psychological/psychiatric/medical factors

Sexual, emotional or physical abuse in childhood increases the risk of eating disorders in later life, and subsequent traumatic life events may link closely with the onset of illness. Early childhood feeding and digestive problems are predisposing factors, possibly for both psychological and biological reasons.

Excessive concerns about weight and shape, often linked to a more general negative self-evaluation, are common premorbid features. Neuroticism and obsessionality have been found to be the most important and consistent predisposing personality traits. General psychiatric morbidity, most commonly depression and anxiety, can both predispose and impact negatively upon prognosis.

Sociocultural factors

Evidence for the importance of sociocultural risk factors is circumstantial. Although eating disorders are not unknown in developing countries, they are only common in areas of the world that have plentiful food supplies, though there are now reports of increasing rates of eating disorders in several less-industrialized countries. The modern 'epidemic' of eating disorders in developed countries has coincided with an emphasis upon the attractiveness of thinness in females. The increased incidence of eating disorders among models, athletes and ballerinas lends weight to social models of causality.

There has been controversy as to whether dieting itself may lead to eating disorders. However, clinical experience rather suggests that it is the reasons for dieting that are important rather than the dieting *per se*.

Presentation and assessment of anorexia nervosa

This section will focus on the typical young female patient. There are specific sections on anorexia nervosa in children and males respectively later.

VIDEO 11.1

Beccy's story: anorexia nervosa – https://vimeo.com/28716938

First health service contact

The first health service contact for people with anorexia nervosa is usually in primary care, making the role of the general practitioner pivotal in its assessment and diagnosis. Diagnosis of anorexia nervosa is challenging in primary care. Patients are generally ashamed and secretive and thus may not readily disclose their symptoms. Despite this, the vast majority will divulge their symptoms; indeed they may be hugely relieved to do so, if a health professional inquires in an appropriately informed and sensitive manner.

Screening studies suggest that 50% of patients in primary care go unrecognized. A study in Glasgow found that, prior to diagnosis, females with eating disorders had consulted at an increased rate over the

preceding 5 years. Presentations varied, but these women consulted particularly with gynaecological, gastrointestinal and psychological complaints. The highly differing rates at which different general practitioners refer patients with eating disorders to specialist services suggest very different levels of awareness. Complaints such as menstrual abnormalities and constipation, especially when associated with weight loss or weight fluctuations, should lead to a high level of suspicion of an eating disorder.

Specialist referral

General practitioners may feel comfortable to adopt a supportive 'wait and see' approach with milder eating disorders, especially if there are adverse life circumstance that may have precipitated the problem and if they know the patient and their family. When the initial symptoms are severe, if the patient minimizes weight loss, or if symptoms are persistent, referral to specialist services is usually required, as primary care teams are unlikely to have the skills for effective management of eating disorders.

Psychiatric assessment

In psychiatric practice, the assessment process frequently blurs into the start of therapy. This is particularly true for eating disorders, when the first interview has the purpose of assessment and of attempting to engage the (often ambivalent or resistant) patient in therapy. It is necessary for the interviewer to be sensitive, supportive and uncritical. It may also be helpful to make 'normalizing' statements before questions such as 'You'll know that a lot of young people have concerns about their weight or body shape. Do you have any concerns like that?' A broad summary of a structure for the first assessment is shown in Table 11.1.

Premorbid/prodromal features

Early childhood feeding and digestive problems are often described, although these may be over-reported by way of retrospective explanation and may in part reflect difficulties in the mother–child relationship. Abuse in childhood, be it sexual, physical, emotional or a combination of these, may be elicited.

Traits of obsessionality and perfectionism are common, which may explain the finding that girls with anorexia nervosa are academic overachievers and may focus on schoolwork, while peers are expending more of their time and energy on relationships and other areas of normal adolescent development. Girls with anorexia nervosa frequently have low self-esteem, are overcautious and anxious, and perceive themselves as ineffective. Symptoms of anorexia can be seen as an attempt to gain control when a girl perceives her life (personally and interpersonally) as being outside her control. Limiting and controlling her calorie intake and expenditure can be the one area of her life in which she feels a sense of control and effectiveness.

Premorbid obesity may occur in as many as one in five girls who develop anorexia nervosa. Teasing about overweight can be a precipitant, especially in the early years after puberty. Stressful life events, such as parental conflict or bereavement, may exert a particularly powerful effect.

Table 11.1 Assessment of possible anorexia nervosa

History	Details of weight and diet history Typical current daily food and calorie intake Exercise patterns Purging activities Possible precipitating events Developmental and personal history Family history Psychiatric and medical history
Mental state examination	Note especially: attitude to food; weight and shape; mood; obsessive–compulsive symptoms
Physical examination	Weight, height and BMI Pulse, blood pressure (sitting and standing), peripheral circulation Skin (including lanugo hair) Muscle strength
Investigations	Full blood count Urea and electrolytes, glucose, thyroid function, liver function tests, ECG (if severely underweight or if hypokalaemic)

Abbreviations: BMI, body mass index; ECG, electrocardiogram.

Central features

The symptoms and signs listed in *International Classification of Diseases*, 10th Edition (ICD-10) for the diagnosis of anorexia nervosa are summarized in Box 11.2.

Box 11.2 Summary of ICD-10 criteria for anorexia nervosa

1. Body weight is at least 15% below that expected (or failure to gain expected weight in prepubertal patients).
2. Weight loss is self-induced by avoidance of fattening foods which may be accompanied by self-induced vomiting, purging, excessive exercise and use of appetite suppressants.
3. Body image distortion with a dread of fatness.
4. A widespread endocrine disorder manifested as amenorrhoea in females or loss of libido in males.
5. Delayed or arrested puberty occurs with prepubertal onset.

Observable behaviour

The onset is often fairly gradual and insidious, and may be difficult to differentiate from a 'normal' diet or the desire to achieve a 'healthy lifestyle' which becomes progressively faddish. Although other measures to reduce weight are also frequently deployed (see the sections 'Restricting and bingeing/purging subtype' and 'Exercise/overactivity'), reduction of food intake is almost always a core feature. Higher calorie foods are the first to be reduced or excluded from the diet, with progressive stages of dietary restrictions ensuing that may lead to vegetarianism or veganism. For example, there may be avoidance of 'fat', as fat in food is equated with bodily fat and a fear of becoming fat. Calories will usually be counted with a daily limit of less than 1000. As anorexia nervosa develops, sufferers generally become more secretive and seek to hide their problems and weight loss from family and friends. Sufferers will often try to avoid eating in company and may change their style of dress to obscure their diminishing size. These clandestine aspects of anorexia nervosa can often contribute to a delay in identification and intervention for the disorder. Eating frequently provokes obvious anxiety, the process slows significantly and watching someone with anorexia nervosa consume a meal can feel like an interminable experience.

Restricting and bingeing/purging subtype

In the restricting subtype of anorexia nervosa, weight loss derives predominantly from restriction of calorie intake. Those with the bingeing/purging subtype will have episodes of binge eating and engage in other behaviours such as self-induced vomiting and laxative misuse to help weight loss. These features also occur in bulimia nervosa and will be discussed further in the section 'Bulimia nervosa'. Management approaches may differ according to the subtype of anorexia nervosa.

Exercise/overactivity

There is some evidence that young people who develop anorexia nervosa are not only more active than their peers during the disorder but have also engaged in more physical activity prior to its onset. The parents of children who develop anorexia nervosa also seem to be more physically active than the parents of children who do not.

Like restricted eating, exercising can be a secretive activity. Compulsive exercise appears to occur more enduringly in people with eating disorders who also have obsessional personality traits and is associated with higher levels of stress, anxiety and depression. Exercise can increase in frequency from the normal range to the point where it becomes a compulsive behaviour: the exercise schedule becomes rigid (often with detailed records); exercise takes priority over other activities; and there is distress and guilt if exercise is postponed or interrupted. Control of weight and shape is seen as a primary goal of exercise (as opposed to fitness), and the degree of emotional distress experienced if exercise cannot occur is entirely disproportionate.

Psychological and social features

In more severe cases, it is often difficult to disentangle the primary psychiatric symptoms of anorexia from the secondary effects of the starvation induced by the anorexia. It may thus be helpful to consider briefly the psychological sequelae of starvation.

The psychological effect of starvation can be studied in situations such as famines, prisoners of war and hunger strikers. Starving people think, talk and dream progressively more about food the longer that starvation continues. This preoccupation has similarities to obsessional ruminations. Eating tends to slow in pace and people become emotionally unstable and

irritable. Sex drive and social activity reduce. Sleep disturbance is common and low mood may occur. These symptoms can be difficult to distinguish from those of a primary depressive disorder. The level of depressed mood has been found to correlate with the degree of weight loss in anorexia nervosa. Cognitive deficits develop with impairment of concentration, attention and memory and can complicate attempts at psychological therapies.

Early successful attempts at dieting will often elicit positive reinforcement from others and give rise to temporary feelings of achievement with improved mood and well-being. As preoccupations with food, weight and shape intensify, the 'success' with which weight loss equates battles against the 'failure' associated with weight gain. Sufferers develop a morbid fear of fatness, whereby they anticipate with dread the likelihood (as they see it) that even a small amount of high-calorie food, or an interruption to other methods of weight reduction, will render them significantly obese. This is coupled with body image distortion, so that cachectic patients can observe their starving frame in a mirror and pronounce themselves to be overweight. This failure to appropriately evaluate one's physique, which is not uncommon at lesser levels in 'normal' people, seems to derive from distorted cognitive processes and not from deficits in perception. Although it can seem to be of delusional intensity, it is usually more accurately regarded as an overvalued idea.

Psychiatric comorbidity

During the acute phase of anorexia nervosa, the majority of patients will also have symptoms of depression and there is an increased risk of suicide. Obsessional symptoms, often related to calorie counting or exercise, are common. When anxiety disorders occur, these are often related to situations that involve eating or loss of control over weight-reducing activities.

Physical symptoms and investigations

Patients are often apprehensive and resistant about physical examination, so extra sensitivity may be required. Emaciation is a universal sign. Weight and height should be measured and body mass index (BMI: weight in kilograms divided by the square of the height in metres) calculated. BMI can be compared against standard values for gender and age, which can be found on widely available charts. Anorexia nervosa is characterized by a BMI of 17.5 kg/m^2 or less (normal BMI is in the range of 20–25 kg/m^2). For younger patients growth charts are often more informative than BMIs. Other important components of a physical examination are shown in Table 11.1.

Other physical consequences of restricting anorexia nervosa are summarized in Table 11.2 and other possible abnormalities in Table 11.3. The additional physical complications that may occur in the bingeing/purging subtypes of anorexia nervosa are discussed in the section on bulimia nervosa. Several of the symptoms and signs can be viewed as bodily responses to chronic low calorie intake, in effect a 'partial shutdown', notably bradycardia, hypotension, acrocyanosis, delayed gastric emptying, constipation

Table 11.2 Physical symptoms and signs of anorexia nervosa

System	Symptoms/signs
Skin/hair	Alopecia, lanugo hair, dry skin, acrocyanosis
Musculoskeletal	Myopathy, osteoporosis, pathological fractures
Endocrine/ reproductive	Amenorrhoea, reduced fertility
Gastrointestinal	Delayed gastric emptying, constipation
Cardiovascular	Hypotension (postural and non-postural), bradycardia, palpitations, syncope
Renal	Renal calculi, oedema
Neurological	Peripheral neuropathies, seizures

Table 11.3 Abnormal investigation results in anorexia nervosa

Investigation	Findings
ECG	Sinus bradycardia, arrhythmias, reduced voltage tracing
Haematology	Anaemia, leucopenia, bone marrow suppression
Metabolic/endocrine	Hypocalcaemia, hypokalaemia, hypoglycaemia, raised cortisol, low LH and FSH, raised growth hormone

Abbreviations: FSH, follicle-stimulating hormone; LH, luteinizing hormone.

and amenorrhoea. Patients may also complain of cold intolerance and may develop mild hypothermia. Lanugo hair is soft and downy and is seen predominantly over the face, trunk and arms. Its aetiology is unknown. The oedema that ensues is ill understood and may relate to hypoproteinaemia. When women have been underweight for a protracted period, a dual-energy x-ray absorptiometry scan of bone is usually indicated to investigate possible osteoporosis. This may help motivate therapy. For amenorrhoeic women who are concerned about their fertility, pelvic ultrasonography can be undertaken.

Differential diagnosis

The main differential diagnoses are severe depression, obsessive–compulsive disorder and atypical psychosis. Physically, other causes of weight loss in a young person – inflammatory bowel disease, chronic cardiac failure, coeliac disease, carcinoma (brain tumours can also cause disordered eating), diabetes, hyperthyroidism – should be considered. In these disorders the characteristic psychopathology – the fear of becoming fat – of anorexia nervosa will not be present.

Anorexia nervosa in children

Eating disorders seem to be becoming more common in children. They should be distinguished from unusual, 'faddy', eating which is common among young children and is discussed in Chapter 14. Before puberty boys may constitute as many as a quarter of cases but after puberty the large female preponderance appears. Although earlier menarche may contribute to younger age at onset, a larger part is probably played by concerns about weight and shape developing at a progressively early age.

Children (and early adolescents) may not present with actual weight loss but with failure to attain expected weight gain. Puberty is usually delayed when anorexia nervosa develops at a young age, and growth retardation is common. Aetiologically, acute physical illnesses and teasing (most commonly about obesity) appear to be more important factors in young children who develop eating disorders. Symptomatically, children often describe their findings differently, depending on their stage of cognitive development and may not be able to clearly formulate their food-related concerns. When weight loss or failure to gain weight appropriately occurs in prepubertal children, indications for concern include:

1. Continued dieting after initial goals have been achieved
2. Dieting and eating behaviour that gives rise to reduced social contact with friends
3. Excessive exercising
4. Increased unhappiness about body shape or weight as dieting continues
5. Any purging activity

Anorexia nervosa in males

Without resorting to gender stereotypes, in general, females will express a wish to be slimmer than they are whereas males will wish to be more muscular. Indeed, although males may be rather less affected by societal aspirations towards attractive body shapes (male magazines contain less about body shape than the female equivalents, but this is changing), in adolescent boys there are clear parallels between disordered eating and the pursuit of muscularity. In terms of body image, puberty is a rather different experience for boys and girls. Girls experience an increase in body fat (distancing them from the modern 'female ideal') whereas boys become more muscular, thus bringing them closer to the 'male ideal' in body shape. Other factors (e.g. genetic or hormonal) may contribute to the marked gender difference between males and females, but most aetiological credence attaches to these psychosocial differences.

The presentations of anorexia nervosa in males are largely similar to females and only the differences will be highlighted here. Premorbidly, it is more common for males to have been athletic and the drive towards athletic excellence, through weight and shape modification, is more prevalent. Reduction in sexual drive is the symptomatic male equivalent of female amenorrhoea and this may complicate assessments of male sexuality. In one fairly large study, over 50% of males with anorexia nervosa were considered to be 'asexual'. This contrasted with bulimia nervosa in which 42% of males were identified as either homosexual or bisexual. It is possible that sexual orientation is linked to differing

body image aspirations. Males collectively present after a lengthier history than do females. This may reflect the traditional male reluctance to seek healthcare, but may also suggest shame and doubt about having developed a 'female' disorder, giving an added motive for secrecy. Psychiatric comorbidity (notably depression, substance misuse and personality disorders) appears to be slightly more common than in females.

There is consistent evidence that dieting is increasing among adolescent boys in industrialized countries. There are recent suspicions of a rise in the prevalence of male eating disorders, but given their relatively low prevalence, it is difficult to be certain. However, given the current blurring and overlap of gender roles and the increased emphasis on physical appearance for both sexes, it might not be surprising if the gap in prevalence between males and females was to narrow.

Management

General considerations

Once diagnosis is reached, patients should be told and encouraged to become as well informed as possible about the condition. Without unnecessary scaremongering, it is helpful for women to know the current and longer term risks of unresolved anorexia nervosa (e.g. reduced fertility, osteoporosis), since hopefully this will help to motivate change and engagement in therapy. The relatively high mortality rates and long-term sequelae can also help to engage reluctant parents.

Most patients will initially be at best ambivalent and often openly resistant to changes in their eating and weight. These changes are generally not rapid, and patients and relatives should know this; patients may find it reassuring and families will not expect too much progress too rapidly. Developing a collaborative and supportive ongoing relationship with a knowledgeable health professional is the first and most important step in the recovery process.

Education/dietary counselling

Management is best conducted within a multidisciplinary team and a specialist dietician will often be involved. If no dietician is available, it is important that a patient has access to appropriate, informed advice about food and nutrition to counteract possible misconceptions. It is helpful from the beginning for a patient to know what she will have to eat to gain weight steadily and to feel reassured that this will proceed at a reasonable rate but without the goal of making her 'fat'. It is usually helpful to set a target weight which would be at least 90% of that expected for her age and height.

Gaining weight

Regular charting of weight is required to monitor progress. It should be remembered that patients may 'cheat', for example, by drinking water before being weighed – a pint of water weighs approximately 500 g. Particularly with more significantly emaciated patients, there is a consensus that weight gain is required before patients have the cognitive and emotional capacity to make use of psychological approaches. A slow and steady weight gain of between 0.25 and 0.5 kg per week would be a usual goal for an outpatient, whereas inpatients may be expected to gain weight a little more quickly. The medical management of the most severely malnourished patients is a specialist enterprise that is beyond the scope of this book. The main danger of unskilled management is 'refeeding syndrome' in which rapid refeeding, especially with carbohydrates, gives rise to cardiac arrhythmias, cardiac failure and confusion, mediated principally by hypophosphataemia and hypokalaemia.

Family therapy

In the therapy of anorexia nervosa, 'it is now widely agreed that family interventions are best viewed as treatments that mobilize family resources rather than treating family dysfunction' (National Institute for Health and Care Excellence [NICE], 2004). For younger patients, there is evidence that family approaches are effective and preferable to individual therapy, although the latter has use as an adjunct therapy. In the context of a supportive and educational approach, parents will be encouraged to take a joint, firm and consistent approach with their child's eating. It will usually be helpful to promote open communication within the family, and to help the parents to acquire control over their child's health.

Individual therapies

An individual approach is usually adopted with older patients. Common elements of this treatment will include support, education and nutritional advice. There is minimal evidence for the greater effectiveness of any particular approach. Therapy needs to be tailored to the individual's needs. For example, a patient's distorted perceptions of herself and her shape might be tackled with cognitive behavioural therapy (CBT), whereas emotional and relationship difficulties might be approached with techniques relating to interpersonal therapy.

Medication

Medication will quite often be used for psychiatric comorbidity such as depression or anxiety (e.g. a selective serotonin reuptake inhibitor [SSRI] antidepressant) and olanzapine can help to promote weight gain. There is little evidence of efficacy of medication for the treatment of anorexia itself.

Treatment setting and compulsory treatment

The large majority of patients with anorexia nervosa will be treated on a voluntary basis as outpatients, especially since admission will separate patients from family and friends and interrupt their work or studies. The following factors, however, may indicate the need for admission to hospital:

1. Severe weight loss (<75% of expected BMI)
2. Suicidal acts or plan
3. Rapid worsening during outpatient treatment
4. Poor motivation and insight
5. Significant medical complications (e.g. hypokalaemia, dehydration, hypotension)

Day-patient treatment ('partial hospitalization') may constitute a compromise between the benefits of admission and remaining at home.

The need for compulsory treatment is rare but may be necessary if the alternative is that the patient may die. Resistance to treatment is common. To enforce the Mental Health Act, it must be clear that the patient is absolutely refusing treatment or is so ill that consent is not feasible. Patients in this category may be so severely physically ill that they are initially managed on a general medical ward and may require nasogastric feeding.

Outcome in anorexia nervosa

Among psychiatric disorders, anorexia nervosa has the highest mortality and one of the poorest prognoses. The risk of premature death in women with anorexia nervosa has increased sixfold in patients who are severe enough to require inpatient care and threefold for all patients presenting to secondary care services. In the acute stages, the highest risk of death is from cardiovascular causes, notably when there is a bingeing/purging pattern. Increased mortality rates endure for at least 20 years following diagnosis from factors related to chronic malnutrition. Suicide rates are elevated throughout this period – about one in five of premature deaths is from suicide.

Around 50% of patients make a full recovery, 30% improve but remain symptomatic to some degree and 20% remain chronically ill. A worse prognosis is associated with comorbid psychiatric problems (depression, anxiety and obsessive–compulsive personality features) and with specific symptoms (bulimia, vomiting and laxative abuse). Good prognostic features include early detection and treatment and, for hospitalized patients, initial length of admission (which may attest to engagement with treatment rather than to the efficacy of inpatient treatment *per se*). Patients who have recovered describe the importance of supportive non-familial relationships, therapy and maturation.

Bulimia nervosa

Bulimia nervosa shares many features with anorexia nervosa that have been described above. It is a less serious condition in terms of physical health.

History and prevalence

The term 'bulimia' derives from Greek and means the hunger of an ox. Although the practice of eating large amounts of food and vomiting thereafter dates back for thousands of years, with perhaps a few cases described in the medical literature, the syndrome of bulimia nervosa was first described by Gerald

Russell in 1979. During the 1980s and 1990s prevalence increased in western societies, perhaps related to improved awareness and increased willingness to present for treatment. During the last 20 years, prevalence rates seem to have fallen, while average age at onset has decreased. The estimated prevalence is 1% among young females, but this may be a significant underestimate.

Risk factors

The risk factors for bulimia nervosa are very similar to those for anorexia nervosa, with the few differences described below. Around 40% of people with bulimia nervosa have experienced a previous episode of anorexia nervosa, which may have been brief or subclinical. A history of weight loss prior to the onset of bulimia is extremely common and dieting is an important aetiological factor. The huge increase in prevalence since the 1970s points strongly to the important causal factor of cultural pressures to be slim by controlling eating and weight.

In childhood, traumatic events and abuse (sexual, emotional or physical) occur with increased frequency. Parental obesity is common, and this seems to be at least partly a genetic predisposition. Families of bulimic patients have increased rates not only of eating disorders but also of depression, alcohol dependence and drug misuse. Serotonin dysregulation seems to play an aetiological role in that even after recovery from bulimia there is reduced platelet serotonin receptor binding.

Presentation, assessment and diagnosis

As with anorexia nervosa, females are about 10 times more likely than males to develop bulimia. It is exceedingly rare in childhood and the average age at onset is around 18 years, rather later than that for anorexia nervosa. The majority of patients with bulimia nervosa will be assessed and managed in primary care settings. As most are of normal weight, relevant questions may not be asked and the disorder remains undiagnosed. Given the high prevalence of bulimia nervosa, the condition should be suspected in younger women who present with any of the physical complaints/signs listed in Table 11.4 or with any of the psychiatric comorbidities covered in that section below.

Table 11.4 Physical symptoms and signs in bulimia nervosa resulting from binge eating, vomiting and purging

System	Symptoms/signs
Gastrointestinal	Dental erosion, salivary gland hypertrophy, oesophageal tears/bleeding, abdominal distension, constipation, raised serum amylase
Renal	Oedema, electrolyte abnormalities (hypokalaemia, hyponatraemia), dehydration
Cardiovascular	Arrhythmias, sudden death
Neurological	Seizures, peripheral neuropathies, tetany

Clinical features

The symptoms and signs of bulimia nervosa can also occur as part of the clinical picture in the bingeing/purging subtype of anorexia nervosa. The major difference between these diagnostic groups is that patients with bulimia nervosa do not have a significantly low BMI or stunted growth. The ICD-10 criteria for the diagnosis of bulimia nervosa are summarized in Box 11.3. (DSM-IV criteria also list exercise as a 'compensatory behaviour' and specify that both binge eating and the compensatory behaviours must occur on an average of at least twice per week over at least 3 months.)

Binge eating entails the consumption of an excessive quantity of food, usually over a period of less than 2 hours, coupled with a subjective feeling of loss of control. Foods consumed are usually high in calorie content, for example, biscuits and chocolate. Several thousand calories may be consumed in a typical binge, which will occur more days than not for the majority of people with bulimia nervosa. Binges may, initially and transiently, provide relief from feelings of tension, anxiety and low mood. However, this quickly gives way to feelings of disgust and guilt. Sufferers, therefore, do their best to keep their behaviour secret. Many will do this successfully, leading to underestimates of prevalence and often lengthy delays in receiving treatment, if indeed treatment is ever received at all.

Compensating behaviours are used to attempt to counteract weight gain. Self-induced vomiting is the most common (induced by putting their fingers down their throat) and laxative misuse the next most common. Especially in tandem, vomiting and laxative misuse can give rise to dangerous hypokalaemia.

Box 11.3 Summary of ICD-10 criteria for bulimia nervosa

1. A preoccupation with eating and a craving for food gives rise to episodes of binge eating.
2. Patients attempt to counteract the fattening effects of food by self-induced vomiting and/or laxative abuse and/or alternating periods of starvation and/or medication (e.g. appetite suppressants, thyroid preparations, diuretics).
3. There is a morbid dread of fatness and a strong desire to be unhealthily thin.

Measures of the severity of an eating disorder correlate quite highly with the number of different compensatory behaviours that a patient deploys.

The characteristic psychopathology is very similar to that in anorexia nervosa. Patients are preoccupied with shape and weight and usually aspire to a low (usually inappropriately low) target weight. Unlike anorexics, patients with bulimia nervosa repeatedly 'fail' in their attempts to 'over-diet', lowering mood and self-esteem further and often forming something of a vicious circle with the bingeing/purging behaviour, which has the same effects.

Psychiatric comorbidity

Depression of mood and anxiety occur with marked frequency and are not only as consequences of the bulimia itself. Of the specific anxiety disorders, social phobia and obsessive–compulsive disorder are the most common. Alcohol misuse is common, significantly more so than in anorexia nervosa. In bulimia nervosa, impulsivity is a common personality trait and is associated with raised rates of overdoses, other self-harming behaviours and promiscuity. There is an association with 'impulsive'-type personality disorders, particularly emotionally unstable/borderline personality.

Physical symptoms, signs and complications

The symptoms and signs specific to bulimia nervosa are those secondary to bingeing and purging (Table 11.4). Screening for electrolyte abnormalities (notably hypokalaemia) is the most important investigation.

Erosion of dental enamel from the repeated flow of gastric acid over the teeth can be striking and dentists can assist in the diagnosis of eating disorders when they observe this sign. Painless swelling of the salivary glands, notably the parotids, can give rise to a characteristic 'hamster'-like appearance and again this can be a helpful diagnostic clue.

People with bulimia nervosa eat more and recognize satiety less readily than control subjects, and this may at least partly be a gastrointestinal effect. It has been found in bulimia that there is an enlarged gastric capacity, delayed gastric emptying and a low postprandial release of cholecystokinin.

Differential diagnosis

When depression coexists, it may be necessary to unravel which disorder was primary, since this will guide treatment. Upper gastrointestinal disorders can give rise to recurrent vomiting, and pathology in the frontal lobes can cause disordered, disinhibited eating, but neither is associated with the characteristic psychopathology of bulimia nervosa.

Management

Management starts by attempting to engage the patient in motivation to change during the assessment interview. There may be major concerns about losing control over weight and shape, but these are generally less than for patients with anorexia nervosa. In part, this has made it easier to engage bulimia nervosa patients in controlled trials and the evidence for the efficacy of treatments is significantly stronger.

Patients frequently present with mixed features of anorexia and bulimia nervosa, without being clearly classifiable into either category (diagnosed as 'Eating Disorder Not Otherwise Specified' [EDNOS] in the US classification system). Patients with EDNOS who have BMIs above 17.5 tend to respond well to approaches used for bulimia.

Psychological treatments

There is strong evidence for the effectiveness of CBT in reducing both bingeing and purging behaviours. Irrational feelings and beliefs about shape, weight and eating are elicited and challenged, in tandem with an educational approach. Behaviours such as vomiting and laxative misuse are addressed, for example, by encouraging patients to eat gradually larger amounts of food without vomiting. Fifteen to 20 sessions, individually or in groups, are usually required.

Self-help approaches are often used for milder cases and sometimes as the first step in a tiered

treatment approach. These can be delivered either by books or online and have a strong educational component. This will include highlighting the ineffectiveness of both vomiting and laxatives as weight control measures and the way in which both physiological and psychological vicious circles are generated by bingeing and purging. Self-help programmes have been shown to afford greater benefit for people with bulimia nervosa than remaining on a waiting list.

Pharmacological treatment

Although CBT is regarded as the treatment of choice, antidepressants are often prescribed as a first line of treatment, particularly at the time of first diagnosis in primary care. Fluoxetine and tricyclic antidepressant drugs have the best evidence of efficacy and are effective in reducing binge frequency, irrespective of whether the patient is also depressed. While CBT has been shown to improve long-term outcome, it is not yet clear whether antidepressants have an enduring effect. In more severe cases, the two are often used together.

Outcome in bulimia nervosa

Keel and Mitchell (1997) reviewed studies that aggregated more than 2000 women with bulimia nervosa. At 5–10 years' follow-up, 50% had fully recovered, 30% had recovered partially and 20% were little changed. Severe symptoms, psychiatric comorbidity and impulsivity appear to be associated with a poorer prognosis. A recent meta-analysis found that bulimia nervosa doubled a woman's risk of premature death.

Binge eating disorder

Binge eating disorder can be viewed as a milder form of bulimia nervosa. Epidemiological studies suggest that it is about twice as common. The symptoms comprise bulimia nervosa without purging or other compensatory behaviours. Binge eating disorder has a female:male preponderance of only about 2:1 and is thus relatively more prevalent in males than other eating disorders. Onset is often at a later age and is more likely to be stress induced. Sufferers are often overweight and perhaps the main reason for attempting to differentiate it from 'ordinary' obesity relates to its treatability. Binge eating disorder has been found to respond to self-help interventions (as described for bulimia nervosa), to antidepressants (most prominently sertraline) and to CBT. Management will usually be in primary care where a staged approach is appropriate, starting with self-help, moving on to SSRI antidepressants and then to CBT if necessary.

Summary

Some of the key points about eating disorders can be summarized as follows:

- Anorexia nervosa is often a life-threatening condition.
- Eating disorders are common, under-detected and under-treated.
- Early treatment is associated with a better prognosis.
- There are significant and wide-ranging physical complications.
- The diagnosis hinges on a sensitive interview and a good mental state examination.
- The management of anorexia nervosa is essentially psychotherapeutic and eclectic.
- The management of choice in bulimia nervosa is CBT.
- Much more could be done in the early detection and prevention of eating disorders.

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CASE STUDY 11.1

Adeline had been a shy young child who was lacking confidence. At primary school she started to become overweight and on going up to secondary school at the age of 11 she was teased about her weight. After her periods started at the age of 13 years she began to become especially conscious of her body shape. She decided to eat a bit less and surprised herself when she took up running to find that she was good at it, getting into the school cross-country team. Gradually, she became more preoccupied with restricting calories and she told her family she had become a vegetarian. By the age of 16 she continued to diet and to exercise excessively, her mother noticed that she was pathologically thin and took her to their general practitioner.

As the family general practitioner, on which areas of her history and mental state would you focus?

You would inquire sensitively about: timescale and degree of weight loss, feelings about her weight and shape, what she eats at present, current exercise and activity, her menstrual history, whether she binges and/or vomits, whether she takes laxatives and whether there is associated psychopathology such as depression/anxiety.

Would you conduct any investigations at this stage?

It is very important to establish weight and height and thus to get a baseline BMI.

The necessity for other investigations (see Tables 11.1 and 11.3) will depend on severity, but if she gives a history of vomiting it is advisable to screen for hypokalaemia.

How would you decide whether you would refer her to psychiatric services?

Since Adeline has a 3-year history and she is currently very thin, it is highly likely that you would refer her. In general, milder and less enduring cases would be those that might not be referred to psychiatry.

CASE STUDY 11.2

Sally was an outgoing but unhappy child. As a teenager, she was particularly moody and volatile, these traits being exacerbated by her parents' divorce. Upon going away to university she felt low and lonely. After a spell of comfort eating and weight gain, she dieted and lost weight rapidly. She could not sustain the diet and started to binge eat (usually on bread and biscuits) with self-induced vomiting thereafter. After 6 months it became apparent to her flatmates that her bingeing and vomiting was occurring daily, and they persuaded her to visit her general practitioner. When she sees you (her general practitioner) she tends to minimize the importance of her symptoms.

How would you explore the impact of her bulimia on her quality of life?

The areas you cover would probably comprise: the frequency of her bingeing and vomiting, the degree of preoccupation with issues relating to body shape/weight and eating, the effect of symptoms upon her social life, her relationships, her self-esteem, her studies and her finances.

She agrees that her symptoms are having a huge effect on her life. How might you discuss the management options with her?

It will be helpful to emphasize that the medium-term prognosis is positive with appropriate treatment.

You would discuss the possibility of prescribing fluoxetine as a measure that may provide some symptom control.

You would tell her that psychological therapy is the usual first choice; given the severity of her symptoms, specialist referral would be preferable to self-help approaches.

CHAPTER

12 Organic disorders

SIMON BUDD

KEY CHAPTER FEATURES

- Definition of organic disorders
- Dementia and delirium
- Assessment, diagnosis and management of organic disorders

Introduction

Organic disorders are those disorders that have an underlying physical or pathological cause. This commonly means medical, neurodegenerative or drug-related disorders (excluding those related to substance misuse). Disorders where there is no currently understood pathophysiological cause are often called functional disorders. A wide range of physical conditions can present with or cause psychiatric symptoms or syndromes. The two most common organic disorders, dementia and delirium, are the focus of this chapter. Other organic disorders are well recognized and details of some of these are given in Table 12.1. This is not exhaustive and only describes their psychiatric presentations. When learning about physical disorders, it is important to develop appropriate knowledge of the psychiatric symptoms and presentations of that disorder as well as the physical symptoms, signs and investigation results, especially in common or easily treatable disorders, e.g. thyroid disease.

It is important to remember that there may be an organic or underlying biological cause for a patient's psychiatric symptoms. Although it may appear as though psychiatrists do not manage most organic disorders, these are always important to consider as part of the differential diagnoses. All patients presenting with psychiatric symptoms should be screened for physical disorders, using physical examination and appropriate blood tests. This is particularly the case in older people where co-morbid physical disorders may lead to a psychiatric condition or cause existing psychiatric conditions to deteriorate. For example, the chronic

Table 12.1 Organic conditions and their psychiatric presentations

Condition	Psychiatric symptoms	Further details
Cerebral lesions		
Stroke	Depression, emotional liability, personality changes, behavioural problems, dementia.	High incidence of depression in first year, atypical presentations, affects rehabilitation.
Cerebral tumours	Local effects may depend upon site of lesion, e.g. occipital may give rise to visual hallucinations, frontal to behavioural changes, hypomania and depression. Distant effects include depression.	Also adjustment disorder and depression linked to the diagnosis.
Epilepsy	Auras, hallucinations and other disorders of perception, *déjà/jamais vu* experiences, psychosis, neurotic disorders.	Complex partial seizures of temporal lobe epilepsy, schizophrenia-like psychosis and increased risk of suicide.
Head injury	Amnesia, behavioural disturbances, cognitive impairment.	Outcome dependent on extent of trauma, loss of consciousness and length of post-traumatic anterograde and retrograde amnesia.
Metabolic		
Porphyria	Emotional disturbance and lability, delirium, depression, panic or anxiety episodes, psychotic episodes.	Acute intermittent type.
Hypercalcaemia	Depression, fatigue and low energy, irritability, cognitive slowing.	
Uraemia/renal failure	Depression, memory problems, delirium (~33%), psychosis.	
Hepatolenticular degeneration (Wilson's disease)	Presents with hepatic or movement disorders, or psychiatric symptoms including dementia.	Inherited autosomal recessive disorder of copper metabolism.
Endocrine		
Cushing's disease	Weight gain, depression, psychosis, insomnia, loss of libido.	
Addison's disease	Depression, apathy, tiredness, weight loss, anorexia, mild cognitive impairment.	
Hypothyroidism	Depression, mania, schizophrenic-like psychosis, cognitive slowing, dementia, ataxia, anorexia, weight gain, depressed mood or psychotic symptoms, loss of libido, poor memory.	'Myxoedema madness'
Hyperthyroidism	Weight loss, increased appetite, anxiety, psychosis, irritability, loss of libido, restlessness, weakness.	
Hypokalaemia	Depression, sleep disturbances.	
Hypoparathyroid	Delirium, agitation, anxiety, depression, cognitive impairment, irritability, emotional lability, psychosis (rare).	
Hyperparathyroid	Depression, fatigue and low energy, irritability, cognitive slowing, dementia.	
Phaeochromocytoma	Anxiety or panic.	Episodic, associated with hypertension.
Neurodegenerative disorders	See the section 'Dementia'.	
Neurodegenerative dementias		
Huntington's disease		
Parkinson's disease		

(*Continued*)

Table 12.1 (*Continued*) Organic conditions and their psychiatric presentations

Condition	Psychiatric symptoms	Further details
Infectious agents		
CJD	Rare, rapidly progressive psychiatric and neurological symptoms suggestive of CJD, severe dementia, pyramidal and extrapyramidal neurological disease.	Incidence 0.1/100,000, M = F peak age of onset at 40–60 years.Characteristic EEG (diffuse slowing, a characteristic triphasic pattern, periodic sharp wave complexes).
HIV	Psychomotor slowing, memory impairments and reduced concentration, with subcortical picture, apathy, social withdrawal and irritability or emotional lability common. Insight maintained initially.	Due to direct neurotoxic effects, widespread inflammatory changes and neuronal loss. Linked with AIDS.
Syphilis	See the section 'Syphilis'.	
Viral encephalitis	Altered consciousness, confusion, seizures dysphasia, movement disorders, sensory changes, abnormal behaviour, hallucinations and nightmares.	Various infective agents – tick-borne viruses, measles, mumps, rubella, rabies, herpes simplex (commonest in the United Kingdom).
Autoimmune		
Limbic encephalitis	Marked confusion, irritability, agitation, depression, sleep disturbances, short-term memory loss, speech abnormalities, hallucinations, delusions and persecutory ideas. May be mistaken for other psychiatric diagnoses.	May be linked to tumours and specific antibodies (paraneoplastic) or idiopathic (non-paraneoplastic). Seizures very common. Includes anti-NMDA receptor encephalitis, Hashimoto's encephalitis.

Abbreviations: AIDS, acquired immune deficiency syndrome; CJD, Creutzfeldt–Jakob disease; EEG, electroencephalogram; HIV, human immunodeficiency virus; NMDA, N-methyl-D-aspartate.

pain of arthritis may exacerbate or lead to depression and even attempted suicide; hypertension and hypercholesterolaemia need treatment to reduce the risk of stroke disease, which is linked to depression and vascular dementia. In younger patients, psychotic symptoms may be related to space occupying lesions in the brain or metabolic disorders, e.g. porphyria (as in the case of King George III).

EXERCISE 12.1

What do you understand as the differences between dementia and delirium?

Why is this relevant to you?

Dementia and delirium are the organic conditions you are most likely to come across in both general medicine and psychiatry, as well as in the general hospital setting, where you are likely to spend time as a junior doctor. They are mainly disorders of older people, but both are well recognized in younger age groups, especially delirium which is common in children. Dementia and delirium significantly affect outcomes, increase mortality both in hospital and after and increase associated morbidity, independent of other risk factors (Figure 12.1). Given the increasing ageing population and especially the ageing hospital population, all doctors need to have a basic understanding of these disorders and their management. This is particularly relevant when one considers the co-morbidity that elderly patients may have and their increased vulnerability to delirium.

Classification of organic disorders

Organic disorders can be classified using the World Health Organization's *International Classification of Disease*, 10th Edition (ICD-10). The classification for conditions that are not a specific dementia classify the presentation and not the underlying organic cause (in the case of postencephalitic syndrome, the cause of the encephalitis is not specified). For organic disorders with a non-specific cause, readers are referred to the specific chapters related to the disorder for further information on presentation and management. For example,

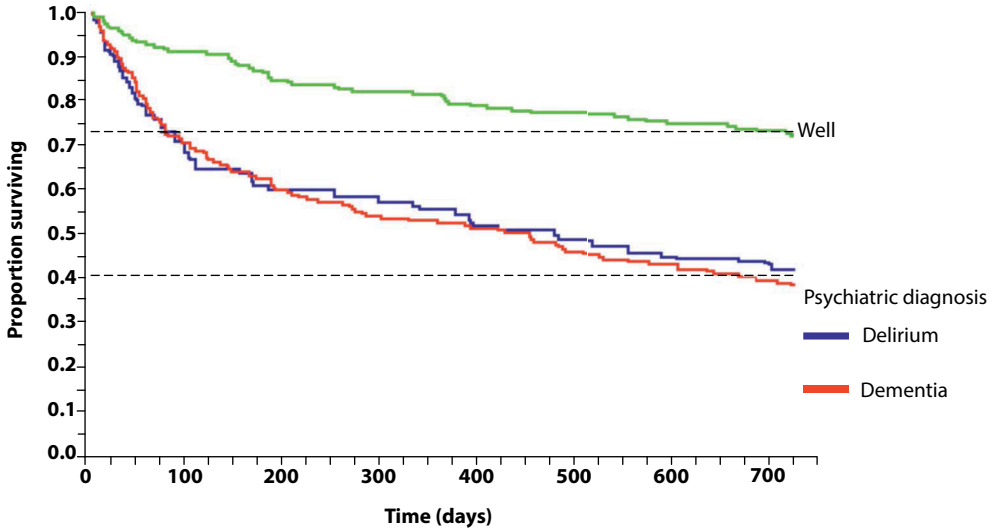

Figure 12.1 Survival after hip fracture in patients with no psychiatric diagnosis, dementia or delirium. Those with organic disorders have a significantly reduced 2-year survival rate when corrected for other confounding factors. (Reprinted from *The Lancet*, Vol. 357, Nightingale S, Holmes J, Mason J, House A, Psychiatric illness and mortality after hip fracture, 1264 -1265, Copyright 2001, with permission from Elsevier.)

mental health problems secondary to head injury, such as organic mood disorder, would be managed similarly to a functional mood disorder.

Dementia

Dementia is defined as a syndrome due to disease of the brain that is chronic or progressive in nature, and involves disturbances in multiple higher cortical functions in the absence of clouding of consciousness. It may be accompanied by deterioration in emotional control, social behaviour or motivation. Although it often presents with memory loss, it also impairs other cognitive functions, leading to dysfunction in daily living. Symptoms should be present for 6 months or longer for the diagnosis (any shorter, delirium or other disorders should be considered). It is a common disorder with an estimated current number of sufferers at about 800–850,000 in the United Kingdom. This number will continue to increase as the proportion of the population over 65 years old continues to expand. The prevalence of dementia increases with age. Different studies have found varying levels of prevalence, but generally prevalence doubles every 5 years. The rates at 65–70 years are about 1%, increasing to about 10% in those aged 80–85 years and then to 25% in those aged 85–90 years. In the over-90s, the rates are 30–40% (Figure 12.2).

There are many causes of dementia, some common and some rare (Table 12.2). The actual prevalence of specific causes is difficult to determine, as, despite the international criteria, the prevalence varies from study to study depending upon the techniques used to determine cases and the populations studied. Alzheimer's disease (AD) is the most common type, affecting more than 50% of patients with dementia.

Classification

Dementias can be divided into two main groups, the primary degenerative dementias (including AD, Lewy body dementia and frontotemporal dementia [FTD]) and secondary (including vascular dementia).

Another division sometimes used is between cortical and subcortical dementias, referring to the primary area of the brain affected by the pathology. However, because of the multiplicity of intracerebral connections this should not be considered the only region affected. Cortical dementias include AD, and are characterized by amnesia, dysphasia, apraxia and agnosia. Subcortical dementias, including Huntington's disease, dementia due to Parkinson's disease, vitamin deficiencies, infections and metabolic states, are characterized by slowed cognition, slowed motor symptoms, memory impairments and changes in personality and mood.

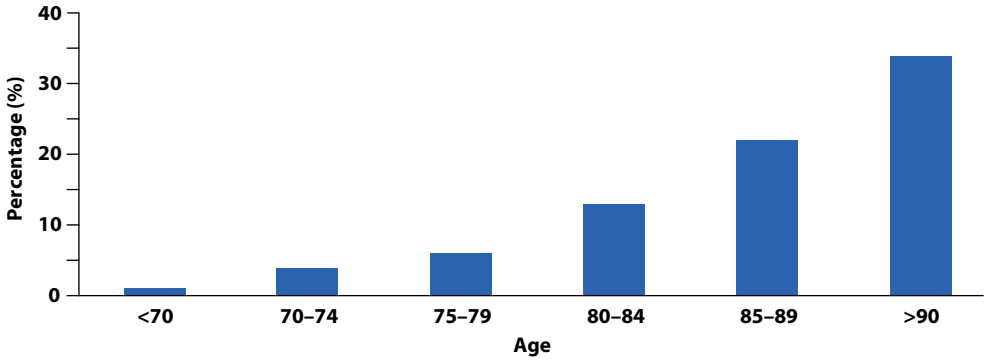

Figure 12.2 Prevalence of dementia with age.

Table 12.2 Causes of dementia

Primary/degenerative	AD (50%), vascular dementia (20–25% with some regional variations, e.g. up to 50% in Japan), Lewy body dementia (10–15%) FTD (about 5%), Huntington's disease, Parkinson's disease, progressive supranuclear palsy
Tumours	Cerebral tumours, remote effects of carcinoma, lymphomas
Trauma	Infection, subdural haematoma, dementia pugilistica, post-severe head injury
Infection	Neurosyphilis (including general paresis), CJD, other prion diseases (e.g. kuru), HIV-associated dementia, chronic infection with tuberculosis or fungi, post-severe meningitis, progressive multifocal leukoencephalopathy, chronic rubella encephalitis
Neurological	Communicating and non-communicating hydrocephalus, progressive myoclonic epilepsy, neuroacanthocytosis, cranial arteritis, subacute sclerosing panencephalitis, motor neurone disease, multiple sclerosis, cerebellar ataxias, limbic encephalomyelitis, 'epileptic dementia', Steele–Richardson–Olszewski syndrome
Metabolic	Renal failure, dialysis dementia, hepatic encephalopathy, inborn errors of metabolism, Wilson's disease, lysosomal storage diseases
Endocrine	Myxoedema, Addison's disease, hypo/hyperthyroidism, hyper/hypoparathyroidism, Cushing's syndrome, hypopituitarism, hypoglycaemia
Toxins	Alcoholic dementia (including Korsakoff's syndrome), chronic drug usage, heavy metal poisoning, industrial agents (e.g. toluene), carbon monoxide
Anoxic	Anaemia, congestive cardiac failure, chronic pulmonary disease, post-cardiac arrest
Systemic	Vitamin deficiency (B12, thiamine, nicotinic acid), folic acid deficiency, porphyria, Whipple's disease, systemic lupus erythematosus, sarcoidosis, xeroderma pigmentosum

Abbreviations: AD, Alzheimer's disease; FTD, frontotemporal disease.

Alzheimer's disease

Risk factors

The biggest risk factor for AD is age. The risk to first-degree relatives of patients with AD who developed the disorder at any time up to the age of 85 years is increased some threefold to fourfold relative to the risk in controls with no family history. People with Down syndrome will develop Alzheimer changes in the brain by the age of 40 years. A history of cardiovascular disease (CVD), Parkinson's disease, hypothyroidism or significant head injury also increases the risk. AD is equally common in males and females. Even when there is a family history of AD, the risk to other family members cannot be accurately determined.

A known genetic risk factor is inheritance of the *E4* allele of the *apolipoprotein E* gene. Among patients with AD, around 40%–80% have at least one

apolipoprotein E4 (*APOE4*) allele. Heterozygosity for this allele increases the risk of AD by a factor of three and homozygosity increases it by a factor of 15. Around 0.1% of cases arise as an autosomal dominant familial AD due to inheritance of mutations in genes relating to amyloid precursor protein (APP) and presenilins 1 and 2.

Pathology

Macroscopically, senile plaques (SPs) are found extracellularly in cerebral cortical grey matter, hippocampus and certain subcortical nuclei. Neurofibrillary tangles (NFT) are found intracellularly, particularly in the hippocampus and cerebral cortex. Amyloid deposits may be found in leptomeningeal and cortical blood vessel walls. These findings are associated with cell loss, often of 30% or more, and reduced synapses. The extent of cell loss, SP counts and NFT counts correlates with the degree of dementia. Macroscopically, there is reduced brain volume and weight and generalized atrophy (greater than that expected for normal ageing), often more prominent in the medial temporal lobe and parietal lobes, along with enlarged ventricles, all of which may be evident on computed tomography (CT) or magnetic resonance imaging (MRI) scans as well as at post-mortem (Figure 12.3).

The underlying biochemical pathology is complex and not yet fully understood. The SPs are made up of deposits of beta amyloid (Aβ). Aβ comes from APP which seems to be metabolized differently in patients with AD. The aggregation of Aβ appears to damage the neurones though the final SP itself is relatively inert and acts only as a marker of the disease process. Abnormal phosphorylation of tau protein results in damage to the intracellular microtubules that are essential to intracellular transport. This results in formation of the NFTs.

The synaptic damage resulting from the above pathology results in deficits in function of a number of neurotransmitter systems, including acetylcholine, noradrenalin, serotonin and dopamine. The cholinergic deficits, particularly those arising from the basal forebrain nuclei, are important in relation to the cognitive deficits as these nuclei project widely to the cerebral cortex and to the hippocampus, which is important in memory processes. Cholinergic neurones are lost, there is depletion of acetylcholine, nicotinic receptors are depleted and, later on, muscarinic receptors become depleted. This provides the theoretical basis for the only available pharmacological treatments, the cholinesterase inhibitors. These drugs principally inhibit the enzyme that inactivates acetylcholine in the synapse.

Presentation

Most commonly, this is seen in community settings and characteristically is slow and gradual. Memory problems and changes in behaviour are most often

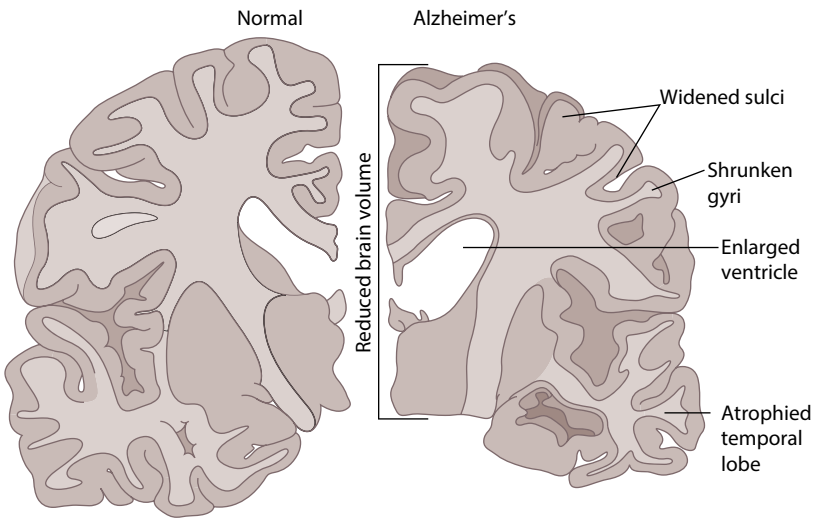

Figure 12.3 Diagram showing differences between normal brain (left) and Alzheimer's disease affected brain, with atrophy, wide sulci, narrow gyri and enlarged ventricles (right).

noted by carers or family rather than the patient though will often be attributed initially as normal ageing. In AD, the onset is insidious, so that the disease process is well established and the problems can be significant by the time of presentation to healthcare services. Deterioration in self-care and repetitive phone calls, episodes of wandering, missed appointments, dangerous lapses of memory, getting lost and failure to learn new routines or other important information can also lead to presentation to services. Some patients will present themselves, especially those of higher premorbid intellectual abilities who may notice that their memory or other cognitive skills are deteriorating. Even in the early stages, patients often have little insight into their difficulties, minimize their difficulties or blame the changes on getting old.

There is a slow intellectual decline in the early stages of the disease, with behaviour and general functioning within normal limits. The initial stages can last from 2 to 5 years, and patients may develop coping strategies to cover their deficits.

In AD the main symptoms can be divided into five symptom areas or 'the five A's of Alzheimer's disease as follows:

- Amnesia
- Agnosia
- Aphasia
- Apraxia
- Associated behaviours

Amnesia

The amnesia or memory decline in Alzheimer's is typically slow but progressive, with forgetfulness and the failure to retain newly learned information. Short-term memory is affected at first, and relatives will often report that patients ask the same question repeatedly, or need to be told the same piece of information repeatedly. Recent events or important dates are forgotten and items, such as bank cards or keys, can be misplaced. They find they cannot retrace their steps to relocate the misplaced object or recall information at all, even with prompts.

Patients have difficulties laying down new memories and retrieving older ones. They will adopt strategies to aid memory function, such as writing things down, or getting family members to help out with tasks that they could manage previously. As the dementia progresses, the misplacing of items can lead to the patient accusing people of stealing their belongings.

Initially distant memories are preserved, so they can give full details about their early life, but as the disorder progresses their long-term memories become fewer and confused, e.g. they may mix up children and grandchildren. They may begin to live in the past and may believe that their parents are still alive (occasionally this may be true, hence the importance of informant histories!). The patient may learn to confabulate to fill the gaps, but some of the confabulations may be true memories. They may try to go to work, despite having retired many years previously, or set the table for deceased family members.

The patient becomes disorientated, usually initially in time, losing track of dates and days, progressing to the season and years. They may even lose day–night awareness. The disorientation progresses to disorientation in place, so they may not recognize clinics or hospitals. They can lose awareness of their surroundings and this may progress to a state where patients no longer recognize their own home (although they may recognize where they used to live) or local environment, which can lead to wandering. If they go out, they may need to be helped home, as they lose track of where they are and what they are doing. Disorientation in person tends to deteriorate later than in time. Orientation in age tends to be lost early on.

Agnosia

Agnosia is the inability to recognize external sensory stimuli in any modality correctly, with relatively intact sensory pathways, which is displayed as the inability to recognize objects and people. Initially, this may be an inability to be able to put names to faces, but may deteriorate to not recognizing family members or even spouses. They may believe that the other person is an intruder and this can occasionally lead to the patient attacking the carer. Patients may not even recognize themselves, which can lead to 'the mirror sign', where sufferers see their reflection in a mirror and will talk to it as if it is another real person. They may not recognize everyday objects, which can lead to abnormal behaviour, such as urinating in a waste paper basket, believing it to be a toilet bowl. This is exacerbated by the general decline with age of the senses, especially vision and hearing.

Apraxia

Apraxia is the decreasing ability to perform coordinated motor tasks, with an intact peripheral neuromuscular system, which begins to develop as the dementia becomes moderately severe. This causes problems with activities of daily living, such as difficulties with dressing, and may result in problems with writing and other tasks (usually in neuropsychological tests) as ideational apraxia develops (this is the loss of ability to conceptualize, plan and execute the complex sequence of motor actions involving the use of tools or objects in everyday life). Significant problems arise when the patient can no longer perform important tasks, such as using cutlery or providing themselves with food or drink.

Aphasia

Strictly speaking, aphasia does not occur until late in the dementia, and rather patients have dysphasias. In the early stages, they may have difficulty in participating in conversations because they have difficulty following the thread or may find that they get stuck and do not know how to continue. The patient's vocabulary becomes contracted, and the ability to express themselves becomes reduced, as does the ability to use metaphors and analogies. They will have increasing difficulty in word finding over and above the normal levels, to the extent it becomes noticeable to others. The ability to name objects diminishes so that they describe objects or use alternative descriptions, e.g. a pen may be called 'that thing you write with' or 'a word scribbler'. Perseveration, characteristic of organic problems, may occur, often repeating the same segments of thought or stories repeatedly at interview. Speech abnormalities and semantic errors increase in frequency and marked dysphasia, both expressive and receptive, can occur (related to effects of the pathological processes in Broca's area and Wernicke's area, respectively). Patients can get very frustrated by expressive dysphasia, as they are aware of what they are trying to say but cannot get the right words out.

Associated behaviours

These can be divided into three areas, the psychiatric symptoms, the behavioural symptoms and personality changes. It is usually these that produce the most stress for carers and lead to alternative care.

Psychiatric symptoms

A full range of psychiatric symptoms may occur in dementia. Depression is common in dementia (~20%–25% of patients), possibly related to the preservation of insight and less to cognitive impairment. It tends to present early, and symptoms of irritability, apathy, retardation and agitation may be present as well as typical symptoms of depression.

Psychotic features are relatively common, particularly delusions (in about 15%), which are often persecutory (often of theft or persecution by neighbours or carers). Delusions, often linked to agnosia, of infidelity, misidentification (including the patient's own reflection), believing intruders are (or have been) in the house and not believing that one's own house is one's own home may occur. These can be distressing to carers or relatives, especially spouses, and may develop into delusional misidentification or the non-recognition may even lead to aggression, e.g. believing that a spouse is a burglar or intruder. Hallucinations are common, are usually visual or auditory and occur in 10%–15% of patients.

Personality changes

These occur as the disease progresses, with the patient frequently displaying accentuated features of their premorbid personality and become more egocentric. They may also display behaviour out of their character, such as sexual disinhibition or antisocial behaviour. They will become much more fixed and rigid in their routines and will have difficulty coping if these change. They gradually stop their usual interests and as the dementia progresses are able to spend the day doing very little. This apathy tends to be more distressing to carers than the patient. Emotional responsiveness and emotional control become reduced, the latter to the point where they can have 'catastrophic reactions', where there is a disproportionate outburst of anger or even aggression when they are pushed beyond their abilities (occasionally seen during cognitive assessment).

Behavioural symptoms

These are important to elicit as they are the most likely cause of admission to 24-hour care. They cause carer distress and may pose significant risk to the patient

and others. They include wandering, aggression, incontinence, disinhibition (both social and sexual), sleep disturbance and marked apathy. Patients' behaviours may deteriorate around teatime (known as sundowning), which may make care more difficult at this time. Aggression may range from irritability to verbal hostility and aggression, but physical violence is uncommon. In later stages, the aggression may be related to resistance to carer interventions (or other residents in 24-hour care settings) with swearing or lashing out, but direct violence is rare. Any underlying reasons for the behaviour should be examined, such as pain or delirium.

Wandering is a common symptom. It ranges from restlessness and pacing about their residence to leaving the house and getting lost, needing help to get home or having to be located by police searches. There may be intent in the wandering, as some believe they are trying to get home or go to work.

Appetite may be reduced with significant weight loss, with patients eating very little, forgetting to eat or only eating when food is provided. Supplements may be needed, but other causes of weight loss should be considered before attributing it to the dementia.

Disinhibition is not common and may range from disinhibited conversation to sexual acts such as exposing genitals or soliciting intercourse.

Incontinence may be due to several factors and can be difficult to cope with. Patients may not recall where the toilet is or not be able to reach it in time or there may be agnosias. Co-morbid conditions need to be considered, for example, constipation and urinary tract infections. Careful toileting and management of fluids is helpful, as well as treating other pathologies.

Sleep disturbance may be problematic as patients wander around at night, keeping their carers awake. Older adults require less sleep generally but disturbed sleep can be exhausting for carers. Nocturnal wakefulness can lead to daytime sleepiness and the sleep–wake cycle, initially normal in the early stages, may become reversed.

VIDEO 12.1

A woman who couldn't remember: delirium associated with Alzheimer's dementia – https://vimeo.com/28811456

Progression and prognosis

The abilities and cognitive functions of the patient gradually deteriorate. They gradually become inefficient or confused with activities of daily living, including washing, dressing, feeding, taking medication and so on. Superficially, the patient may appear cognitively intact, but testing reveals impairments of judgement and reasoning, and abstract thinking also becomes impaired. They may have poor judgement over money matters and have difficulty managing their affairs. They may withdraw large amounts from the bank, carry it around with them, storing it at home (often misplacing or losing it) or spending it and not knowing how it has been spent. They may forget to pay bills or sign up for services they do not need. They become vulnerable to exploitation from criminals or even their family or carers.

Patients eventually lose all ability to self-care and become difficult to engage with, inhabiting a world of their own; they resist interventions and require full nursing care as the link between body and brain is broken. The disease shortens the expected lifespan, with the average time from onset to death about 7–8 years, often due to other illnesses such as pneumonia.

Vascular dementia

Vascular dementia is the second most common cause of dementia. It is related to arteriosclerosis, with the underlying pathology being strokes, usually multiple and small. Risk factors are as for other vascular or ischaemic diseases, such as smoking, hypertension, hyperlipidaemia, ischaemic heart disease, peripheral vascular disease, valvular heart disease, atrial fibrillation and diabetes. A family history of stroke and/or vascular dementia is also a risk. Unsurprisingly, those ethnic groups at high risk of cardiovascular disorders (such as Indian, Bangladeshi, Pakistani and Sri Lankan) are also at increased risk of vascular dementia. It is more common in males.

There are essentially three main types of vascular dementia – the first caused by stroke, the second by small vessel disease and the third being a combination of the two. Dementia from strokes may develop after a single event (such as a single stroke) or may be secondary to multiple smaller ones (often called multi-infarct dementia), and onset can be relatively

quick. Dementia caused by small vessel disease is, as the name implies, caused by damage to small blood vessels in the brain. If the damage is primarily in the deep white matter regions, the diagnosis is referred to as subcortical vascular dementia (in a severe form, it is called Binswanger's disease). The damage is the result of the thickening and narrowing (atherosclerosis) of arteries that feed the subcortical areas of the brain. The symptoms develop more gradually than those of stroke and are often accompanied by walking problems. Symptoms are similar to Alzheimer's, but due to the nature of strokes the presentations may be variable. Affective symptoms may present earlier and typically there are sudden deteriorations in the cognitive functions linked to further cerebrovascular events, classically with a stepwise progression (Figure 12.4). On examination, there may be physical signs, such as focal neurology. On cognitive assessment, patients may score highly on screening tests but have marked deficits on day-to-day functioning. Any underlying vascular risk factors should be screened for and actively managed, as this reduces the risk of further cerebrovascular events. It is also worth educating those at risk to minimize the frequency of ongoing transient ischaemic attacks, because although these in themselves may not cause any ongoing or permanent damage, they are a strong signal that all is not well.

Lewy body dementia

This accounts for about 15% of dementias and is more common in males. The age of onset is slightly earlier than AD typically in the sixth or seventh decade. Diagnosis is made clinically, but pathologically there is generalized atrophy and microscopically Lewy bodies are seen intracellularly, although there is no correlation between the number present and the degree of dementia (and they can also be found in Parkinson's disease and AD).

Presentations vary, but clinically there is a cortical dementia. Cognition may be fluctuant with pronounced variations in orientation and alertness (which may even mimic delirium). Performance on cognitive tests of attention, visuospatial function and the frontal lobe may show marked impairment, but memory impairment is not great in the early stages. About 40%–50% have depressive episodes. There may be recurrent detailed visual hallucinations (auditory hallucinations also occur frequently) and delusions (in about 70% of patients). Patients develop spontaneous motor features of parkinsonism including bradykinesia, tremor, rigidity and mask-like features. There will be recurrent falls or episodes of syncope. Care must be taken in treating the psychotic symptoms as there is marked neuroleptic sensitivity, and reactions may be severe or even fatal.

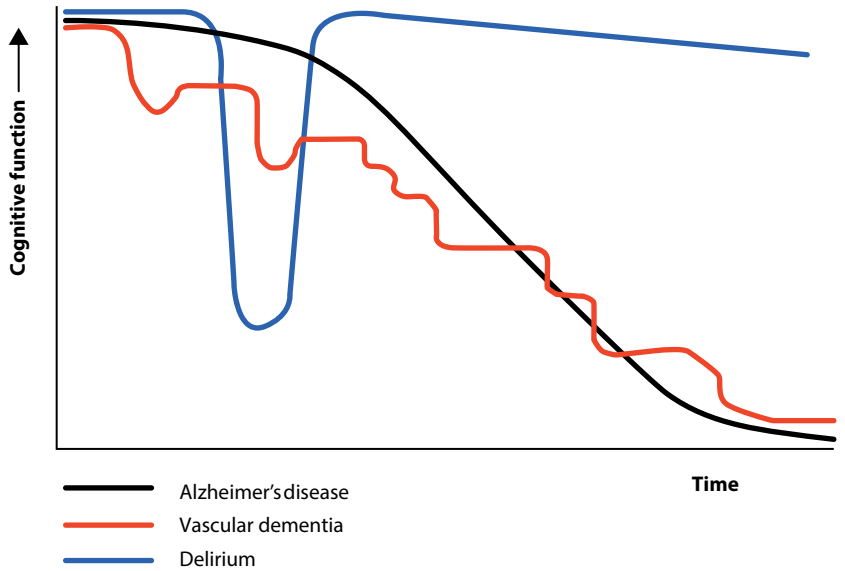

Figure 12.4 Diagrammatic representation of the progression of Alzheimer's disease (gradual deterioration), vascular dementia (stepwise progression) and delirium (sudden crisis with resolution).

Frontotemporal dementia

This is a group of dementias with prominent changes in personality, social conduct, language and understanding, and includes Pick's disease. The onset is insidious with slow progression, and the age of onset tends to be younger (50–60 years). It is uncommon but is probably underdiagnosed, with prevalence rates of 5%–15%. Prevalence rates tend to be higher in the younger age ranges. It is more common in patients in younger age groups with early-onset dementia. There is a family history in up to 20% of cases.

There are three subtypes which are as follows:

- Behavioural variant FTD
- Predominant frontal lobe involvement, with changes in personality, behaviour, interpersonal and executive skills
- Progressive non-fluent aphasia (PNFA)
- Predominant temporal lobe involvement, with loss of ability to produce or understand language
- Semantic dementia

Loss of memory of the knowledge of things and concepts. Semantic memory is knowledge store about objects, concepts and the meaning of words based on the patients experience of the world.

Although originally termed Pick's disease, only 20%–30% of patients with FTD have histopathological changes of Pick's disease (intracellular Pick bodies).

Core diagnostic features are insidious onset and gradual progression, with early loss of insight, and an early decline in social interpersonal conduct and impairment in regulation of personal conduct (including social disinhibition, neglect of affairs and responsibilities, loss of interest in personal appearance and hygiene).

Striking changes in personality occur and the patient may present as overactive, socially disinhibited and fatuous, or conversely as apathetic, inert and emotionally blunted. Patients lack appropriate basic emotions, such as sadness, and social emotions, such as sympathy and empathy. Inflexibility and mental rigidity may occur, but patients may also demonstrate distractibility, impulsivity or disinhibition. The presence of repetitive, stereotyped behaviours (e.g. rocking, marching on the spot or mannerisms such as singing, dancing or hoarding) and hyperorality (Klüver–Bucy syndrome – ingesting inedible objects) or other unrestrained exploration of objects in the patient's environment is supportive of the diagnosis. Affective symptoms including depression, anxiety, hypochondriasis and bizarre somatic preoccupations are also recognized symptoms.

Language becomes progressively impaired, with reduced output, eventually becoming mute. The content is empty and there are word-finding difficulties and poor verbal fluency. Echolalia and perseveration are common.

Cognitive testing shows frontal lobe dysfunction (reduced verbal fluency and difficulty with abstraction, set-shifting and trail-making tests) but there is relative sparing of memory, orientation and visuospatial abilities. CT scans will show frontal and temporal lobe atrophy, but the aetiology of FTD is unclear. The differential diagnosis includes atypical presentations of functional psychiatric disorders, atypical AD or other organic disorders, e.g. tumours, cerebrovascular disease. Treatment is symptomatic combined with good social and carer support. Cholinesterase inhibitors should be avoided as they may worsen the disorder.

Dementia in Huntington's disease

Huntington's disease is an autosomal dominant inherited disorder, with the gene linked to the disorder located on the proximal arm of chromosome 4. It usually presents in the fourth decade of life, but there is a wide variation in the age of onset. It is uncommon, with prevalence rates varying from 4 to 7 per 100,000 of the population, although there are regional variations. There will be family history with males and females affected equally.

Psychiatric symptoms tend to precede motor symptoms and dementia symptoms occur early in the disease, with a subcortical picture, including non-specific memory disturbances, slowed cognition and apathy. Frontal lobe and executive functions are affected early, but there is a lack of language disorder. Patients are easily distractible and exhibit poor visuospatial functioning. Depressive symptoms are common and insight is retained, with suicide accounting for 5%–10% of deaths. Prognosis is poor and the disease is inevitably fatal, but progression is slow with the average duration of the disorder being around 14–15 years. This is probably one of the most distressing diseases that families have to deal with. Services are generally poor but genetic testing is available.

Dementia in Parkinson's disease

Unfortunately, dementia is an integral part of this illness, with increased incidence and prevalence with advanced disease. In total, 20%–40% of patients develop a dementia later in the illness. There is a subcortical pattern as discussed under the section 'Classification', with slowing of cognition, apathy, decreased intellectual function and memory disturbance. Frontal and executive symptoms are common and motor symptoms may make the confusion appear worse. The dementia worsens the delirium-producing effects of L-dopa medication.

There is a degree of overlap in symptoms and histopathological changes both of AD and of Lewy body dementia, with evidence of both being present. It is most likely that there is a continuum and overlap between these disorders. There may be psychotic symptoms, including hallucinations and persecutory delusions.

Potentially treatable dementias

While these are not common, treatment may reduce or even reverse the level of dementia and so are covered here.

Vitamin deficiency

Although psychiatric manifestations of vitamin B12 or folic acid deficiency are rare, these deficiencies may present as dementias. Patients with dementia have poor nutrition so it may be difficult to determine cause and effect. Treatment of both deficiencies reverses the dementia, although improvement can be slow.

Depression

This is an important differential diagnosis to consider and is covered in Chapter 15 under 'Pseudodementia'.

Thyroid deficiency

Thyroid dysfunction can cause psychiatric symptoms, and in hypothyroid states a dementia can develop which clinically is not distinguishable from AD. Diagnosis is aided by other signs and symptoms of hypothyroidism, thyroid function blood tests and the patient's response to the treatment. Hypothyroidism is a risk factor for AD, with an increased relative risk of about 2.3.

Normal pressure hydrocephalus

This is rare but usually presents with the classic triad of symptoms of ataxia, urinary incontinence and dementia. The cognitive problems are often the first symptoms and are associated with a general slowing and apathy that may resemble depression or FTD. There may be a history of significant head injury, subarachnoid haemorrhage or cerebral infection. CT or MRI scan can show enlarged ventricles but not the widening of sulci associated with Alzheimer's. Treatment is the insertion of a shunt, but the benefits need to be balanced against the risks and complications of surgery.

Subdural haematoma

This usually occurs after a head injury, although in older people, where this is most common, the actual injury may be minor or even not recalled. Physical signs are often minimal, but there is frequently an associated apathy or psychomotor slowing. Headache is not a common symptom and a confirmed diagnosis may only become apparent on CT scan because of the varied presentations.

Alcohol and other drugs

Chronic alcohol abuse can lead to the development of a dementia, as well as an increase in the risk of Wernicke's encephalopathy, Korsakoff's syndrome and vitamin B12 deficiency-related cognitive problems. This is probably due to the direct toxic effects of alcohol on brain cells, and up to 50% of heavy drinkers over 65 years have some cognitive impairment.

Many drugs, particularly anticonvulsants, may occasionally cause cognitive impairment to a degree that mimics dementia. Withdrawal of the offending drug may cause a reversal of symptoms, although improvements may be slow.

Syphilis

The psychiatric symptoms emerge in the tertiary stages of syphilis and constitute neurosyphilis, or general paresis of the insane (GPI) which is linked to spirochaetes (*Treponema pallidum*) in the brain tissue. Although now rare in the developed world, as syphilis is generally treated before this stage is reached, the

psychiatric sequelae are notable and may increase in the future because of the increasing rates of syphilis across the world, particularly associated with human immunodeficiency virus (HIV) infection.

About half of the cases present with uncharacteristic behaviours such as crimes, violence or recklessness with associated personality changes. Classically, GPI can be divided into subcategories relating to the clinical presentation including dementing (with memory impairment, slowed thinking, apathy, lethargy and loss of insight) and grandiose (with euphoria and delusions of power and grandeur, frequently with delusions of being a famous person).

Diagnosis depends upon a high index of suspicion combined with positive syphilis serology performed when neurosyphilis is suspected or clinically indicated and examination of the cerebrospinal fluid.

Assessment and management of dementia

Assessment

The most important part of the assessment is the history. A good history will often give the diagnosis before any cognitive testing or other investigations. The patient might not tell you that anything is wrong and have no insight into their problems, or in the early stages they may be aware of some deficits but may minimize the effects they are having. It is therefore imperative to get an informant history. As well as being able to provide useful information on the patient's symptoms, they can confirm or complete biographical details or provide necessary information, such as past medical history.

If the informant is present during the interview, they should be asked not to answer for the patient if possible so that any differences in accounts can be ascertained (although this needs to be done tactfully and the reasons explained – this is particularly important during cognitive testing!). All the domains of a psychiatric history need to be covered as all are relevant to the differential diagnosis.

The structure of the history is as described in Chapter 3. Specific issues to highlight are as follows:

- Current disorders that may affect functioning or cognition, or be risk factors for the dementia, previous disorders that are risk factors.
- Risk factors of and for depression, other psychiatric illness that may be part of the differential diagnosis.
- The more severe the dementia, the less coherent the personal history may become with gaps, errors, mixing up of details and confabulations.
- Use of drugs that affect cognition, smoking as a safety issue (leaves burning stubs) and risk factor, alcohol as an agent affecting cognition, risk for vitamin deficiency, delirium, possible primary cause.
- Factors that may raise concern for self-care and safety.
- How were they before they developed the dementia? What has changed?

Mental state examination

This may show in deterioration in self-care (unshaven, unwashed, food stains on clothes, disorganized clothing, unsuitable footwear, etc.), frequently looking to informants for support for answers, poor concentration, mood change, speech and language phenomena (such as perseveration and confabulation) and any psychotic phenomena. Insight can be assessed.

VIDEO 12.2

Mini Mental State Examination – https://vimeo.com/28816445

Cognitive assessment

A cognitive assessment is vital to look for deficits in cognitive function that are not apparent from the history, to assess the level of impairment and to provide a baseline to monitor progress. The main cognitive domains to assess are orientation in time, place and person, attention and concentration, memory (both anterograde and retrograde), language and praxis or domains indicated by the history. A person's ability to complete the test should be optimized; for example hearing aids should work, spectacles should be clean.

Cognitive impairment screening tests

There are several screening tests for cognitive impairment. It is important to remember that these are

not diagnostic and only show evidence of cognitive impairment. You should then look for the reason for that impairment, which may be dementia, but may be due to other causes, such as delirium, schizophrenia, etc. The results should be contextualized for the patient – someone who is illiterate may perform artificially poorly, those of high intelligence may not show deficits on screening tests until later in the illness. Several are readily accessible via the Internet.

The abbreviated mental test score (AMTS) is a 10-item test that has been recommended by the Royal College of Physicians and the British Geriatric Society for routine cognitive assessment in secondary care settings (see Box 12.1). A score of less than 8 suggests that further testing is warranted. The Six-item Cognitive Impairment Test (6-CIT) and the General Practitioner Assessment of Cognition (GPCOG) are available for use in primary care. In secondary care the mini-mental state examination (MMSE) has been widely used for many years but has now been copyrighted. The score is susceptible to the effects of age, educational level, language skills and good vision and hearing. It also has relatively little testing of memory and is not sensitive to early dementia. Studies have shown correlations between MMSE and AMTS scores and density of pathological lesions in the brains of patients with dementia. The Montreal Cognitive Assessment (MoCA) is now available for use and can be downloaded from the Internet. In psychiatric settings, the Addenbrookes Cognitive Examination-III (ACE-III), a more inclusive test that covers a wider range of domains in more depth, is recommended. Clock drawing can also be used as a quick and easy screening assessment. The patient is asked to draw a conventional clock face with the hands in separate quadrants, e.g. with the time at 3:55 or 7:10, etc. Although there are formal scoring schemes for this test, patients with dementia will draw abnormal pictures; the more severe the dementia, the more distorted or incorrect the images becomes. It is included in both the MoCA and ACE-III.

Box 12.1 Items in the AMTS (Hodkinson, 1972)

- What is your age?
- What is the time to the nearest hour?
- Give the patient an address, and ask him or her to repeat it at the end of the test, e.g. 42 West Street.
- What is the year?
- What is the name of the hospital or number of the residence where the patient is situated?
- Can the patient recognize two persons (the doctor, nurse, home help, etc.)?
- What is your date of birth? (Day and month is sufficient.)
- In what year did World War I begin? (Other dates can be used but should be events reasonably distant in time.)
- Name the present monarch.
- Count backwards from 20 down to one.

Other investigations

Bloods

A full battery of blood tests should be performed looking for treatable causes of dementia and for any other pathology that might be worsening the condition. Risk factors for vascular dementia can be monitored. This should include full blood count (looking for anaemias), urea and electrolytes (renal impairment), blood sugar (diabetes), B12 and folate, thyroid function tests, and possibly syphilis serology (if suspected, as this is no longer recommended routinely). In patients with vascular risk factors, e.g. hypertension, diabetes etc., it may also be worth testing blood lipids and cholesterol.

Brain Imaging

CT is valuable in establishing the cause of dementia, as there are characteristic changes in particular dementias. These may be more obvious on MRI scanning, but these are not universally available. Characteristic findings on CT are given in Table 12.3. A CT scan may also show rare causes, such as normal pressure hydrocephalus or space occupying lesions. Functional imaging, such as single photon emission computerized tomography (SPECT) or positron emission computerised tomography (PET), may also be useful in measuring cerebral blood flow. Other investigations such as electroencephalograms (EEGs) can be useful in Creutzfeldt–Jakob disease (CJD) and FTD, but are not routinely used. DAT scans (imaging of the dopamine transporter) can be used in Parkinson's disease and Lewy body dementia.

Table 12.3 CT or MRI scan findings of common dementias

Dementia type	CT or MRI findings
AD	Reduced brain volume and generalized atrophy (or involutional change), often more prominent in the medial temporal lobe
	Widened sulci, smaller gyri
	Enlarged ventricles
Vascular	Periventricular ischaemia
	Ischaemic changes in other areas, especially deep white matter
	Multiple infarcts
Mixed	Periventricular ischaemia
	Ischaemic changes in other areas, especially deep white matter
Lewy body	Generalized atrophy (though 40% have preserved medial temporal lobe structures)
Frontotemporal	Atrophy of the frontal and temporal lobes

Abbreviations: AD, Alzheimer's disease; CT, computed tomography; MRI, magnetic resonance imaging.

Diagnosis

Older people and their relatives are often afraid of the diagnosis of dementia often based around misconceptions and stereotyped views of care. Care and thought should be given to breaking the bad news and patients should be supported via specialist services and voluntary sector support such as the Alzheimer's Society post-diagnosis. As well as pharmacological agents, there is much more support available and patients should be encouraged to 'live well with dementia'. Dementia management is that of a chronic disorder, not palliative care.

Management

It is important to consider how to provide a holistic service that ensures that patients with dementia are helped to have as good a quality of life as possible. This may be challenging especially if staff feel insufficiently untrained. Management may also entail working closely with family members (see further below). While it is important to be mindful of the care that people with Alzheimer's might need, especially if they have behaviours such as wandering which put them at risk, it is important not to infantilize them, talk over them or exclude them from decisions about their care. Communication should be short and clear, and there may be a need to gently steer it towards the main focus. It requires patience especially as more and more functioning and memory is lost. Distraction can be a useful technique to get back on track.

Patients with dementia often reminisce as they lose orientation in time and place. If they recall particularly distressing events, they may become acutely distressed as for them the event is happening at that time. Gentle reassurance can offer considerable support.

As a foundation-level doctor, you may be called to manage some of the behavioural problems associated with dementia in patients admitted primarily for medical problems. It is therefore important to be aware of the risks of using medications to manage aggression as this may exacerbate rather than help the situation. Using the principles for managing acute distress is often helpful – keep calm, minimize the number of staff involved, reduce stimuli and provide reassurance. It can be helpful to use an (antecedents, behaviour, and consequences) ABC approach; that is, consider the antecedents (What is potentially causing the behaviour?), the behaviour itself (What does it mean? Who is distressed by it?) and consequences (Is it bringing any rewards? e.g. contact with others). This ensures that if necessary these issues can be addressed before turning to medication.

Another point worth stressing is that people suffering with dementia should not be denied other medical care on the basis of their diagnosis. While it may take more time, it is still important to investigate treatable problems for which even minor interventions can make significant impact on quality of life such as treating infections and pain management. Now we turn to more specific management interventions.

Biological

Any treatable cause of dementia and any co-morbid conditions or risk factors need appropriate treatment or management. Any sensory disability should be minimized; the correct spectacles should be obtained and kept clean. Hearing should be optimized, by keeping wax to a minimum and using an appropriate hearing aid (with batteries that work!). Correct footwear and any mobility aids should be used to prevent falls and keep patients active.

Treatments for the dementia depend on the type. For Alzheimer's, acetylcholinesterase inhibitors were introduced in the late 1990s. These are designed to increase cholinergic transmission by reducing the breakdown of the acetylcholine at the synapse, allowing more to be available for the postsynaptic receptors. Three are currently available: donepezil, galantamine (which also interacts with nicotinic receptors) and rivastigmine. They should be initiated in secondary care and are subject to National Institute for Health and Clinical Excellence guidelines. They are not curative but they do slow the pathological processes and delay institutional care. Response is variable with about a third doing well, a third getting some benefit and another third not responding well. Treatment should be monitored and stopped if there is no benefit. Alternatively the *N*-methyl-D-aspartate (NMDA) receptor blocker memantine can be used.

Adverse effects with these drugs include nausea, vomiting, diarrhoea, headache, anorexia, insomnia, syncope and increased confusion. Less commonly muscle cramps or urinary incontinence may occur. Caution is needed with these drugs in patients with renal or hepatic disease, sick sinus syndrome, conduction abnormalities, susceptibilities to peptic ulceration and asthma or chronic obstructive pulmonary disease.

In vascular dementia, there is no recommended specific treatment for the dementia, but aspirin (or other anti-platelet agents) may help reduce further strokes or ischaemia, although its efficacy in preventing progression is not clear. It is also important to introduce a tight control of risk factors for heart disease using the general health promotion ('healthy heart') advice. Given that most dementias are of a mixed type, this is actually important for all people with dementia to minimize risk of future deterioration from CVD.

In Lewy body dementia, acetylcholinesterase inhibitors can be used, but antipsychotics should only be used to treat the psychotic symptoms with caution. Antipsychotics will frequently worsen parkinsonian features, may cause unsteadiness and are associated with a marked increase in mortality in patients with Lewy body dementia. FTD should be treated symptomatically.

Behavioural problems should be managed with non-pharmacological interventions if possible, such as effective nursing care, establishing what may lead to distress and avoiding exacerbation of behavioural problems. Patience and gentle reassurance may be required. Many may become acutely distressed and confused, and the principles for managing acute confusional state as discussed below and in Chapter 18 will need to be implemented.

If medication is required for behavioural disturbance, acetylcholinesterase inhibitors or memantine can be tried. Routine use of antipsychotics is not recommended in dementia. For patients with dementia and psychosis, there may be a limited role for the use of antipsychotics in low dose with a suggested time limit of 3 months of use. Currently risperidone may be used but at as low a dose as possible, as patients with dementia are at greater risk of adverse effects. Evidence of increased risk of cerebrovascular events, sedation, falls and decreased cognitive function in elderly patients on antipsychotics has driven guidelines minimizing their use. Medication for sleep disturbance should be kept to a minimum as the risk of falls increases, and daytime drowsiness may occur, exaggerating the underlying problem.

Psychological

Psychological therapies include reality orientation, cognitive stimulation therapy, reminiscence therapy, validation therapy and the use of memory aids, such as labels on items to remind the patient what they are. Various artistic therapies such as music and art therapy may also be beneficial. Patients should be encouraged to keep as active and stimulated as possible, as the adage 'use it or lose it' does seem to have some truth in it.

Social

Social treatment and support are the mainstay of dementia care. Quality of life should be prioritized, so that attention should be paid to adjusting or enhancing the patient's environment to ensure that they can manage. Most patients want to remain at home and, as retaining familiarity can help, this should be the goal

for as long as it is safe and practical. In the early stages this is usually managed through support from family, but as the dementia progresses increased input via social services and home care teams, memory services or other services is required.

Safety needs to be reviewed. Gas appliances may need disconnection and instead microwave ovens may be used for cooking. Meals may need to be provided by meals-on-wheels services or prepared by home care. Heating (particularly use of fires) and access to shopping facilities need to be considered. Respite care (for example, attendance at day centres, sitting services and full 24-hour residential care) may provide some support for carers. The carer role is discussed further below.

Patients diagnosed with dementia should be advised to inform the Driver and Vehicle Licensing Agency (DVLA) and may need to stop driving. Doctors may have to inform the DVLA directly if potentially dangerous driving continues against advice.

The ability to take medication correctly should be monitored and may require supervision. Concordance aids such as dosette boxes or electronic reminders can be used. Treatment regimens should be simplified, with reduced tablet numbers and frequency of administration.

Financial and legal issues warrant early consideration because of capacity issues. Patients should be advised regarding benefits and Lasting Power of Attorney, for financial and health and welfare matters. Will arrangements should be made early. Day-to-day finances may need to be taken over by carers as the disease progresses. Patients become vulnerable to exploitation and should be monitored for elder abuse both financially and socially. Elder abuse is well recognized and it is now an offence under the Mental Capacity Act to wilfully neglect or mistreat a patient.

A move to a flat or sheltered accommodation may be required. This may be disorientating for patients, but there may be few other options if the patient can no longer be managed at home because of deterioration or carer stress.

For patients presenting with challenging behaviours, admission to hospital may be necessary, and use of the Mental Health or Mental Capacity Acts may be required. Some patients may require guardianship to ensure that it either allows access to carers or to ensure they live in the most suitable accommodation. Other patients may need authorizations under the Deprivation of Liberty Safeguards (DOLS) in order to be managed in their best interests (part of the Mental Capacity Act legislation). If patients do not have the capacity to make decisions about their care, it should be provided in their best interests, which are identified through consultation with concerned parties, including carers.

Carers have high rates of stress and psychiatric morbidity so good support is required. Not only do carers have to cope with supporting the management of the tasks of daily living, but they also have to cope with the loss of a loved one in the way that they knew them. Carers need support through education about the dementia along with advice on communication and management of difficult behaviours. Interactions with voluntary sector groups should be encouraged such as the Alzheimer's Society (www.alzheimers.org.uk), which can offer advice and carer support. As the population is now more elderly than before, the number of carers helping to look after people with Alzheimer's is increasing. This means that there needs to be a range of resources to help support carers varying from individual or group psychoeducation, peer support groups, telephone and Internet support. There is obviously a need to involve family members in the care decisions, but this should not replace the need to involve the person themselves.

Delirium

Delirium (often described as an acute confusional state) has been recognized for centuries and its name comes from the Latin 'to be out of your furrow' (*de* away from and *lira* furrow in a field). Although it is easy to recognize in its florid form, it is often still missed and underdiagnosed (especially in elderly patients), with room to improve management. It is a syndrome representing the neuropsychiatric symptoms of an acute physical disorder and presents as an acute confusional state with symptoms of a physical illness. Typically, the patients present as agitated or overtly confused, but often missed are those who are withdrawn and do not

VIDEO 12.3

Delirium: essential facts – https://vimeo.com/56426649

display overt behavioural disturbances. A high index of suspicion is required in patients at risk, as early treatment has a positive effect on outcomes.

It is the most common acute disorder in general hospitals, and is a common cause of mortality (up to 20%–25%) and morbidity. It is common in older people but can occur at any age, including in children. Its presence indicates more severe illness in younger patients, and it is well recognized on intensive care units.

Delirium is defined in ICD-10 as: 'An etiologically non-specific organic cerebral syndrome characterized by concurrent disturbances of consciousness and attention, perception, thinking, memory, psychomotor behaviour, emotion, and the sleep-wake schedule.' Core features include a rapid onset, global disturbance of cognitive functions and evidence of the physical cause. The incidence of and prevalence of delirium is variable depending upon the cohort studied and the criteria used. However, in community settings, delirium rates range from less than 0.5% in working-age adults, increasing to over 14% in those over 85 years old. In hospital the rates are much higher, with prevalence rates between 10% and 40% on admission and 25%–60% developing it during their stay. Overall, the average is about 20%–25% in older adults, but unfortunately between 32% and 66% of all cases go unrecognized. Rates are higher in certain groups such as stroke (> 30%), hip fracture (40%–60%), vascular surgery (20%–40%) and terminal illness (> 50%). Delirium increases the risk of developing dementia about threefold and increases the length of stay in hospital. It increases morbidity and mortality, both short and long term. Mortality rates vary between 10% and 20%, double that of similar patients. The mortality effects continue even after discharge, with 2-year survival after an episode of about 40% (see Figure 12.1). Hence, the early diagnosis and treatment of delirium is a priority.

Risk factors

The underlying pathophysiological cause for the development of delirium is not fully understood and is

Table 12.4 Delirium risk factors

Premorbid risk factors	Causative aetiological factors
Age	Infection (particularly urinary tract)
Dementia	Severe acute physical illness
Alcohol dependency	Hyponatraemia
Visual impairment	Hypoxia
Depression	Dehydration
Severe physical illness	Constipation or retention
Physical frailty	Pain
Stroke	Sepsis
Brain damage	Surgery (particularly cardiac and orthopaedic)
Metabolic abnormalities (particularly renal impairment)	Intensive care admission
	Multiple changes of environment
Polypharmacy	Physical restraint
Immobility	Drugs (particularly those with cholinergic side effects)
	Opioid analgesia
	Withdrawal from drugs (particularly alcohol and benzodiazepines)
	Cardiological, e.g. infarction, failure
	Respiratory, e.g. pulmonary embolus
	Endocrine dysfunction
	Acute brain injury, including stroke and trauma
	Neurological disorders

multifactorial, but several premorbid factors increase the risk of patients developing delirium, especially age, dementia (both presence and severity), alcohol dependence and physical health problems. These are outlined in Table 12.4.

The aetiology of delirium is usually due to a combination of premorbid risk factors combined with precipitating causes. Drugs of any class affecting the nervous system, including psychotropics, are particular aetiological agents that should be considered, especially if there is polypharmacy (Table 12.4). When patients have mild cognitive impairment/reduced cognitive reserve, their vulnerability to delirium may be increased especially if they suffer from any acute insults such as surgery, dehydration and so on.

Clinical presentation

Presentation is variable, but is clearly illustrated in the definition, with disturbances across a wide range of cognitive and behavioural domains, combined with impaired consciousness and confusion, the latter succinctly defined as 'an inability to think with one's customary clarity and coherence' by Lishman. Onset is usually rapid, over hours to days, but may occasionally be a few months. Most of the features fluctuate and vary in their degree or intensity. Symptoms often tend to deteriorate towards evening and the sleep–wake cycle is reversed, with nocturnal insomnia and drowsiness during the day.

VIDEO 12.4

"She didn't know where she was…": delirium – https://vimeo.com/28822821

There is disorientation in time and place, with difficulty following time. Patients have impaired anterograde memory, indicated by a reduced digit span and deficits in short-term recall (they will not recall a test address). Retrograde memory will be affected with poor long-term recall of biographical details or learned information. They may confabulate to fill the memory gaps.

Psychomotor changes are evident, with withdrawal or little activity in mild states or in hypo-alert states. Detection of delirium in these states is often missed. In more severe states, hyperactivity and agitation occurs with illusions and hallucinations (commonly visual in nature) and other perceptual disturbances. Lilliputian hallucinations are characteristic. There may also be purposeless motor activity and emotional lability. In older patients hyperactive delirium is not necessarily more severe than the hypoactive form.

Diagnosis

The crux of the management of delirium is the detection and treatment of the underlying cause. In elderly patients there may also be contributory causes, all of which require attention. In high risk groups, such as elderly surgical patients, being on alert for such states may help earlier diagnosis and reduced morbidity as well as putting preventive measures in place when possible.

To establish the correct diagnosis, a thorough history is needed including reasons for admissions and any changes since admission, medication and alcohol history, previous episodes of delirium and any relevant factors. This may be difficult or not possible to obtain from the patient, so other informants must be used, e.g. relatives, or other sources such as the general practitioner (GP), old notes or care staff, both before and since admission. Much delirium has its onset during admission, so admitting ward staff are also key informants. The importance of communication within and between professional groups is important to gain a thorough history.

A thorough examination should be performed if the patient's mental state allows. In agitated patients this may be difficult, but even observation may give important clues to the underlying disorder or exacerbating factor, e.g. hypoxia may be evident as cyanosis. It is important to check for problems such as dehydration, constipation or retention that can be remedied simply. Baseline physical observations and cognitive assessment should be recorded. This allows monitoring of progression of the course of the delirium.

As well as full histories and mental state, a full battery of physical investigations should be undertaken, including blood (full blood count, urea and electrolytes, inflammatory markers, e.g. *c*-reactive protein), specimens such as urine and sputum for cultures, radiological investigations such as chest x-ray or CT head scan or drug screens in younger people. Readers are directed to textbooks on clinical medicine for further details.

Box 12.2 Confusion Assessment Method (Inouye et al., 1990)

For a diagnosis of delirium, both A and B, and either C or D are needed, described as follows:

A – Acute onset and fluctuating course: onset is hours to days, lucid periods often in morning

B – Inattention: easily distracted, attention wanders in conversation

C – Disorganized thinking: cannot maintain a coherent stream of thought

D – Altered level of consciousness: drowsy/overactive fluctuation, nightmares/hallucinations

Subsyndromal and fluctuating pictures are very common, so if there is any doubt, one should proceed on the presumption that a degree of delirium is probably present. Delirium, like fever, is an important non-specific sign that there is something physically wrong with the patient, and it should be investigated urgently. Although, for most cases, the clinical diagnosis will be clear, the EEG can be useful in determining the diagnosis and its continuing presence but is not usually used. It is also possible to use the Confusion Assessment Method (Box 12.2), which has been validated as a screening test for delirium, but is not a substitute for a history and examination.

The differential diagnosis includes the following causes, but it is important to stress that in many cases many of these conditions coexist:

- Dementia
- Substance misuse
- Withdrawal states, e.g. alcohol (delirium tremens), benzodiazepines
- Schizophrenia or other psychotic disorders
- Depression and other affective disorders
- Conversion disorders
- Temporal lobe epilepsy
- Non-convulsive epilepsy

Management of the delirious patient

Prevention is better than cure! It is important to look for and avoid potential risk factors as much as possible. Awareness of the risks and minimizing any potential areas of risk are important, e.g. in non-acute settings this may include volume replacement before surgery, ensuring electrolyte imbalances are reversed and correcting anaemia. Patients should be adequately hydrated and their nutrition optimized, and they should be given supplementary vitamins, especially thiamine, particularly those with a history of alcohol misuse. It is advisable in elective settings to advise patients and carers about the possibility of delirium occurring, given its relative frequency. Consideration needs to be given to any medications the patient is taking. Patients should be mobilized and actively rehabilitated as soon as possible to prevent functional decline.

If delirium does occur, medical treatment and management of the underlying medical or surgical cause is a priority, and readers are referred to relevant medical and surgical texts for further details. Any drugs or medication that may be a cause should be withdrawn and treatment reviewed.

Given the nature of delirium, treatment will require access to a full range of diagnostic, monitoring and treatment facilities, and a full range of physical treatments may be needed, including intravenous fluids and antibiotics, oxygen and electronic monitoring. Attention should be paid to areas such as hydration and nutrition and ensuring homeostasis, including managing any hypoxia, fluid and electrolyte imbalance or pain. Secondary complications such as dehydration, constipation or pressure sores should be prevented.

Symptoms tend to resolve as the underlying disorder is treated, but symptoms can persist and can take up to 6 months to fully resolve. If symptoms continue, then the possibility of an underlying dementia needs to be considered.

Non-biological management of delirium

As well as the physical aspects of care, it is very important to appropriately treat accompanying symptoms and behaviours. It is often these that cause the problems for staff and which may interfere with the treatments for the underlying disorder. Attention should be paid to communication and environmental aspects, although it may not be possible to have the ideal environment for the patient.

Delirium can be frightening for the patient. It is therefore important to communicate clearly and regularly with patients, orientating them and explaining what is happening. All care activities and, particularly,

EXERCISE 12.2

The delirious experience:
Imagine that you are confused and frightened, thinking the staff is trying to harm you, and someone comes towards you with a needle and syringe.

How might you feel?

How might you behave?

How would you approach such a patient as a doctor?

procedures should be clearly explained. Some patients are able to recall their experiences of delirium, and this may be quite disturbing, often like that of recalling dreams or nightmares.

Patients should be approached calmly and you should avoid startling or frightening them. You should try to minimize the possibility of any misinterpretations. For agitated, distressed or aggressive delirious patients verbal and non-verbal de-escalation techniques should be used.

To aid this, you should optimize the patient's sensory input, by ensuring that any spectacles and hearing aids belonging to the patient work properly and are used correctly. They also need to be accessible and within reach of the patient. It is not unheard of for hearing aids not to have batteries, or for spectacles to be in bedside lockers! If possible, patients should be managed in side rooms, with consistent input from the same members of staff. Noise should be kept to a minimum, and lighting levels need to be considered – well-lit, but not too bright or too dull as this may lead to misperceptions or illusions. Any clocks should be visible to the patient and kept to the correct time. In order to promote orientation, moves on or between wards and departments should be kept to a necessary minimum, which is often difficult in modern care settings.

Delirium can be frightening for the carers too, so they also need clear explanations of what may be happening. However, they can help manage the patient and should be encouraged to stay as much as possible with the patient, helping to reassure and orientate the patient. They should be asked to bring in familiar objects, to promote reorientation and to help reassure patients.

Pharmacological management

The main causes of referral of delirious patients to psychiatrists are agitated or aggressive behaviour. These should be managed primarily with non-pharmacological means as outlined above, but if pharmacological means are needed, care should be taken in deciding both the drug and the dose. Withdrawal of any drug that may be causing the delirium should be considered before adding psychotropic therapies. Drugs will not stop patients from wandering and patients should not be restrained (unless as a temporary measure to prevent injury).

The National Institute for Health and Care Excellence (NICE) suggests use of either haloperidol or olanzapine, preferably for no longer than 1 week. Haloperidol is recommended due to its relative lack of anticholinergic side effects, but it can cause drowsiness or parkinsonian side effects, which may lead to falls. Haloperidol may be given intramuscularly if necessary but care needs to be taken with cumulative doses. Though olanzapine may increase risks of cerebrovascular events, this risk is felt to be low with short-term use. Drugs with anticholinergic effects should be avoided if possible. Alternatively, lorazepam can be used, especially if short-term sedation is needed (though traditionally advice has been to avoid benzodiazepines because of a potential to disinhibit, but this is rare). Regular small doses should be prescribed, particularly in older people, to help prevent over sedation (which may worsen the confusion). 'As required' treatment should only be used to treat acute flare-ups not responding to other methods; when used alone patients may develop alternating periods of sedation and agitation. Care should be taken not to exceed *British National Formulary* limits as this may lead to potentially serious unwanted side effects, particularly over sedation.

Disturbed patients may respond to simple measures outlined above, but if these do not improve the patient, help should be sought from liaison psychiatry teams. The patient may require additional pharmacological treatment or even transfer to a psychiatric unit. Consideration also needs to be given to the impact of a disturbed patient's behaviour on the other patients in hospital settings. Capacity issues need to be addressed, for delirium is likely to render a patient incapacitous, particularly regarding treatment and restraint. Any treatment should be in the patient's best interests.

Delirium tremens

This acute disorder is considered separately to delirium by ICD-10 (under substance misuse disorders) though it is essentially a delirium secondary to the withdrawal

Table 12.5 Differences between delirium and dementia

Characteristic	Delirium	Dementia
Onset	Acute (hours to days)	Chronic (months to years)
Progress	Fluctuant	Gradual deterioration
Variability	Changes over hours to minutes	Variable over days (good and bad)
Duration	Days to weeks	Years
Response to treatment	Usually good	Deteriorates despite treatment
Consciousness	Clouded or impaired	Clear
Psychotic features	Early	Late
Hallucinations	Common	Uncommon
Delusions	Common	Uncommon
Orientation	Marked early disorientation	Gradual loss, time and place early on
Memory	Impaired but variable	Early short-term memory loss, long term lost later
Concentration	Fluctuant, marked inattention	Gradual deterioration
Psychomotor activity	Variable (increased, reduced or fluctuant)	Normal until later stages
Sleep–wake cycle	Characteristically disturbed	Variable
Language	Incoherent, inappropriate, psychomotor slowing	Good, some word-finding problems, expressive dysphasia

from alcohol in dependent patients. This is covered in Chapter 19, along with the consequences such as Wernicke's encephalopathy and Korsakoff's syndrome.

What do you understand as the differences between dementia and delirium?

Earlier in the chapter, we asked the question 'What do you understand as the differences between dementia and delirium?' Even without investigations, we can determine whether someone has one or the other from key features in the history. These are illustrated in Table 12.5.

Summary

In this chapter we have seen that organic conditions are common, more so in older people. Physical disorders may present as psychiatric disorders, rather than physical ones, and should be excluded. Organic disorders have significant effects on mortality, morbidity and quality of life. The most common disorders are delirium and dementia. In delirium, early diagnosis and treatment of the underlying cause improve outcomes. In dementia, early diagnosis is key; much can be done now for AD and other dementias. In managing dementia, a holistic approach should be taken, with emphasis on psychosocial carer support and quality of life and care issues.

Further reading

(Note: Websites of national Alzheimer's associations, for example, https://www.alzheimers.org.uk/, can be very useful resources.)

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CASE STUDY 12.1

Mrs V was an 84-year-old woman who lived with her husband in rented accommodation. She was noted by her husband to have become increasingly forgetful over the past 12 months, but he became particularly concerned when she was brought home by the police after going shopping. She has a history of hypertension, diabetes and hypercholesterolaemia. On her way to the GP surgery, she trips, falls and fractures her left neck of femur.

How would you assess this lady? What are you going to do to prevent her developing delirium in hospital?

As the pre-fall history is suggestive of dementia, she needs to have a full history and particularly an informant history from her husband. Although the gradual decline is suggestive of Alzheimer's disease, she has particular risk factors for vascular dementia. A CT scan would be helpful.

She requires surgery and preoperatively she should be hydrated, her blood pressure should be optimized and she should be given adequate nutrition, vitamins and analgesia prior to being nil by mouth. Postoperatively she is at high risk for delirium, and she should be monitored for this. Hydration, nutrition and analgesia should be maintained, and early mobilization should be encouraged. Electrolyte imbalance, anaemia and any infections should be assertively managed.

CHAPTER 13

Psychiatric aspects of intellectual disability

SHAHID H ZAMAN WITH HOWARD RING

KEY CHAPTER FEATURES

- Importance of psychiatry and low IQ
- Definition and manifestation of intellectual disability
- Intellectual disability and social context
- Outline of risk factors for the development of an intellectual disability
- Clinical descriptions and exemplars of intellectual disability
- Assessing and managing the health needs
- Delivering healthcare

Introduction

Approximately 2% of the population of the United Kingdom have an intellectual disability (ID) and the individuals to whom this term refers manifest an enormous range of different abilities, aetiologies, diagnoses and co-morbidities. However, they also share an array of challenges across intellectual, communicative, motor and social domains and have frequently been marginalized members of society, often poorly served by services that the rest of the community depend upon. Not surprisingly, people with ID need those working with them to have a particular set of skills and knowledge, and the aim of this chapter is to introduce these.

Why is this relevant to you?

Individuals with ID may present to any health service and often have multiple health needs. The evidence is that the care they receive can be less than optimal. It is important for all doctors to be aware of specific issues

related to ID so that these can be managed appropriately. There may be limitations to how much people with ID can contribute to their own management plans. However, that does not mean they should not be involved in any aspects of their own care.

Terminology and definitions

Many terms have been used to describe those with lifelong impaired development characterized by clearly limited intellectual abilities to understand, participate in or adequately safeguard their own basic needs in everyday life. Such terms have included imbecility, idiocy and mental retardation. The current choice of terminology aims to minimize the stigma associated with terms that have become prejudicial, but adequately reflecting the pervasive yet varied nature of such states. Although the description 'learning disability' is still widely used in the United Kingdom, the term 'intellectual disability' is becoming increasingly applied and reduces the risk of confusion, described in the following, with the very different diagnosis of a 'learning *difficulty*'. In this text, ID will be used.

EXERCISE 13.1

Consider what readily transferable clinical skills are critical in the practice of psychiatry with those with an intellectual disability (ID).

Could you explain the importance of taking anti-epileptic medication correctly to somebody whose verbal abilities may be at the level of a 3-year-old and to their carer whose main preoccupation up to now has been trying to support this patient to eat and drink adequately?

How would you approach the issue of deciding what to do if you discovered a breast lump in a woman with a severe ID?

What is ID?

Intelligence quotient (IQ) is a neuropsychological measure of general intellectual functioning including the ability to solve problems, think in abstract and acquire new learning and skills. ID is *partly* defined by a low IQ (see Box 13.1). To have an ID is to have limited abilities across a wide range of everyday functions including cognitive, language and motor processes and social activities. ID is considered as a developmental disorder in which the individual is either never able to acquire the educational and functional skills expected for their age or early in life suffers an insult that arrests their development such that they cannot go on to develop the expected level of functioning. Hence, those with an ID include individuals with a developmental disorder that has been present since intrauterine development, or birth, and also those who were born with apparently normal brain functioning and intellect but who sustained a major insult to brain function in the early years of life that subsequently led to pervasive limitations in cognitive development. For most cases of ID the cause of the ID are unknown. In others, there has been an abnormality in brain development, which may be genetically determined (for instance, Down syndrome or fragile X syndrome) or arise from environmentally related pathological processes during pregnancy or birth (for instance, in association with intrauterine infection or significant birth trauma). Additionally, some with ID may have major pathology sustained during infancy, arising from brain injury (caused by head trauma, bacterial meningitis) or from a brain tumour and its management. Recent genetic investigations

Box 13.1 Some definitions

- Intellectual disability = low IQ plus deficits in adaptive functioning
- WHO definition: 'A condition of arrested or incomplete development of the mind, characterized by impairments in cognition, language, motor and social abilities'. Therefore, there has to be a pre-natal or early life neurodevelopmental failure or delay.
- The severity of the ID (mild, moderate, severe or profound) is determined by the IQ measure.
- Low IQ = an IQ below two or more standard deviations from the mean of the general population (i.e. ≤ 70).
- Adaptive functioning: Everyday skills required for independent living, e.g. ability to shop, budget, cook, clean, undertake personal care, plan activities, use of community resources and routines.

are revealing many types of genomic abnormalities including point mutations, deletions and duplications that will help inform future clinicians in the management of ID. It is well known that intelligence is normally distributed in the population with the implication that there will be some individuals towards the lower end of the distribution who have a low IQ in the absence of any identifiable aetiological pathology.

Range severity of ID

Although it is very important not to focus exclusively on IQ, this does provide a summary measure that, with various caveats, can indicate an individual's likely level of functioning (see Boxes 13.2 and 13.3). The IQ measure is calculated by dividing a person's mental age by their chronological age according to the formula – (mental age/chronological age) x100. In this context, mental age is considered as performance on a range of standardized problem-solving tests that investigate a range of verbal and motor performance tasks. It is difficult, though, to develop tests that do not incorporate some educational or cultural bias. However, regardless of which tests are used, a person with ID will always function at a mental age considerably below their chronological age. Across the whole population, intelligence as defined by IQ is normally distributed, with a mean of 100. In practice, ID is considered to be present in those with an IQ of 70 and below. The ranges of disability are shown in Table 13.1 and the size of each group diminishes as IQ falls. Hence the diagnosis of ID includes individuals with widely varying abilities, extending from those who can live more or less independently in the community and who may never formally be identified as having an ID to others with multiple complex disabilities severe enough to mean that they cannot communicate, self-care or indeed apparently generate intentional activity of any kind.

Those with mild ID may be delayed in the rate at which they acquire abilities but will ultimately

Boxes 13.2 Level of abilities of adults with ID

Severity of ID: Mild and Moderate

Mild (IQ 50–69; mental age 9–12 years)	**Moderate (IQ 35–49; mental age 6–9 years)**
Basic reading/writing	Limited comprehension and use of language
Speech for everyday purpose	Need lifelong support in planning and organization of their activities
Independence in self-care, in practical and domestic skills	Personal care depends on degree of training or practice, cognitive ability and co-morbid physical disability e.g. cerebral palsy
Potentially capable of work that is practical than academic	Complete independent living rare
	Can engage in simple social activities
	Some practical work with supervision
	Usually mobile

Severity of ID: Severe and Profound

Severe (IQ 20–34; mental age 3–6 years)	**Profound (IQ < 20; mental age below 3 years)**
Require lifelong assistance and support in personal care tasks and communication	Very limited in ability to understand or comply with requests or instructions
Need support in accessing community facilities/ services	Most immobile or poor mobility
Word and gestures for basic needs	Little or no ability to care for their own basic needs
Need support for most activities	Need constant help, support and supervision
Motor impairment common	

Table 13.1 Ranges of disability

IQ range	Description	Proportion of ID population
50–69	Mild ID	75%
35–49	Moderate ID	20%
20–34	Severe ID	4%
< 20	Profound ID	1%

develop sufficient language function to be able to use speech for everyday purposes. Many in the upper part of this band will lead largely independent lives, although budgeting, maintaining work and coping with complex social demands are likely to require support. With appropriate education some reading and writing skills may be developed. With moderate levels of ID, individuals will need at least some supervision throughout life, and language skills are likely to be clearly limited. At lower levels of IQ, full support will be required for most or all aspects of daily living.

ID and learning difficulties

It is important to note that ID (or learning *disability*) is *not* the same as learning *difficulty*. There are a wide range of specific learning difficulties (for instance, dyscalculia, dyslexia) and they can occur in individuals with IQs in the normal range.

Epidemiology of ID

It is not easy to obtain accurate estimates of the prevalence of ID. A variety of agencies are involved in supporting those covered by this descriptive term. Some, even with moderate or severe ID, are looked after largely by their families, and a difficult to determine proportion of those with mild ID are unknown to services and live independently or supported by family, friends and their local social circles. However, various estimates of prevalence have been obtained, with results varying depending on how data were obtained and whether the studies were social care, healthcare or population based. In Western Europe, prevalence within the overall population of around 0.7% has been estimated using national registers. Higher figures of around 1.6% have been obtained in population studies, while healthcare-based research has reported approximately 0.4% of patients to have an ID.

In 2001, the UK Government White Paper *Valuing People* stated that in England there were approximately 210,000 people with a severe and profound ID, of whom 65,000 were children and young people, 120,000 of working age and 25,000 older people. Additionally it was estimated that a further 1.2 million people had a mild or moderate learning disability. Research and examination of health trends also suggest that the number of those within society who have an ID will increase significantly in future years. The size of this increase has been estimated at 14% by 2021. As the care for those with ID improves, much of this increase will be in the number of adults aged over 60 years and this will have implications for planning of future health and social care services. Further reasons for a likely increase in the prevalence of ID include the improved survival rates of very premature neonates and infants sustaining brain damage through trauma or disease and better management of significant co-morbidities in those with ID, most commonly epilepsy and infections, leading to a general increase in life expectancy. At the same time, better education and increased genetic screening may somewhat reduce the magnitude of this expected increase in prevalence.

Historical context

To understand the issues raised and the approaches followed in providing care for those with ID it is helpful to be aware of the historical and cultural contexts within which this part of the population lives. From the later seventeenth century the move into the 'Age of Enlightenment', with critical aspects of humanity considered in terms of rationality, education and self-aware thought, emphasized the differentness of those with an ID and served to separate and diminish them with respect to the general population. This differentness was incorporated in a variety of social structures and laws. For instance, in the United Kingdom, the Poor Laws of 1837 referred to idiots, imbeciles and those of weak intellect. In the earlier Victorian era, those with ID were not viewed as any threat to society. However, from the mid-nineteenth century concern emerged about a 'lack of moral sense' within that part of the population. Interestingly, in some circles the wider

interpretation of Darwin's *Origin of Species* in the later nineteenth century also increased concern about the 'debilitating' effect on society of those with intellectual disabilities – a concern that was also expressed in the eugenics movements of the earlier parts of the twentieth century. However, after the first half of the twentieth century demonstrated so clearly the inhumane and terrible consequences of philosophies espousing eugenics and an overly functional conception of society, more humane perspectives became widespread, although for those with intellectual disabilities this was often still translated into long-term institutionalization.

By the late twentieth century social and philosophical considerations suggested that society should empathize with those having an ID, and that this would have a role in supporting the cultivation of humanity. In other words, a view developed that by looking after those who could not care for themselves the general population were improving their own humane attitudes. Although such an approach may have benefited people with an ID to some extent, it did not acknowledge that people with ID could have rights of their own. Instead, it left them dependent on the wider population continuing to hold to the belief that looking after those with ID was a civilized thing to do.

Most recently, we have moved towards a human rights agenda. This has been a key step in developing social and health agendas to improve the lives of people with an ID in the United Kingdom and led to the development and publication of the Government White Paper *Valuing People*. This was based on the fundamental principle that people with learning disabilities have the same human rights as everyone else:

> *All people with a learning disability are people first with the right to lead their lives like any others, with the same opportunities and responsibilities, and to be treated with the same dignity and respect. They and their families and carers are entitled to the same aspirations and life chances as other citizens.*

Risk factors

A very wide range of causes as well as no apparent cause at all can result in somebody growing up with an ID. Potential categories of causes with relevant examples of specific conditions are listed in Table 13.2.

Table 13.2 Some causes of ID

Cause	Example
Genetic causes	
Single gene disorder:	
X linked single gene	Fragile X syndrome
Autosomal dominant	Tuberous sclerosis
Chromosomal disorder	Down syndrome
Imprinted gene disorder	Prader–Willi syndrome
Unknown but strongly hereditable	Autism spectrum conditions
Intrauterine causes	
Toxins	Fetal alcohol syndrome
Infections	Rubella
Perinatal causes	
Hypoxia	Cerebral palsy
Brain damage in infancy	Meningitis, brain tumour
Environmental causes in infancy	Extreme deprivation

Presentation and diagnosis

The presentation of an ID will, as indicated earlier, depend on the nature of the cause and severity of the condition. Those diagnoses associated with major structural anomalies may be diagnosed during intrauterine development or at birth. Others may be diagnosed by early abnormalities in development, for instance, difficulties with feeding, abnormal muscle tone or failure to achieve developmental milestones within expected age range. Although in those with developmental ID of moderate or greater severity, parents, health visitors or the family doctor will generally identify the likelihood of there being a problem in development during the neonatal period or early infancy, individuals with mild ID may never be identified or receive the extra help that they need. Problems may only come to light in later childhood or adulthood in the context of problems with learning, working or developing an independent life.

As well as often being associated with physical anomalies and delayed motor milestones, a large proportion of syndromes of ID are associated with abnormalities of various aspects of behaviour, and in some cases the precise pattern of behaviours may be relatively indicative of a particular genetic condition. A potentially useful concept in conceptualizing

the presentation, diagnosis and management needs of genetically determined ID syndromes is the 'behavioural phenotype'. A behavioural phenotype is described as a behaviour consistently associated with and specific to a chromosomal or genetic aetiology where that phenotype is the result of the underlying genetic lesion. Examples of conditions in which a behavioural phenotype can be recognized include fragile X syndrome and Prader–Willi syndrome (PWS). Although this concept is valuable in making clear the existence of a relationship between an underlying genetic anomaly and observed behavioural symptoms, its limitations include the finding that the same behaviour is rarely found in every individual with the same syndrome. Also, we have a very incomplete understanding of how specific genetic consequences are linked to particular behaviours or behavioural disturbances.

There are many different syndromes of ID, and each of these will not only have a constellation of signs, symptoms and co-morbidities that distinguishes it from other ID syndromes but also variations among individuals receiving the same diagnosis. This variability among individuals within a specific diagnosis may arise for several reasons, including IQ level, presentation of co-morbidity, unsuspected genetic variations and the modifying effects of early and later environment. The last point is worth emphasizing. It is well known that, particularly in childhood, adverse social pressures (for instance, emotional or physical deprivation or abuse) can have a major impact on the pattern of later development, including mental and physical well-being and intellectual functioning. This effect may be even more profound in people whose ultimate level of intellectual or emotional functioning is already impaired.

Clinical features of the more common ID syndromes

Key features of several well-recognized syndromes of ID are described as follows.

Down syndrome

Down syndrome is the most common specific cause of ID, occurring in one in 700–1000 live births. It develops as a result of trisomy of chromosome 21 in 95% of cases. This is because of primary non-disjunction in maternal meiosis, the risk of which increases with maternal age (potentially occurring in one in 40 births to mothers aged over 45 years compared with one in 1400 births to mothers aged less than 25 years). Approximately 5% of cases are due to balanced and unbalanced translocations and mosaicism. Balanced translocations are associated with a higher risk or recurrence. Mosaicism may be associated with milder forms of the condition that are not always diagnosed.

Fifteen per cent of those with Down syndrome have borderline intelligence or mild ID, with the rest having moderate or severe impairments in intellect. As well as the intellectual impairment, Down syndrome is associated with several well-described anomalies of physical development, including congenital heart defects in 40%, hypothyroidism and a characteristic facial appearance.

Psychiatric co-morbidities are relatively common in those with Down syndrome, with attention deficit hyperactivity disorder (ADHD) and conduct disorder occurring at increased rates in children. Twenty-five per cent of adults with Down syndrome will have a psychiatric diagnosis, most commonly of depression. Down syndrome is the most common cause of early-onset dementia. Almost all the brains of those with Down syndrome over the age of 40 that have been examined at post-mortem have been found to have neuropathological changes of Alzheimer's disease (AD). Forty per cent of those over the age of 50 years have clinical manifestations of AD. The amyloid precursor protein (*APP*) gene is located on chromosome 21, and as Down syndrome has trisomy of this chromosome, there is overproduction of β-amyloid and this is believed to be the mechanism underpinning the high rate of AD. People with Down syndrome tend to have lower blood pressure than the general population, low levels of atheroma, and so vascular dementias are uncommon.

Autistic spectrum disorder

Autistic spectrum disorder (ASD) is a pervasive developmental disorder, starting in early childhood and persisting throughout life. It is being increasingly recognized, and current prevalence estimates across the whole population suggest a combined frequency for all ASDs (including atypical autism and Asperger's syndrome) of around 1%. ASD is more common in those with an ID than in those with normal IQ. The core features of autism are absent, delayed or abnormal use of verbal

and non-verbal language, difficulties in developing reciprocal social relationships, a narrow and restricted range of interests and absence of symbolic play. (The clinical features of autism are further discussed in Chapter 14 on disorders of childhood.) However, in those with severe ID the impairment of language and communication skills often associated with such severe intellectual deficits may make it more difficult to make a diagnosis. Language and social communication problems can lead to social isolation for those with an LD as for those within the normal IQ range. In addition, in people with ID and autism there are increased rates of challenging behaviour, a collection of symptoms that may include aggressive actions directed at self or others, more fully described in the section 'Challenging behaviours.' Clinical experience indicates that it is often difficulties in understanding and meeting the needs of people with autism and ID that lead to episodes of challenging behaviour. Also, understanding the sensory integration problems of people with ASD can help to improve behaviours and symptoms.

ASDs are also associated with increased rates of psychiatric conditions and epilepsy. Studies suggest that in those with high functioning autism and Asperger's syndrome, there are slightly higher rates of schizophrenia than that existing in the general population. In those with more severe autism and low IQ, epilepsy rates are particularly raised.

Prader–Willi syndrome

PWS is a genetically determined neurodevelopmental disorder that results from the absence of expression of one or more as yet unidentified 'maternally imprinted/paternally expressed' genes located on chromosome 15. (Normally the PWS critical region of chromosome 15 is expressed from the paternally inherited allele, whereas the maternally inherited allele is silent. In PWS this region is not expressed at all, most commonly because there is a deletion in the PWS critical region in the paternally inherited allele.) PWS has a population prevalence of approximately 1:50,000. Infants with PWS are extremely hypotonic and fail to thrive, requiring assisted feeding. As early as 2 years of age, however, the presentation changes to the phenotype that characterizes the remainder of childhood and adulthood. This comprises mild developmental delay, a tendency to skin pick and, most significantly, obesity, which is related to the difficulty in controlling eating behaviour. Those with PWS find it very difficult to stop eating until they have consumed very high calorie loads. Later, short stature and a failure of normal secondary sexual development become apparent, as do behavioural and psychiatric problems. Most people with PWS have mild learning disabilities with an average full-scale IQ score of 60, with good spoken language and adequate functional abilities. PWS is associated with diabetes mellitus, and sleep and respiratory disorders and has a yearly mortality rate of 3%, largely associated with obesity-related morbidity.

Fragile X syndrome

Fragile X syndrome is the most common cause of single gene ID (see Box 13.4), occurring in around 1 in 3500 males of and 1 in 4000–6000 females of European descent. Although an X-linked condition, it is also responsible for mild-to-moderate ID in 1 in 4000–6000 females in the United Kingdom. In affected boys, physical signs include mild facial dysmorphia with long face, large ears and macroorchidism by around the onset of puberty. However, these signs are not sufficiently

Box 13.4 Fragile X syndrome features

- Commonest inherited cause of ID
- X-linked dominant, variable expressivity
- Males – 1 in 3500; females – 1 in 4000–6000
- Mutation in *FMR1* (fragile X mental retardation) gene at 5′-untranslated end
- CGG repeats, need > 200 but lesser numbers can cause symptoms ('pre-mutations') often within a normal IQ range
- IQ: normal to mild to more severe
- Developmental delay
- Long faces, large ears and forehead, hypotonia and hyperextensible joints, flat feet, macroorchidism
- Autistic features: Gaze avoidance, repetitive behaviours
- Stereotypical movements – hand ringing, biting
- Aggression
- Social anxiety
- ADHD common but less in adults
- Seizures

constant or specific to be pathognomonic. Intellectual and behavioural signs include delayed language acquisition, impaired numerical and visuospatial skills and behavioural problems, including autism-like behaviours of social anxiety and aversion to eye contact in around 30% (largely in those with lower IQ). Hyperactivity and self-injury when excited or frustrated (for instance, hand-biting, scratching) are also recognized.

Co-morbidities in those with ID

One of the most important aspects of supporting the health of individuals with ID is to recognize and treat the physical and psychiatric co-morbidities that exist at higher rates in this group than in the general population. Historically these have been underdiagnosed and inadequately managed for various reasons. These include difficulties experienced by those with limited communication skills in making their symptoms known, and modification of symptoms and their expression by the presence of an ID and often the syndrome causing it. An additional factor may be that carers are not alert to the possibility of co-morbidities or inappropriately accept those they do notice as 'just part' of the individual's presentation. Unfortunately, there have also been and there continue to be reports of prejudices and uninformed attitudes in healthcare staff that have led to management decisions that would not have been made if the individual had not had an ID.

Physical co-morbidities

The more common physical health problems in those with ID include poor oral hygiene with dental disease, gastrointestinal problems, including gastro-oesophageal reflux and constipation, and epilepsy. It should be readily appreciated that in those with limited communication skills the pain and discomfort associated with toothache and bowel symptoms may lead to a variety of manifestations of distress, including behavioural symptoms ranging from self-injury (for instance, hitting their own face repeatedly), to hitting out at those that try to get close, to apparent loss of previous skills. Surveys of those with ID have also revealed high rates of motor and sensory deficits. Whereas motor problems may be obvious in, for instance, an individual with cerebral palsy, possible partial sightedness and hearing disorders may not be diagnosed without specialist assessment and should be part of the management of children with ID, for if they remain undiagnosed they can significantly impair an individual from reaching their full potential.

Epilepsy is common in those with ID, its frequency increasing progressively with more severe intellectual impairment. Lifetime prevalence of epilepsy in those with mild to moderate ID (IQ > 50) is 15%, whereas in those with severe ID (IQ < 50) prevalence is 30%. A recent population-based study of adults with ID reported a prevalence of epilepsy of 26%. Hence more than 200,000 people in England have ID and epilepsy. As well as being relatively more common, epilepsy in those with ID has a worse prognosis than epilepsy in the general population. Individuals are more likely to have multiple seizure types, to have lower rates of seizure freedom on anti-epileptic medication and to have higher rates of sudden death in epilepsy (SUDEP). This means that even compared with those with ID but no epilepsy, who in any case have an increased standardized mortality rate (compared to the general population) of 1.6, those with ID and epilepsy have an even higher rate of 5.0. The frequency of epilepsy varies among different ID syndromes. In those with tuberous sclerosis (an autosomal dominant disorder involving one of two genes (*TSC1* or *TSC2*) and leading to the development of multiple hamartomas in brain and other organs) severe seizures in infancy may be the presenting complaint.

Psychiatric co-morbidities

Overall, psychiatric symptoms and psychiatric diagnoses exist more frequently in those with an ID than in the rest of the population. This is the case for both children and adults. Relatively common conditions include affective disorders and autism. Affective disorders and schizophrenia are both considered to occur at least twice as often in those with ID as in the rest of the population, and ADHD also occurs more frequently in adults with ID than in adults with an IQ in the normal range. The obsessional need for sameness with intolerance of variation or unpredictability, as seen in autism, also occur in various syndromes of ID, often in association with severe impairment of IQ.

As indicated in the preceding discussion of behavioural phenotypes, particular syndromes of ID may be associated with increased rates of a specific neuropsychiatric state. Down syndrome is associated with high rates of AD and there is an increased prevalence of psychotic symptoms in people with PWS.

How do people with an ID manifest psychiatric symptoms?

Boxes 13.5 through 13.7 show common features of adults with ID, common issues in ID practice and aspects of clinical assessment in ID, respectively.

It is more difficult to make psychiatric diagnoses in people with severe ID who generally lack the language abilities to verbally describe their psychological experiences, than it is in people with mild or moderate impairments. Possible biological symptoms of depression such as sleep disturbance and change in appetite should be discussed with carers. Behavioural changes such as decreased social interaction, loss of interest in activities previously engaged in, apparent agitation or distress, tearfulness and a decrease in previous levels of functional abilities may all suggest significant lowering of mood. The reliable eliciting of these signs and symptoms involves patient, careful and clear questioning of family members and carers. To describe hallucinations or delusions requires significant language abilities, and thought disorder may be difficult to identify in an individual whose behaviour and communications may already be difficult to follow. In these circumstances, information gathered from a family member or paid carer who knows the individual well may be invaluable. In addition, for those at the most severe end of the ID spectrum it remains unclear whether behavioural disturbances that appear to indicate psychological distress reflect psychological states that are congruent with the depressive and psychotic states described in those with higher IQs. The diagnosis of psychotic states may be particularly problematic in this context, and this may relate to the observation that there are increased rates of depression and psychosis in those with moderate ID compared with those with severe or profound ID.

Self-injury as a symptom is well recognized in the general population. It is also a common problem among those with more severe ID. In this group, however, it probably has a different meaning from that ascribed to it when it is carried out by those without an ID or mild ID. In people with ID it often takes the form of biting, scratching or head-banging. It may at times be understood as a self-stimulatory behaviour and can occur for a significant proportion of the time and may be hard to stop. However, when it develops in an individual for whom it is not habitual, it is important to consider the possibility of physical health problems, environmental changes and mood disturbance.

Box 13.5 Common features of adults with ID seen in clinic

- Language/communication problems: often visual means of communication much easier
- May appear more able than actually are
- Poor ability to focus for long – distractible
- Slower processing of information
- Suggestible
- Repetitive or stereotypical behaviours
- 'Autistic traits' i.e. may have features of autism but do not fulfil criteria for diagnosis of autism spectrum disorder
- Carers tend to speak for them – can be frustrating and give a biased picture
- May have negative self-image, easily frustrated, low self-esteem, dependent, rights overlooked, open to abuse, concrete thinking, poor at conceptualising time, rigid problem solving, good in some aspects of cognition – e.g. visual memory better than verbal

Box 13.6 Common issues in ID clinical practice

- Under-recognized physical health problems
- Behavioural problems that are not always due to mental health problems
- Epilepsy
- Autism
- Mental capacity; safeguarding of the vulnerable; deprivation of liberty
- Ageing population
- Little research evidence to guide management
- Needs are best met with all professionals and agencies working together
- Professional carers often need training and guidance
- Psychotropic medication overprescribed
- Stigma in healthcare

Box 13.7 Aspects of clinical assessment in adults with ID

- Need lots of time
- Need information from all sources
- Home environment may be preferred to clinic setting
- May need to see more than once
- Need to have carers who know the person well
- Gather behavioural data – sleep, mood, aggression, engagement with others and activities, appetite, weight, seizures etc.
- Consent, mental capacity, confidentiality and sharing of information
- Impact of communication problems/ sensory impairment/sensory integration
- Level of disability
- Impact of unsuitable social environment (housing, activities) or support
- Be aware of diagnostic overshadowing – the tendency to misappropriate symptoms and behaviours to ID *per se* and not another condition or disorder
- Behavioural phenotypes of those with known syndromic diagnoses
- Safeguarding of the vulnerable
- Focus on changes of presentation to distinguish pre-morbid features from presenting ones and use a hypothesis-based approach to diagnosis and management

Challenging behaviour

In addition to these psychiatric diagnoses, there is in those with ID an additional diagnosis based on the occurrence of episodes of potentially damaging behaviour. This diagnosis is named 'challenging behaviour' and it is acknowledged that this is simply a term to describe particular classes of behaviour. The term does not convey any information relating to a possible basis to the presentation. In those with ID, behaviour can be described as challenging when it is of such an intensity, frequency or duration as to threaten the quality of life and/or the physical safety of the individual or others and is likely to lead to responses that are restrictive, aversive or result in exclusion. The prevalence of serious or aggressive challenging behaviour (CB) in those with ID has been estimated at around 10%. Those with ID are the largest single group in society with lifelong disabilities and are among the most complex and heterogeneous group, requiring inter-agency, multidisciplinary and, often, expensive support. Despite the various co-morbidities described earlier, it is the presence of CB that is often a major factor in determining quality of life, cost of care and carer health. Within the heterogeneous population of those with an ID, although described as a diagnosis, CB is very often a presentation resulting from any of a variety of physical or emotional causes or environmental triggers. These may include pain, for instance, earache or toothache, changes in care staff or care routines or side effects from some prescribed medications. Although several studies and reports have investigated environmental and behavioural strategies to try to limit the development and intensity of these behaviours, the causes still remain poorly understood. This limited knowledge base has in turn severely restricted the development of successful management strategies.

Management

How to interact with people with an ID

In most branches of medicine a doctor will come across individuals who have an ID. Although the level of interaction will clearly vary depending on the level of ability, there are some general principles that can be described.

Communicating with the patient using speech

- Remember that ID encompasses a wide range of intellectual abilities so communication needs to be flexible.
- Those with mild to moderate ID will understand some language.
- Those with very little speech may well be able to make use of signs or other communication aids.
- Before greeting a patient with ID whom you have not previously met, ascertain their level of ability with respect to communication.

- Speak clearly and not too quickly.
- Use short, clear and unambiguous sentences.
- Time should be allowed for the content of each sentence to be absorbed, particularly if the content is unfamiliar.
- Actual understanding of the communication should be regularly checked.
- To demonstrate respect and to develop a productive therapeutic relationship, it is important to communicate in a way which encourages the patient to engage and makes them feel they are being heard.
- At least until the level of a person's awareness of their environment is established, it should be assumed that they expect to be greeted and where appropriate addressed directly and not simply referred to in conversation between you and an accompanying carer.

History taking from carers but keeping the individual involved

Initially do not assume that the patient can not understand you. It is also important not to overestimate an individual's capacity to understand. To avoid doing this, it may be necessary at an early stage to talk to the person accompanying the patient, having obtained the patient's permission as appropriate and having explained your intentions. It is equally important to explain this to the patient. Mismatches between receptive and expressive language abilities are common in those with ID and should be considered if conversations do not follow the expected course or if the individual's answers do not seem appropriate to the question asked. It will often be helpful to use communication aids such as pictures, gestures and signs. Other specialties within multiprofessional teams may also have specialist skills that can support communication and assessment, including speech and language therapy, occupational therapy, specialist ID nursing and psychology. If any written records or prescriptions are brought by the patient or a carer, then these should be reviewed. In addition, however, as with psychiatric histories in general, gathering a collateral account is frequently an important part of the clinical process. If the patient has the capacity to decide whether or not you can approach an informant, then you should be guided by their response.

Principles of management

Conditions leading to developmental intellectual disabilities are, by the time they are diagnosed (with the exception of rare conditions such as phenylketonuria, which is screened for), generally not directly treatable or reversible. Management of the ID itself is aimed at ameliorating those aspects of the presentation for which treatment strategies may be available. In addition, however, and as noted earlier, the presence of an ID brings with it an increased risk for a range of physical, behavioural and psychiatric co-morbidities. These co-morbidities should be the focus of active management efforts. Sometimes the treatment approach will be similar to that used for the population without ID, but in other circumstances management strategies will need to be altered. However, in all circumstances it is important that the person with ID, if at all possible, understands their treatment and the reasons for it the best they can. In the case of pharmacological or other physical treatments, it is essential to be aware that the individual may be unable to report the presence of a side effect. Carers and clinicians should discuss the nature of possible effects, consider how these may be manifest in a particular individual and be on the lookout for these as well as for any other changes. On some occasions, it may not be immediately obvious whether a treatment has led to changes warranting either continuation or cessation. It is generally very helpful for clinicians, informed by the patient where possible and by the carers, to develop a written record in which the effects of the intervention can be reported day after day by the patient or their carers. This is often invaluable in determining how successful treatment changes are.

These principles of management are equally applicable to treatment of co-morbid physical, psychiatric and behavioural symptoms. Bearing this in mind, the treatments available for management of psychopathology in the general population will in many cases be applicable to those with an ID. People with IQs in the mild to moderately disabled range can often make good use of cognitive behavioural approaches as long as these are pitched at levels appropriate for them. For those with more severe impairments of function, behavioural management programmes can frequently lead to clinical improvements, although these will need to be developed by clinicians skilled in working in this area. Clinical indications for the use of psychotropic

medications generally resemble those in the wider population. It is essential to resist therapeutic nihilism; even if the ID syndrome, physical circumstances and life opportunities faced by an individual are not likely to be amenable to change or treatment, any additional medical or psychiatric problems should be actively treated. This may lead to symptomatic relief but also improves the quality of life which is no less important in those with ID than in others. Careful explanation, appropriate psychoeducation and ongoing support will be very important components of care as usual.

Decision-making and mental capacity in people with ID

Physical examination and investigation of individuals with ID raises several important and potentially complex issues. As full an explanation as possible of what you wish to do is critical, although for some patients this may be quite limited. Where possible use visual aids or mime what is intended before carrying it out. Issues of capacity to consent to examination, investigation and treatment are of enormous importance and can get quite complicated. In the United Kingdom these issues are considered under the Mental Capacity Act (2005) (see Chapter 5).

Simply having a diagnosis of an ID does not necessarily mean that an individual is unable to make decisions for themselves, although as the severity of the ID increases this becomes much more likely. The principles discussed in Chapter 5 are important to bear in mind.

With respect to an individual with ID the following points should be considered:

- An individual should be presumed to have capacity unless shown not to.
- Capacity should be assessed in relation to the particular issue in question.
- When assessing somebody with ID simply considering their IQ or 'mental age' is not sufficient. An assessment relating to the particular decision to be made should be carried out.

Information describing the relevant issues may be presented by any means appropriate, including spoken language, images, gestures and signs. Similarly, the individual's decision may also be communicated by any of these means. When assessing capacity in an individual with ID, the following approach is appropriate:

1. Obtain relevant background information: personal and medical history and accounts from informants.
2. Consider any current psychiatric/medical diagnoses.
3. Consider intellectual level (the presence of ID does not necessarily preclude capacity), and examine intellectual functioning with respect to the specific decision to be made.
4. Enhance capacity if possible, for instance, by providing information at the appropriate level of complexity and in the most accessible form for that individual. Assistance of a skilled speech and language therapist or psychologist may be appropriate.
5. Keep full clinical notes of the assessment.

If an individual lacks capacity for a specific decision, then whoever must make the decision in their place must follow the principle of 'best interests'. In the context of the inability to make a decision about medical treatment, the treatment finally decided upon must be for life, health or well-being and it must represent a necessity. Where there is a choice of treatments possible, the least restrictive or invasive alternative should be chosen. However, it is *very important* that the need to follow the 'best interests' approach does not end up disadvantaging the individual by excluding them from a treatment that might otherwise have been indicated had they not had an ID. Over the years, there have been well described accounts of people with ID being excluded from potentially life-saving treatments not because it was not in their best interests but purely because they have had an ID. Such an approach would be discriminatory.

How and where should healthcare be delivered?

In the United Kingdom, until the latter part of the last century, most healthcare for those with moderate and severe ID was delivered in institutional settings, often large long-stay hospitals or 'communities'. These original institutions have now almost all been closed down, and the vast majority of their residents have moved into a variety of 'community settings', including hostels,

group homes and supported living projects. Others with ID continue to live with their families and in all these settings they may, when required, be supported by clinicians working in multidisciplinary community ID teams.

In addition, arising out of a series of White Papers, from *Valuing People* (2000) to *Valuing People Now* (2008), a more assertive model of general healthcare delivery has been developed. This includes the registering of all those known to have an ID with their local general practitioner (GP) surgeries and the development of individual and regularly reviewed health action plans.

Mental healthcare is generally delivered by community-based multidisciplinary teams comprising professionals with a range of skills, including psychiatry, psychology, specialist ID nursing, behaviour therapy, speech and language therapy, occupational therapy and expressive therapies, including drama and music. The social care provided for people with ID, including everything from accommodation to daytime activities to 24-hour staff support if required, is the responsibility, in the United Kingdom, of local council social service departments. The well-being of people with ID is often critically dependent on these aspects of care, and therefore it is commonly the case that the health teams work very closely with social service agencies.

Summary

In this chapter the nature and range of intellectual disabilities, the more common conditions associated with ID and their most frequent co-morbidities have been reviewed. Because of the history of marginalization and discrimination that have characterized the lives of many with an ID over the years, a brief social history of the relationship between people with an ID and society at large has been provided. People with an ID present in all branches of medicine, and therefore this chapter has also provided some guidance in how to approach an individual and how to go about gaining a history. The point is also made that most of the skills that will support a successful interaction with a person having an ID are readily transferable to the rest of medical practice.

Further reading

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Fisch GS (ed) (2003). *Genetics and Genomics of Neurobehavioural Disorders*, Totowa, NJ: Humana Press.

Holt G, Hardy S, Bouras N (2011). *Mental Health in Intellectual Disability: A Reader*, 4th Edition. Brighton: Pavilion Publishing Ltd.

Lai M-C, Lombardo MV, Baron-Cohen S (2014). Autism. *Lancet,* 383:896–910.

Web-based resources for learning disability

A Step by Step Guide for GP Practices: Annual Health Checks for People with a Learning Disability. Available at: http://www.rcgp.org.uk/clinical-and-research/a-to-z-clinical-resources/~/media/Files/CIRC/CIRC-76-80/CIRCA%20StepbyStepGuideforPracticesOctober%2010.ashx

Royal College of Psychiatrists. Available at: http://www.rcpsych.ac.uk/Psychopharmacology and physical treatments

CASE STUDY 13.1

Each new set of information is followed by one or more questions. Think about possible answers to each set of questions before proceeding to the next set of information.

1. A 19-year-old man with mild learning disabilities, autism and epilepsy is becoming increasingly agitated. At his day placement, he is unwilling to cooperate with anybody and he stays in a corner and shouts at people to go away.
   a. What other information would you obtain in order to construct a differential diagnosis?
   b. Based on the aforementioned information, what conditions/situations may have contributed to this presentation?

(*Continued*)

CASE STUDY 13.1 (*Continued*)

2. There have been no changes to his environment, no changes in timetable, and no changes in the mental states of the other people he is surrounded by. However, he is observed to repeatedly shout 'leave me alone, go away'.
   a. What might be causing this?
3. When you go up to him he is willing to talk to you and he tells you that he is shouting at the angels to go away and leave him alone.
   a. What do you think may be causing this state?
   b. What aetiological factors do you think there may be?
4. He tells you that the angels have been calling him since his older brother died in a motorbike accident some months ago.
   a. What treatment options would you consider?
   b. What role do you think psychological interventions may have in these circumstances?
   c. You decide to prescribe an antipsychotic medication. How will you explain your planned course of action to the patient?
   d. How might you check that he understands what you are saying?
5. Once he has been treated and appears no longer to be psychotic, he tells you that in order to help him remember his brother, he would like to take all his money out of the bank and use it to buy a motorcycle. He tells you that he knows that he should not drive it as it can be dangerous and because he has epilepsy but he would just like to keep it in his front garden and look at it and clean it and occasionally sit on it.
   a. What factors would you consider when deciding how to respond to his request?
   b. How will you respond to this request?
   c. How would you determine whether he should be able to make such a decision?

Disorders of childhood and adolescence

CHAPTER 14

NISHA DOGRA

KEY CHAPTER FEATURES

- Behavioural problems in the under-fives, children and adolescents
- Attention deficit hyperactivity disorder
- Autistic spectrum disorder
- Emotional disorders specific to childhood (such as attachment disorders, separation anxiety and school refusal)
- Self-harm as a presentation
- Family problems
- Major mental illness and the differences in children and adolescents

Introduction

In this chapter we cover the disorders that are usually identified in childhood and adolescence or are specific to that developmental period. It is probably most helpful to categorize the disorders as behavioural, neurodevelopmental and emotional, although there is often overlap. It is also helpful to divide childhood into under-fives, childhood and adolescence (over 12 years of age). This enables the disorders to be seen in the context of expected developmental stages. It may be obvious, but in assessing child disorders, child development (physical, social, emotional, cognitive, moral) and family relationships are crucial components of the process. The aetiology chapter will have alerted you to the environmental factors that influence the development of mental health problems. For children the key environmental factors that may be significant are the family and school. For each disorder, a definition is provided before describing the prevalence and risk factors. The presentation features, any issues pertaining to assessment and management are then discussed. A clinical case scenario is presented to provide a clinical picture.

We begin with behavioural disorders in the under-fives and then consider oppositional defiant disorder before discussing conduct disorders. We then review two major neurodevelopmental disorders, attention deficit hyperactivity disorder (ADHD) and autistic spectrum disorder (ASD). In emotional disorders we consider anxiety disorders specific to childhood. Phobias present in childhood as they do in adults, and treatment options that are more widely used in child mental health are discussed. Self-harm is considered separately, as it is a behaviour that presents relatively frequently but is more often related to distress than mental health problems *per se*. We also discuss family problems, as these can cause quite significant distress to children. Major mental illness is only discussed in relation to issues specific to this age group as the diagnostic criteria for schizophrenia and bipolar disorder are the same as for adults. Depression may present in young people as it does in adults, but there are variations which are discussed briefly. Eating disorders are also not uncommon in this age group; however, these are covered in the chapter on eating disorders. Finally, we mention substance misuse, as it often presents in conjunction with other problems. Dependence can and does occur in young people but often not to the same extent as in adults, and often there are other underlying issues such as family problems.

Why is this relevant to you?

You may be wondering why you need this information as a medical student who is not even thinking about working with children, let alone children with mental health problems. Whatever area of clinical medicine you enter, you will come across children or parents, and having a basic knowledge of child mental health may help you deliver better care even if it is not for the child's mental health problem itself. In your foundation training years you are very likely to come across deliberate self-harm in young people, and again having some awareness of the issues the young person may be experiencing can help improve the care you provide. Up to 25% of young people may suffer from mental health problems with about 10% suffering from severe mental illness. However, only a fraction of these (approximately 10%) have access to services, and in many countries child mental health is not even considered.

When is a problem a problem?

There are several aspects to the young person's presentation that are helpful in establishing whether there is a problem or not as the symptoms are likely to be on the spectrum of everyday experience, and these are outlined in turn.

Age

A behaviour that is appropriate at one age can become a problem if it continues (for example, temper tantrums may be okay at 3 years but are not okay if they continue when the child is 10 years old).

Frequency

Something happening once may not be a problem, but happening more regularly it can be – such as panic attacks.

Severity

Feeling a little anxious may not be a problem and indeed may actually be helpful, but extreme anxiety that prevents you from daily activities may be very disabling.

Individual characteristics or temperament

A particular event may be managed appropriately by one child, but another child, because of his or her temperament or personality, can develop problems.

Impact on others

Behaviour may not be seen as a problem if adults around the child are managing it. The same behaviour can be seen as a problem if it cannot be managed or if it begins to have a negative impact on others (for example, a child's aggressive behaviour may be presented as a problem if it is a risk to other family members). Child psychiatry also has another issue to contend with, and that is when parents are convinced the child has a psychiatric diagnosis to explain behaviours that they are struggling to manage. There are concerns that children may be being over-labelled with disorders such as ADHD as adults displace their own responsibilities.

Clinicians should only make a diagnosis if there is the evidence to support this and not because of pressure from parents or other adults (for example, teachers).

Family/social circumstances

Behaviour may not be seen as a problem if the family are managing, but a change in family circumstances (such as parental depression, unemployment, financial difficulties) can mean the family's ability to manage is compromised. In addition, the presence of another disorder can compound already existing behaviours that previously were not seen as a problem (for example, a withdrawn child may not be a problem; however, a withdrawn child who refuses to go school may be a problem). Sadness is another example – being sad following bereavement is to be expected, but sadness out of the blue may be a problem that needs addressing.

It does not necessarily follow that a referral to specialist services is indicated as increasingly resources are being placed in the community to try and help the prevention of problem development through early recognition and support.

Behavioural problems in the under-fives

These are mentioned here as they occur commonly and most are not severe so that they can usually be managed with relative ease in primary care contexts and sometimes community paediatric services. It is important to note that the prevalence of actual behaviour problems is probably similar across different cultures, but what differs is how these are perceived. Asian families in the United Kingdom have in the past been less likely to present with sleep problems but may have greater concerns about non-compliance or oppositional behaviour. Much of the work in assessing and treating children with such problems is really through educating their parents/carers. Severity can usually be decided on by considering the factors discussed above in 'When is a problem a problem?' Only severe cases and cases that fail to respond to treatment (if treatment is effectively implemented) warrant more specialist interventions (which may or may not be specialist child psychiatric services depending on local arrangements).

Sleep problems

Settling to sleep

The child either does not settle when put to bed or is constantly up during the night. The behaviour is usually perpetuated by parents inadvertently reinforcing it by responding to the child when they leave the bedroom.

Nightmares

These can occur at any age and may be associated with specific life events such as an accident or abuse. They may occur in association with flashbacks and anxiety. A child who has had a nightmare is usually able to give a vivid account of their experience. Management usually involves identifying potential causes for the nightmare and addressing those issues (e.g. if the child is being bullied and the nightmares reflect this, tackling the bullying will help the nightmares). Reassurance can be helpful. Recall and discussion of a nightmare may lead to anxiety and reassurance needs to be provided in a way that does not reinforce the anxiety.

Night terrors and sleepwalking

These are most common between the ages of 6 and 9 years. Night terrors present with a history of the child sitting bolt upright while still asleep and appearing terrified. They may awake with no recall of what has just happened, but if awake are often disorientated and they can be difficult to engage. Children may sleepwalk in the presence and absence of night terrors. Both occur in the same stage of sleep. Most children outgrow both problems. Management largely consists of reassuring the parents and helping them ensure the child is safe if they sleepwalk. They also need to know not to try and wake the child during a night terror as this is likely to distress the child and also disorientate them.

Feeding problems

About 10% of preschool children can show difficulties or problems with their feeding at some stage.

Weaning

There may be problems weaning the child and for various reasons breastfeeding may go on for longer than needed (the length of time breastfeeding continues

will be dependent on several factors such as cultural, familial and economic).

Faddiness

The child is faddy and will eat a very limited repertoire. If given other foods they may be vomited back up, raising parental concerns. Parents worry about the child not growing, but these concerns are often unfounded. If faddiness is humoured, it is likely to continue. The most severe faddiness may lead to non-organic failure to thrive (that is when there is no identified medical explanation for why a child is failing to grow). This may need to be monitored. It is important to recognize that faddy eaters are not usually concerned with weight gain or such issues. Often they initially dislike the textures or tastes and the parental responses may shape further faddiness. Difficulties with feeding are common in children with ASD, discussed in the section 'Autistic spectrum disorder'. They are often also present in children with physical illness.

Behavioural problems

In younger children, the child is presented as being uncooperative, with unwillingness to comply with adult requests, and they have 'temper tantrums' when they do not get their own way. The behaviour is often described as wilful, defiant and may also be aggressive. This is most marked between 2 and 4 years, when the child is gaining a concept of self but does not yet have the skills for independence.

Oppositional defiance disorder

This is the continuance of many of the features described above such as temper tantrums, defiance, aggression and so on. It will extend beyond the family into school life to such a degree that it may impact significantly on learning and peer relationships. It can be diagnosed from toddler years up to age 10.

Conduct disorder

Oppositional defiance disorder beyond 10 years of age is usually defined as conduct disorder. It is included in both *International Classification of Diseases* (ICD) and *Diagnostic and Statistical Manual of Mental Disorders* (DSM) classifications but there is considerable debate about whether this is appropriate. Conduct disorder is grouped into socialized and unsocialized types, with some overlap. Socialized conduct disorder is usually viewed as less serious and tends to be phasic in nature. Unsocialized conduct disorder is more serious and potentially leads to criminality and a later diagnosis of antisocial personality disorder.

It is also worth noting that sometimes children of any age may develop behavioural problems in response to adverse life events, but this will usually resolve more quickly especially if there are protective factors in place.

Risk factors for behavioural problems include:

- Parents who lack confidence
- Parents who received indifferent parenting and struggle with setting clear boundaries
- Conflicting expectations by different adults involved in the child's care
- Family conflict, especially witnessing violence and aggression
- Child temperament
- Co-morbidity with neurodevelopmental disorders
- Learning disability
- Child abuse
- Organic problems (such as anaemia, lead poisoning)

Management

Parents who have not received adequate parenting themselves (for example, having grown up in care) may need basic education about limits and boundaries. Parents who were brought up by very strict parents may feel that they do not want to be as strict with their own children but then struggle as the child is in charge of the family. Understanding the different experiences of both parents can also be a useful exercise, as it can reveal much about the dynamics of what the child has to understand and negotiate their way through.

Simple behavioural therapy is usually the most effective front-line treatment. However, parents often require considerable support, especially if the problems have been long-standing, as they can lack confidence. Underlying issues such as parental conflict may also need to be resolved. For most behavioural problems in young children, the approach is straightforward. However, the application may not be, for carers may struggle to hold their own.

There is a need to be clear with the child in terms of what is expected from them, and the expectations have to be specific. It is unhelpful to say the child has to be 'good' as the behaviour that constitutes what is good will vary from adult to adult, and even from day to day. The adult responses to the child have to be consistent. It is unhelpful to young children if the same behaviour elicits different responses at different times as they then have no clarity about what is expected of them. The child also has to learn that there are consequences for unacceptable behaviours. The rewards and punishments (if used) must be meaningful to the child and realistically attainable for their developmental stage. It is unhelpful to expect young children to wait for weeks for rewards when they have little conceptual understanding of the future beyond a few days. It is also important for the adults involved in the child's care to be consistent, for the same reasons that they need consistency from the same individual. This can be especially difficult if children have to cope with growing up in two or more households.

When implementing the behavioural strategies, it is important that the adults remain in control and do not show emotion and engage in justifying what they are doing to the child. An under-five should not be treated as an equal. If a child is told not to do something and their response is to cry, the adult should not seek to comfort the child at that point as it leads to a lack of clarity about what is happening. If children learn that their crying distracts the parents, they will use this strategy again. Some parents worry that this will scar the child – it rarely does, but what it does do is help the child learn appropriate behaviour. The scarring is prevented by being emotionally responsive to the child at other times rather than while they are exhibiting undesirable or unacceptable behaviour. Parents may also need to ensure that their expectations are age and/or developmentally appropriate (for example, a 1-year-old child is unlikely to have the skills to eat their dinner without some degree of mess).

For sleep issues, it is useful to have good and clear sleep hygiene, which should resolve most sleep issues. Sleep hygiene is essentially the factors that interfere with falling and remaining asleep. For young children, a consistent routine is often required and limiting excitement before bedtime can be helpful.

A review is warranted if these simple techniques do not work; it may be that other disorders need to be excluded. It is worth noting that behavioural problems that are not addressed when children are young do not simply resolve themselves, and most will develop into oppositional defiant disorder and then into conduct disorder. The older the child is the harder it becomes to make effective interventions. In long-established presentations, family therapy may be warranted to help the family change the way they think about the situation and the role the child has come to be known by. It is not unusual for families to ascribe certain roles such as 'the naughty one' or 'the mischievous one' to children that can be difficult to change.

Attention deficit hyperactivity disorder

ADHD is a heterogeneous behavioural syndrome characterized by the core symptoms of hyperactivity, impulsivity and inattention (Box 14.1). If the *International Classification of Diseases*, 10th Edition (ICD-10) criteria are used, the prevalence is approximately 1%–2% of children and young people. Using the broader *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition (DSM-IV) criteria, ADHD prevalence rises to possibly 3%–9% of school-age children, and 2% of adults. Prevalence also varies across different samples (community versus clinical, and in different countries). However, as a condition, it is not culturally specific but it is likely that different families may tolerate different levels of symptoms.

Box 14.1 Key features of attention deficit hyperactivity disorder

- Triad of hyperactivity, poor concentration and inattention impulsivity
- Not situation or context specific, so assessment needs to reflect this
- Onset before age 6
- Mild cases do not require medication
- Moderate and severe cases require methylphenidate, plus psychoeducation and supportive therapies

Risk factors

Children with birth injury are more prone to ADHD as are children with learning disability and ASD. Fetal alcohol syndrome babies are also at risk. There is some genetic risk but this is unclear. ADHD is more common in boys than in girls.

Clinical factors

The key is a triad of impulsivity, hyperactivity and poor concentration. These features are pervasive in all contexts and are not situation specific. Onset is by 3 years of age, although it may not be identified at that stage. Children are, however, often presented because of secondary problems related to these symptoms, such as lack of academic attainment, aggressive and other behavioural problems, and poor peer relationships.

Assessment

Different localities will have different arrangements for who initially assesses children with possible ADHD. The assessment needs to be of a reasonable length of time. Distractibility for a few minutes does not indicate ADHD! Even if the child appears to have ADHD in the clinic context, a diagnosis should not be made unless there is evidence that this also occurs in other contexts (such as school and home). School observations are a useful adjunctive assessment context. There are many questionnaires that can be used to indicate various symptoms, but the diagnosis should be made on clinical assessment and not on questionnaire completion.

Management

Once the diagnosis is made and an explanation given to the child and carers, mild ADHD can be managed with behavioural strategies and psychoeducation of the child, carers and teachers. Moderately severe and severe cases usually require medication. The first-line medication is methylphenidate, and atomoxetine is another option.

Medication, if it is used, is only part of the treatment. Psychoeducation and behavioural strategies are also still required. Parent and child support groups are also recommended, but availability varies. This enables parents to learn appropriate management strategies and the child to learn appropriate social skills and management of their impulsivity.

Children on methylphenidate need regular monitoring because it can affect growth. As it is also a stimulant, it can cause psychosis, so any treatment should only be initiated by a specialist.

Autistic spectrum disorder

ASD is a lifelong condition that affects how a person communicates with, and relates to, other people. It also affects how they make sense of the world around them. ASD is a term that is used to describe a group of disorders, including autism and Asperger's syndrome (which was assumed to be a milder form without all aspects of the impairments being present). The word 'spectrum' is used because the characteristics of the condition vary from one person to another. Those with autism may also have a learning disability. There was a view that those with Asperger's syndrome tend to have average, or above average, intelligence and thereby would be less seriously affected. However, as they still have social interaction difficulties but can understand that they do not fit in, the problems they face may be greater. It is probably most helpful just to use the term ASD rather than try and guess where someone will be along the spectrum. The prevalence of ASD is around 1%. There have been concerns that the prevalence has been increasing, but this is probably most likely to be due to improved identification and less rigid application of the diagnostic criteria. It is significantly more common in boys (around four times as common) and indeed some experts have commented that the mild end of the spectrum is fairly typical male behaviour!

Risk factors

Risk factors include the following:

- The aetiology is unclear, but there is a genetic risk and it does appear to run in families.
- Children with learning disability are at increased risk of ASD.
- The condition is present before 3 years of age, but those with less marked features might not be identified until they are older.
- There is no credible evidence to link ASD with the measles–mumps–rubella triple vaccine.

Clinical features

This disorder also has a triad of key clinical features that are shown in Box 14.2.

In the under-fives, ASD may present through delayed development, especially language development. There may also be odd behaviours such as intolerance of specific textures, distress at apparently minor changes, an indifference to pain, a lack of engagement with primary carers, and a baby or toddler who is difficult to settle. Faddiness is also not uncommon. However, given how common faddiness is in young children, this behaviour may not reveal much.

There may also be adult concerns about the child's social relationships – for example, poor eye contact, thought to be deaf as does not respond to social interactions, often does not want to play with others, does not have imaginative play and appears not to understand social cues and rules. In school-age children, presentation is likely to be through behavioural problems, which are an indication of the child struggling with expectations within a new environment and increasing expectations that the child should be learning to work with peers. The relationships they have are usually on their terms and they struggle to understand other people's perspectives. They are unable to pick up the subtleties of non-verbal language or understand the fine nuances of language. They often do not understand jokes, sarcasm or a play on words. They may have some very obscure interests and talk to people about them without realizing that the other person does not share their interest.

Box 14.2 Clinical features of autistic spectrum disorder

- Impairment of social interaction.
- Impairment of communication.
- Restricted, repetitive and stereotyped patterns of behaviour, interests and activities.
- There is also a school of thought that has impairment of imagination as part of the triad of impairment.
- Management involves providing appropriate supportive therapies and helping the child's perspective to be understood.

In adolescence anxiety, features of obsessive–compulsive disorder (OCD) and behavioural reasons are often the features that lead to presentation at services. In all these situations, the young person rarely sees that they have a problem and usually feel the issue is for others to address.

Some individuals with less marked features or features that those around the individual have adapted to may not present until adulthood.

Treatment

Despite many claims, there is absolutely no evidence that ASD can be treated, however there are interventions that can be made to reduce the impact of the difficulties. Management is a case of helping the family to understand the young person and helping the young person learn to operate in a world they do not really understand. Many of the interventions are educational and based in educational settings, or behavioural strategies to manage difficult behaviours. One of the key clinical features is that children with ASD struggle with change so can present with anxiety or what appears to be OCD. A thorough history and assessment will identify the underlying problems. Cognitive behavioural therapy, by the very nature of how it works, may be less useful in children with ASD, but they can be helped to manage their feelings in socially appropriate ways.

Emotional disorders of childhood

These may include anxiety, worry, fear, sadness and depression, and anger. Anger is not in itself a mental health disorder, but an inability to manage anger and other emotions in appropriate ways can be a problem as it may impact on the young person's development.

Only those anxiety disorders specific to childhood are covered here as the features of the other disorders (such as generalized anxiety, phobias and OCD) are similar to those in adults. Interventions will usually involve parents as co-therapists but similar techniques and strategies are employed. They are not generally treated with medication as the first response.

The disorders that will be covered here are attachment disorders, separation anxiety and school refusal. Before we discuss these, it will be helpful to consider the concept of attachment.

Attachment refers to a specific type of biologically based relationship, which provides a secure base from which children can explore the world. Work by Mary Ainsworth et al. in the early 1970s and then others has enabled attachment to be classified as either secure or insecure, with three types (ambivalent, avoidant or disorganized) of insecure relationships identified.

When children have secure relationships with their primary caregivers, they feel secure and safe in the understanding that they will have their needs (physical and emotional) met. They form a sense of confidence about themselves and the world around them and are able to explore the world beyond their immediate surroundings in the knowledge that they have a secure base to which they can retreat for safety. They separate easily from their caregivers, although they may be upset, as they trust that the caregiver will return.

Children who, for various reasons (such as primary caregivers with a history of postnatal depression, unwanted pregnancy and/or other adverse perinatal events, other parental illness, abuse and neglect, temperament clash between child and primary caregiver), do not develop secure attachment are described as having ambivalent, avoidant or disorganized attachment. These bring different types of behaviours and may have different consequences, but common to all will be that the child's psychological development is limited, and their ability to develop appropriate relationships with others will also be affected.

Ambivalent attachment tends to lead to very anxious and clingy individuals who fear trying anything new and lack confidence. Even when the parent seeks to offer comfort, they are likely to remain distressed and may be aggressive towards the caregiver. They are wary of strangers and as adults often struggle to trust others but at the same time are very needy.

Children with avoidant attachment styles tend to avoid parents and caregivers and appear to be unaffected when they are left by them. These children might not reject attention from a parent, but neither do they seek comfort or contact. Children with an avoidant attachment show no preference between a parent and a complete stranger. They are often very friendly and seem to be socially skilled. This is often seen in children who have experienced multiple caregivers and in some ways may be a way of protecting themselves. As adults, those with an avoidant attachment tend to have difficulty with intimacy and close relationships. They struggle to share their thoughts and emotions with others and may appear very self-reliant.

Children with disorganized attachment show a combination of ambivalent and avoidant features that reflect lack of consistency in the care given.

Attachment disorders

Attachment disorders appear under disorders of childhood and give the false impression that the disorder lies within the child. A diagnosis is a reflection of the child developing and presenting particular symptoms that reflect a failure for the child to have developed appropriate attachment. However, the reasons for this often lie in the adults caring for the child and their interactions with the child. It is important that attachment disorder is not diagnosed without clarity of context, as attachment is a two-way process.

Presentation

Broadly, two types of attachment disorder have been identified: (1) those where children are indiscriminate about who they form 'attachment' to – they are inappropriately friendly with everyone and show little wariness; and (2) another group whose attachment is more defined in relation to the child's behaviour towards the caregiver. Children with insecure attachment, especially disorganized attachment, are at significant risk of later emotional and behavioural problems.

Children will show developmentally inappropriate social relationships and may also present as having an anxious relationship with caregivers and/or disinhibited behaviour (behavioural problems, inattention, poor concentration, aggression) or asocial behaviour (lack of empathy or ability to see another perspective) in which ADHD or ASD may be mistakenly diagnosed. Learning disability may also need to be excluded, because children who demonstrate friendliness to strangers may be doing so as they do not understand the contexts for different social relationships.

Management

To date there is no known effective treatment for attachment disorders, although several psychotherapeutic techniques are used on the basis of limited evidence even though this is increasing. Therapies such as holding therapy have gained popularity despite the lack of evidence. Working with the

child–parent dyad in a therapeutic environment, helping them build an appropriate relationship, may be helpful. Family therapy may be appropriate in some contexts but would be inappropriate if there is ongoing abuse (emotional or otherwise). Social cognitive treatment approaches may be more beneficial given that there is greater evidence of efficacy for changing specific behaviours (such as peer rejection). It is also helpful to treat any mental health issues in the parent (for example, depression) if this is present.

Separation anxiety

This is anxiety when the child is separated from their primary caregiver. Separation anxiety is part of normal development but is considered a disorder when it persists beyond about 15–18 months (although it may reappear in childhood at times of distress or adversity). Prevalence is approximately 4%, but definitive rates are difficult to establish as many do not present to services. It most commonly presents in preschool or early school years and is characterized by features of anxiety associated with actual or anticipated separation from a particular adult, usually the mother.

Insecure attachment relationships, coupled with high levels of parental anxiety, are associated with increased risk of anxiety disorders generally in children, particularly, separation anxiety. Temperament may be of relevance, as may previous experience.

Presentation

At separation or the thought of separation, the child becomes distressed and exhibits signs of anxiety such as hyperventilation, feeling sick, panic and alarm. The anxiety may result in difficulty settling at night and reluctance to go to school or to be alone – this can be to the extreme of refusing to be alone in a room even when others are in the house. Parents may report that the child follows them everywhere and may even physically cling to the parent. The parent may also experience anxiety at the prospect of separation. Children may be the ones who decide on the rules in the family as the parents struggle to cope with the child's distress.

Assessment should explore the presenting features and likely precipitants, especially parental anxiety, which may exacerbate the problem. The dynamics of the parent–child relationship need exploration, particularly the transmission of anxiety between them, to identify parental behaviours and anxieties that perpetuate the problem.

Management

Some work with the family is usually indicated, with individual work with the child dependent on age and cognitive development. Specific interventions are mostly behavioural and usually involve graded exposure to separation starting in the situations defined by the family as least distressing. Unintentional rewards for clingy behaviour should be identified and avoided. Separation should be rewarded with positive comment and praise. If the features re-emerge (as they may do at times of transition and stress), cognitive behavioural therapy may be warranted. Family work should address parental expectations, anxiety or behaviours which in themselves may exacerbate the anxiety. There is insufficient evidence to support the use of medication.

School refusal

School refusal presents throughout childhood, especially at times of transition, for example, when starting or changing school. School refusal usually peaks during early adolescence, when it may represent a combination of adolescent stress and the revival of an earlier overdependent parent–child relationship. The increased need for independence and autonomy posed by the demands of secondary school may precipitate an avoidance of school. It may sometimes follow difficult experiences at school, such as bullying or being told off by a teacher.

The problem can manifest after a time of change at school or a period of illness. The unwillingness to go to school is often expressed openly, and the young person may say there is a particular student or lesson that they dislike or find anxiety provoking. Younger children especially may complain of headaches and abdominal pain to avoid school. There can be reluctance to accept that the anxiety rather than school is the problem.

Delay in the recognition of the underlying psychological basis for the problem greatly exacerbates the difficulties and by the time young people are referred to specialist services (in those where mental health is

an issue), the patterns are long established and may be difficult to change.

Management

School refusal is most appropriately managed by the education welfare service unless there are specific mental health problems that warrant specialist Child and Adolescent Mental Health Services (CAMHS) involvement. There should be a clear and consistent approach to returning young people to school. The school should involve the parents, and if appropriate the child, in devising programmes or plans. Parents need to support the programme to introduce the child or young person back to school. A phased return into the school environment especially when absence has been prolonged can be a useful way to start the return. The longer it is left, the more the young person will feel that getting back is insurmountable. Parents need to be firm about what is expected of the young person and resist the temptation to accede to the young person's distress. Cognitive behavioural techniques to help with the anxiety may be indicated. Tuition at home is usually an unhelpful strategy, as it can perpetuate the problem and impedes appropriate social and educational development.

The problems may manifest themselves after school holidays and parents need to ensure that their management remains clear and firm.

The prognosis is not good for a significant minority of adolescents. Up to one-third of school refusers fail to maintain regular school attendance and may have a predisposition to anxiety or agoraphobic symptoms. If there are concerns about parental commitment, this may be appropriately discussed with social services or addressed legally.

Self-harm

It is important to emphasize that self-harm is a symptom or behaviour and not a diagnosis. This is common in adolescence but does occur in younger children. Exact prevalence rates are hard to state as so much of it does not present to services. There is now some good evidence that most young people who self-harm are not mentally ill but that self-harm is usually related to life problems and there is generally low suicidal intent. There is a tendency for non-mental health professionals to hear the term 'self-harm' and make an immediate assumption that this then falls under the remit of mental health services. All doctors should be able to undertake a basic psychiatric history and risk assessment to identify those that need specialist help and those that might be managed through psychoeducation and help with the problem that has led to the self-harm. Repetition of self-harm is common and appears to be most likely where there is significant psychosocial disadvantage or mental health issues. Asking young people about self-harm does not increase the likelihood of them self-harming. In fact the effect can be quite the contrary, as in asking about it the young person may be relieved of guilt for having such feelings and of feeling understood. Also, assurance that feelings of self-harm are not uncommon can also be helpful.

Risk factors

Risk factors associated with self-harm include:

- Previous self-harm
- Substance use and misuse
- Presence of a mental health problem such as depression
- Being in a vulnerable high-risk group
- Physical, emotional or sexual abuse
- Being lesbian, gay or bisexual
- Loss of relationship
- Poor or inadequate coping skills
- Family history of self-harm and suicidal behaviour
- Availability of means (availability of firearms increases likelihood of completed suicide)
- Family discord
- Parental mental illness and/or parental substance use
- Intercultural stressors
- School and peer problems
- Unemployment
- Poverty/homelessness

Clinical features

Clinical features of self-harm include:

- Self-harming behaviour or indication that they are considering self-harm.
- Self-harm can be cutting, overdose or strangulation.
- Recent common adverse events, such as relationship break-up, falling out with parents or peers.
- Chronic psychosocial problems such as family problems, bullying, peer relationships.
- Substance misuse.
- A small number may have mental illness.

Young people who self-harm typically exhibit:

- Feelings of insufficiency and low self-esteem
- Lack of supportive relationships and low family cohesion
- Cognitive distortions and attribution styles
- Responses to problems using maladaptive cognitive strategies relating to expectancies, commitments and explanations for events

Management

Self-harm requires assessment and it may be that increasingly more of the less serious self-harm assessments are undertaken by non-specialist healthcare staff. There is a need to undertake a risk assessment. For most young people and their families, the assessment is an opportunity for the young person's perspective to be heard. It is really important not to be dismissive of the young person's actions or feelings. Most young people self-harm because they lack appropriate coping skills and humiliating them does not help them develop the relevant skills. Few young people who have self-harmed are likely to need specialist CAMHS but can be directed towards counselling support or family support depending on the underlying issues.

Family problems

The reason for mentioning this here is that young people often present with potential 'mental health symptoms' such as self-harming, low mood, general unhappiness, irritability and behavioural problems (such as petty criminality and substance misuse). An assessment shows that the underlying problems are family issues or a difference between parental expectations for the young person and the young person's own wishes. Cultural issues in the broadest sense can be quite significant in these types of problems.

Issues that may give rise to problems are:

- Pressures to conform to practise their family's religion or other practices that do not sit comfortably with the young person
- Pressures to conform to expected gender roles (boys wanting to pursue careers generally considered to be in the female domain such as nursing, child care and vice versa)
- Pressures to conform to the social norms, e.g. the expectation that a young person will go on to further education despite this not being what the young person wants
- Pressures to conform to family expectations that differ from what the young person wants, e.g. an expectation that the young person work in the family business
- Sexual orientation
- Impending forced marriages
- Difficulty in reconciling the culture in the private and public domains of their lives (feeling they have to be a different person in the two contexts but not being able to do that comfortably)

EXERCISE 14.1

Consider your own teenage years. Were there areas of conflict between you and your parents? How were these differences resolved?
Differences between parents and young people can be a part of the process of growing up as adolescents establish their own values. These only tend to come to the attention of services when the young person responds with maladaptive coping strategies (self-harm or conduct disorder) or if they present with emotional problems (such as anxiety or depression).

Mental disorders

Depression

Depression is the persistence of low mood that lasts over several weeks. The issues of definition of depression and the accuracy of screening methodologies may be even more salient than they are among adults, but estimates of prevalence are in the range of 1%–2% for prepubertal children and 3%–8% for older adolescents. From the age of 14 years onwards, the adult preponderance of females with depression emerges. There is reasonably consistent evidence that the prevalence of depression in young people, certainly among adolescents, has increased in recent years. Changing social contexts of expectations of young people and family structures may be plausible explanations.

Risk factors

As with most disorders of childhood, there is no single aetiological factor. Family history is a risk factor, although sometimes it can be difficult to differentiate whether the risk is family history or that family members experience the same adverse circumstances. With the exception of physical illnesses, risk factors for depression in young people are not too dissimilar from those among adults, although the aetiology is generally held to be more psychosocial than biological. That being said, although parent–child relationships will clearly play an aetiological role, one review estimated that parenting accounted for only 8% of the variance in the liability for childhood depression.

Clinical presentation

Presentation varies with the young person's developmental stage. Symptomatically, adolescents and adults do not differ greatly, although adolescents report fewer of the features of somatic syndrome. Irritability and anger are more commonly observed than in depressed adults. Associated with the low mood, there may be biological features such as sleep problems, poor concentration and motivation, appetite disturbance (in young people increased appetite may be present or the usual loss of appetite), anhedonia and negativity. Issues of self-esteem and poor confidence are also common in adolescence. In some young people the depression can be severe enough to present with psychotic features – delusions of worthlessness are not uncommon. Thoughts of self-harm are very common in adolescents and suicidal ideation is common in those with low mood. Asking young people about their self-harm can be very helpful as it may help the young person from feeling isolated and alone. Co-morbidity is not uncommon and young people may use alcohol to treat their low mood without realizing that the alcohol actually worsens the situation. Depression and anxiety copresenting is not unusual, and it can be difficult to identify the primary problem. Young people with depression may have self-esteem issues and it can be difficult to tell if these lead to relationship difficulties which cause depression or result from the low mood.

Depression can occur in younger children, but exploration often shows them to be unhappy with the world around them rather than being depressed. Younger children may present with changes in behaviour, becoming more irritable and moody. As with adults, they may also present with physical symptoms such as headaches and stomach aches.

In both groups, there may be deterioration in their academic work and social relationships. There may be family arguments because the family does not recognize the young person's problems.

Management

For depressed children, psychotherapy and/or counselling are the usual treatments of choice. Family involvement (often with a strong educational component and involving parents as co-therapists) is frequently deployed. From the age of about 10 years and over, cognitive behavioural therapy (CBT) is the more usual first-line treatment and there is evidence of its efficacy in adolescents both in the short and in the longer term. There is no good evidence that antidepressants are effective in childhood depression.

For severe to moderate depression in adolescents the first-line treatment is medication. Only fluoxetine is licensed for the treatment of depression in young people under the age of 16 years. Alongside the medication there is a need to offer counselling or supportive psychotherapy to help resolve some of the issues that precipitate or perpetuate the illness.

Dependent on the relevant factors, family therapy may also be required. Initially, CBT may be difficult as the young person is not in a state where this can be utilized, but as their mood improves cognitive behavioural therapy is a very useful adjunctive treatment. There is a slight but statistically significant increase in self-harm (but not suicide) among young people who are prescribed selective serotonin reuptake inhibitor (SSRIs). Sometimes this may be due to a syndrome of unpleasant restlessness/agitation that can occasionally occur as an adverse effect and about which patients and parents should be warned. In light of the warning prescriptions of SSRIs fell, but sadly suicide rates rose thereafter among young people in Holland and the United States, suggesting that untreated depression is the major risk factor for suicide.

Psychosis

Like self-harm, psychosis is a symptom and not a diagnosis in itself. The most likely causes of psychosis in young people are substance misuse, depression, organic medical problems, bipolar disorder and schizophrenia. A thorough history and assessment is required. Medication is used to manage the psychosis and then appropriate treatment is given for the diagnosis. Psychosis may present with risk to the young person and/or others dependent on the nature of their perceptual symptoms.

Bipolar disorder

Bipolar affective disorder is relatively uncommon in children and adolescents. A young person may be treated for an episode of depression but then later turns out to have bipolar disorder when a manic episode occurs. The diagnosis of bipolar disorder in children is a contentious issue and it is more commonly diagnosed in the United States. A more conservative diagnostic approach is generally adopted in Europe. Conservative diagnosis would require a typical episodic picture of adult manic symptoms, whereas liberal criteria would consider irritability and mood fluctuations/lability to be symptomatic of possible bipolar disorder. Differentiating such symptoms from adjustment difficulties in various phases of development is clearly problematic, and there is the additional complication of possible co-morbidity.

Schizophrenia

The prodromal phase of schizophrenia may begin in adolescence. The family may describe something not being quite right and the young person often insists all is well. Assessments are helpful to establish the history but it can be difficult to provide a definitive answer. The presentation and clinical features are as for adults and not discussed any further. It is rare for a definitive diagnosis of schizophrenia to be made before the age of 16 years, though this is occasionally justified by the history, symptoms and behaviours presented.

The management is as for adults in that medication is used to help manage the psychosis. Psychoeducation of the young person and their family is crucial. For understandable reasons, clinicians can be reluctant to make the diagnosis at the time of the first presentation given the long-term implications. However, it is important to identify this early to be able to provide the necessary support.

Substance misuse

Young people are unlikely to present with dependence on substances, although this is changing. In most young people substance misuse is related to adverse personal and social circumstances. Alcohol is often used to manage the symptoms of anxiety and/or depression. Substance misuse may lead to affective presentations that necessitate referral to specialist services. A joint approach between specialist services for substance misuse and child mental health services is usually appropriate as the former can address any substance misuse issues and the latter any individual or family issues.

Anorexia nervosa and bulimia nervosa

These are not covered here as they are addressed in Chapter 11. Presentations are increasingly occurring in younger children. Family therapy has proven efficacy in children under the age of 14 years and is the treatment of choice. It is often supported by individual work with the young person.

Summary

In this chapter the most common mental health disorders of children and young people have been covered. Although a detailed knowledge of them is not needed for most doctors, awareness of the conditions is relevant, as early identification and referral to appropriate agencies can ensure that the potential negative impacts are reduced.

Further reading

Dogra N, Parkin A, Gale F, Frake C (2009). *A Multidisciplinary Handbook of Child and Adolescent Mental Health for Front-Line Professionals*, 2nd edition. London: Jessica Kingsley.

CASE STUDY 14.1

Oliver is 3 years old and his mother presents to you as her GP at the end of her tether. She and her husband have been having marital problems and she feels Oliver maximizes the tense situation. Oliver defies his mother in all aspects, refusing to cooperate with any bedtime routine and being uncooperative at meal times. Playschool staff also have some problems but not as often and of the severity as experienced by his parents.

What is the likely diagnosis? What would be the most appropriate management?

The most likely diagnosis is oppositional defiance disorder.

There is a need to implement straightforward behavioural therapy. The parents need to agree on the rules and enforce them consistently. They need to ensure that they do not reinforce unacceptable behaviour by giving into Oliver's demands and undermining their own roles.

CASE STUDY 14.2

John, aged 16 years, was referred to CAMHS as he had told the GP he was unhappy and wanted to die. John was offered the opportunity to meet with the child psychiatrist alone but was unable to make a decision so was seen with his mother. John thought he had been referred to CAMHS because 'I get depression' and 'I find school very uncomfortable'. When the concept of depression was explored with him, he felt he did not want to live but also did not want to die. He had not tried any self-harm but often wondered what drowning might be like. He wondered about the point of it given we all ultimately die. His unhappiness stemmed from difficulties with peers. He had never had good peer relationships and his mother recalled that he had never really had a friend. John felt he did want friends but none of his peers at school met his high expectations or standards. He does not enjoy school because of the problems with peers but manages the academic work.

There were no problems with John's pregnancy. He was an irritable baby compared with his siblings. His mother recalled that he had always been a picky eater and had a very small, rigid and limited repertoire. He was always very sensitive to smells, textures and noises. As a toddler in a playschool he preferred to play alone than with peers. He liked to play with the same toys and would often spend hours lining up the toys in a particular order and become distressed if the order was disrupted. His mother could not recall if he was an affectionate baby, but she felt he was less responsive than his sisters. John was cooperative with the assessment. When he struggled with a question he was even more inclined to avoid eye contact, which was poor throughout the interview. He struggled to express himself clearly and often used words such as 'depression', which he could not describe further. It was as though he had heard the words but did not really have an understanding of the concept.

(*Continued*)

CASE STUDY 14.2 (*Continued*)

His speech was monotonous with no variation in tone or pitch. He did not appear depressed and was not suicidal as exploration revealed that he saw life as pointless on a philosophical level rather than because of how he felt.

What is the most likely diagnosis? What would be the most appropriate management? What would be the place of counselling using a psychotherapeutic approach?

ASD is the most likely diagnosis and John fulfils the triad of impairment.
An explanation of the disorder was given to John and his mother. This included explaining the impact of the clinical features on everyday life. The diagnosis with permission was communicated to the educational psychologist who was able to initiate school interventions to help John better manage school. His mother was also advised of a support group for parents to help manage some of John's difficulties and also develop strategies to manage aspects of his difficult behaviour. His mother learned that it was important to be aware that John did not understand concepts such as compromise and understanding other people's perspectives.
John's mother queried the possibility of individual work with John using a psychotherapeutic approach. Given John's very rigid and inflexible thinking, it was agreed that this would not be helpful. A more behavioural approach was taken to help John learn socially acceptable behaviour.

CHAPTER

15 Old age psychiatry

SIMON BUDD

KEY CHAPTER FEATURES

- Presentation of mental disorders in older people
- Features of mental illnesses in older people
- Intellectual disability in older people
- Management of mental illness in older people
- Elder abuse

Introduction

When do we become old? To children, their parents may seem old. There are people in their 80s or even 90s doing things that younger people cannot or dare not do. Ideas about old age have changed considerably with increased life expectancy. The changes of aging both biologically and psychosocially mean that those who have become older have different presentations, disorders and needs, compared with those in their earlier years.

Patients over the age of 65 years old are typically seen by psychiatric services for older persons. However, the Royal College of Psychiatrists recommends providing needs-based services rather than age-specific services; so younger patients may be appropriately seen by services for older patients because their needs may be similar.

Why is this relevant to you?

The United Kingdom has a current population of roughly 65 million and about 10–11 million of these are over 65 years old. About 14%–15% of the over 65s are over 85, with over half a million people over 90 and nearly 14,000 people over 100 years of age (a rise of 70% over the past decade). Overall life expectancy has risen to about 79 years for men and 83 years for women. The proportion of older people in the population and the life expectancy are growing, both in the United Kingdom and across the world in

Box 15.1 Reflective exercise

What are the physiological and psychological changes of normal ageing?

developed and developing countries. It is estimated that by 2050 the over 80s will have increased over fivefold from the start of the century to nearly 380 million. Hence, knowledge of psychiatric disorders in older people is important, if not essential to medical practice irrespective of speciality or geography of practice.

There are variations to the presentations of psychiatric disorders in older people and conditions such as depression and organic disorders, especially dementia and delirium, are common. Alcohol misuse may remain unidentified. There is a marked co-morbidity of alcohol misuse with physical disorders and the effects of the aging process have effects on the management of health problems of older people. Older people are highly represented in in-patient groups, especially in general hospitals, and in some wards they will be the main group, e.g. elderly medicine or orthopaedic wards. They form a high proportion of attendees in primary care and have multiple physical, pharmacological and social issues as well as mental health problems.

Organic mental health disorders

Organic mental disorders, particularly dementia and delirium, are covered in Chapter 12 and should always be considered in the differential diagnosis of patients presenting with psychiatric symptoms.

Physical disorders

Older people have more and frequently multiple co-morbid physical disorders. The symptoms of these disorders may be the same as those for psychiatric disorders, e.g. low energy and motivation in the case of heart failure, and low energy with reduced appetite and weight loss in the case of malignancy. Physical disorders may present along with psychiatric symptoms, may worsen current psychiatric disorders (pain may seem much worse if you are depressed) or may precipitate psychiatric disorders (chronic pain is a recognized risk factor for depression and suicide).

Substance misuse

Although alcohol misuse is a problem across all age groups, it is more likely to be missed or go unrecognized in older people. Drinking levels, of one in five men and one in ten women of older people, are in the harmful range, with increasing numbers drinking enough to cause harm. These figures have increased by 40% and 100% in males and females, respectively, during the past 20 years. Old age can bring life changes or transitions, which can trigger alcohol misuse or increase pre-existing alcohol problems. Life events such as bereavement, illness, social isolation, boredom, poor mobility and pain or a combination of these may precipitate use or increase use of alcohol. Older drinkers are more likely to drink at home and so misuse may be missed in the clinic or hospital setting.

Older drinkers may have less understanding of the risks involved and may use mixers in the belief that it makes their drink less alcoholic. Given that alcohol is a water-soluble drug, the effects per unit are increased compared with that in younger adults (see the section 'Pharmacology' below). Older people may under-report their substance misuse as they may feel stigmatized or not see its relevance.

The use of alcohol increases the risk of physical disorders (infections, stomach problems including ulcers, anaemia and liver abnormalities), falls (with the attendant complications such as fractures, reduced mobility and subdural haematomas) and problems with drug interactions (the effects of certain drugs, e.g. warfarin, may be reduced).

Alcohol is also neurotoxic and can cause alcohol-related brain damage, with older people particularly at risk. This may range from mildly impaired cognitive function to dementia and high levels of alcohol consumption increases the risk of dementia. For those with co-morbid schizophrenia, the combined effect may be greater than either risk factor alone.

In severe cases or when drinking stops suddenly, Wernicke's encephalopathy and Korsakoff's syndrome

may occur (see Chapter 12). Korsakoff's syndrome and dementia linked to heavy alcohol misuse may lead to admission to 24-hour care.

Patients should be screened, particularly if they have physical or mental health problems or if they are affected by major life events. 'Adult' safe limits may not apply for older patients and there is no 'safe' limit for older people with mental health disorders. Doctors should avoid stereotyping or making assumptions such as older women cannot have alcohol problems. Women with alcohol misuse are more likely to be widowed, separated or divorced or have a partner who also misuses alcohol. They may have a history of depression (up to one in seven people with depression may have an alcohol misuse problem) or be taking psychotropic medication.

Screening can be can be undertaken with screening questionnaires such as the Alcohol Use Disorders Identification Test (AUDIT, recommended by the World Health Organization as the screening tool of choice) though lower cut-off or thresholds should be used for the elderly. The Short Michigan Alcohol Screening Test-Geriatric Version (SMAST-G) was designed specifically for use in older people. The usefulness of the CAGE (cut down, annoyed, guilty and eye-opener) questionnaire may be limited in older adults.

Older people can and do benefit from treatment. Any underlying condition that may be perpetuating the misuse should be treated. Thiamine, vitamin B_1 (and other vitamins), should be prescribed for any patient who is suspected of having alcohol-related problems (it is cheap, has minimal interactions and may prevent cognitive problems, especially Wernicke's encephalopathy or Korsakoff's syndrome). Support from the drug and alcohol services should be sought and patients should be managed along pathways similar and appropriate to younger adults as discussed in Chapter 10.

Benzodiazepines are one of the most commonly prescribed drugs in older people (in up to 15% in the community). There is a 50% increase in the use of these drugs between the ages of 65 and 75 and a fifth of older people admitted for alcohol misuse problems also misuse benzodiazepines. They are often prescribed for sleep problems but increase the risk of confusion, falls and over-sedation, especially when used with alcohol. Patients can quickly become dependent, both physically and psychologically. Benzodiazepines should be avoided if possible in older people. Advice on the physiological changes with age and on sleep hygiene should be considered first, as well as other possible causes of insomnia, such as depression.

Although the exact numbers of older people using illicit drugs is not clear, the rates of use are increasing. Good practice would be to enquire about illegal drug misuse in patients in this age group.

Very late onset schizophrenia

The term paraphrenia was used in the earlier days but the preferred current diagnoses are either 'very late onset schizophrenia' (VLOS) or 'delusional disorder', depending upon the symptomatology.

VLOS is regarded as schizophrenia symptoms developing in those over 60 years. It is relatively common with nearly 3,000 new patients presenting per year in the United Kingdom, affecting nearly 35,000 patients all together. Psychotic symptoms are probably under-reported, as patients are often socially isolated and suspicious about revealing their beliefs. They may have had symptoms for many years before presenting to services. It is more common in women than men, with the female to male ratios varying from 3:1 to 20:1.

The cause of late onset schizophrenia is unclear. It has been hypothesized that it is linked to age-related neurological deterioration, triggered by some external environmental factor or stressor (such as life events or illness). There is evidence of increased structural damage in the brain of these patients compared with those of patients with early onset schizophrenia. There may also be possible genetic predispositions and personality traits, such as schizoid or persecutory, linked with VLOS. Sensory impairment, particularly deafness, and social isolation have been considered as potential significant risk factors (there is an association but causality has not been established). There is no clear link between VLOS and dementia, though dementia as a differential diagnosis should be considered in patients presenting with psychotic symptoms.

Patients suffering from VLOS have very similar psychopathology to those diagnosed with early onset

schizophrenia. The main difference is that those with VLOS have much better preserved personalities and rarely have negative symptoms or formal thought disorders. They often have good premorbid levels of psychosocial function (though may be socially isolated later in life) as well as good educational levels and occupational history. Delusions and hallucinations are common, and the hallucinations may be olfactory or tactile, as well as auditory. Persecutory delusions that someone wants to harm them are common. The so-called 'partition delusions' may be present and are more common in older than in younger patients. These delusions occur when the patients believe that people, gas, electricity, or some other force enter their home through the walls from a neighbouring dwelling with a persecutory intent, e.g. the neighbours are pumping gas through the walls or heating systems into the patients' houses to poison them. Auditory hallucinations may be third person, usually talking in negative or derogatory tones, a running commentary or discourse about the patient's actions. The voices may accuse the patient of being bad or evil or of having committed a crime or illegal activities.

The behaviours resulting from delusional beliefs or hallucinations may have an impact on the neighbours and lead to conflicts with housing or social services. Patients often have little insight into their illness and resist the involvement of psychiatric services refusing medication. It may be best to establish a therapeutic alliance in the community through nursing colleagues. The treatments are the same as for early onset disorder – antipsychotic medication and psychological therapies as discussed in Chapter 9. Patients frequently make good recoveries though there are often residual memories of the symptoms, which get rationalized to account for the illness and its effects.

Affective disorders

Bipolar disorder and mania

The majority of older patients presenting with manic symptoms are graduate patients with bipolar affective disorders. Primary manic episodes are uncommon in older people so organic causes should also be considered as frontal lobe tumours (primary or secondary) can present with symptoms of mania. Other organic disorders should be considered in the differential diagnosis including dementia, delirium and thyroid disease.

Hypomanic symptoms in older people can be similar to those in younger patients but irritability may be a predominant symptom. They may report their mood as being good but appear tense and unhappy and may even get weepy. Elation is uncommon. They may become disinhibited and display out of character behaviours that might be considered normal in younger adults, e.g. taking up online dating. Their spending also needs to be checked as older people may not have financial reserves or are vulnerable to unscrupulous salesman or traders.

Management of manic or hypomanic episodes is the same as for younger people – biologically with the use of antipsychotics and mood stabilizers and psychosocially with input from community psychiatric teams and social services, or hospital admission where appropriate (see Chapter 6).

Depression

Depression is very common in older people and prevalence rates vary depending upon the study, with ranges of 1.5%–10% in community studies. In 24-hour care settings, rates may be as high as 40%. In general hospital settings rates are also high, ranging

Box 15.2 Reflective exercise

Bereavements and loss are common in older people. What are the symptoms and phases of normal grieving and bereavement?
They may include the following:

- Emotional numbness
- Denial
- Yearning
- Anger
- Guilt
- Agitation
- Low mood and sadness
- Reflection and thinking
- Resolution and moving on (or letting go)

How does this differ from depression or adjustment disorder?

from 10% to 50%. Older women are more likely to be depressed than men.

The majority of older people with depressive symptoms are diagnosed and managed in primary care, but depression is under-detected. Diagnosis is difficult as the picture can be complicated by co-morbidity of depression with physical illnesses and as the symptoms of depression in older people are similar to those for physical disorders e.g. reduced appetite and/or energy, or disturbed sleep.

Psychological reactions to an illness may precipitate depression or the disorder itself may lead directly to depression e.g. hypercalcaemia or effects of tumours.

Chronic conditions, especially chronic pain and those that affect quality of life, can precipitate or worsen depression. Life-threatening situations and disabling conditions also increase the risk of depression. Patients with chronic pain are up to five times more likely to have depressive symptoms than those without. Indirect effects of the disorders may be the precipitating factors, for example restricted mobility may leave patients isolated or unable to participate in activities they found enjoyable.

Other risk factors for depression include previous depressive episodes or other mental health problems such as anxiety, and/or substance misuse. Some prescription medications may cause depression or lower mood as a side effect, e.g. steroids, beta blockers, benzodiazepines, calcium channel blockers, statins and acetyl cholinesterase inhibitors. Similar to dementia and delirium (see Chapter 12), depression also affects the course and outcome of physical disorders. Physical symptoms may be exacerbated by depression and depressed patients admitted to general hospital remain as inpatients longer on average and have reduced survival outcomes compared with those without psychiatric diagnoses (see Figure 15.1).

Older adults may not complain directly of low mood and may deny feeling depressed or sad. On presentation, they may complain of medical symptoms such as headache, backache or abdominal pains. They may complain about trouble sleeping and patients may present asking for sleeping tablets as a first sign (rather than complaining about being down or low in mood). The majority of patients with depression will not actually discuss their symptoms with professionals unless they are asked to do so directly.

The core biological symptoms as described in Chapter 6 apply to older patients too. Older patients may also worry and ruminate excessively, blame themselves without reason or have guilt about things they have done (or not done). These are often based around events in the past. They often feel that they

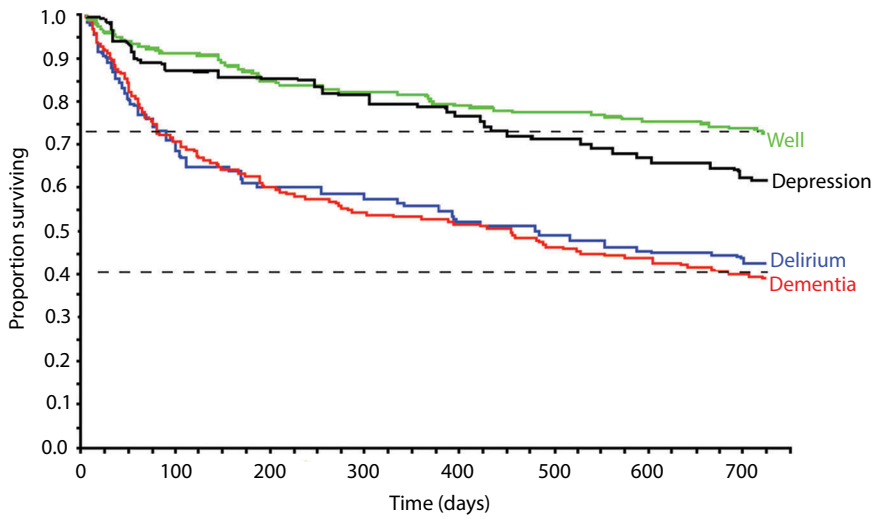

Figure 15.1 Survival after hip fracture in patients with no psychiatric diagnosis, depression, dementia and delirium. This is the same cohort as Figure 12.1. Those with depression had reduced survival rates (though not as severe as those with organic disorders). (Reprinted from *The Lancet*, Vol. 357, Nightingale S, Holmes J, Mason J, House A, Psychiatric illness and mortality after hip fracture, 1264–1265, Copyright 2001, with permission from Elsevier.)

are a burden, or feel worthless. Negative features may progress to psychotic intensity, with delusional ideas or the development of Cotard's syndrome (a nihilistic delusion characterized by a belief that the sufferer is dead). Auditory and derogatory hallucinations may also occur. A fuller list of symptoms of depression in older people is given in Table 15.1.

They may become preoccupied with death and mortality. It is essential to assess risk of suicide for all older people as an integral component of a comprehensive mental health assessment. Between 70% and 95% of older people who complete suicide have a mental health disorder, with depression being the commonest diagnosis in this group. Worldwide, suicide rates are highest in males over 70, though in the United Kingdom, the rates in this group have fallen (to 15 per 100,000). In 2012, the 80–84 age group had the United Kingdom's highest suicide rate, with over 20 per 100,000 people taking their own lives. Men are still three times more likely to kill themselves than women in this age group. Risk factors for suicide include male gender, social isolation, bereavement, chronic pain or disability, previous history of attempts at suicide, evidence of premeditation, expressions of hopelessness, sleep problems and misuse of alcohol. Most older adults who commit suicide will have seen their doctor within a few months of their death and a significant number in the week before, though they may not contact mental health services. Decline in mental health as part of the aging process should not be seen as inevitable by professionals or patients.

Social isolation and loneliness may also delay patients presenting to services. In a short primary care consultation, depression in older people can be easily overlooked if the focus is around medical symptoms. Depression should be considered if the patient is worried about physical illnesses or presents with somatization or hypochondriacal symptoms.

Pseudodementia

Depression in elderly people may present with cognitive impairments, such as poor concentration and associated memory lapses, so that dementia is

Table 15.1 Symptoms of depression in older adults

Biological	Psychological	Social
Sadness, low mood; diurnal variation of mood (typically worse in the AM)	Anxiety and worries, panicky episodes	Neglecting personal care (skipping meals, forgetting meds, neglecting personal hygiene)
Fatigue and lack of energy	Irritability	Lack of motivation for usual activities
Anhedonia: losing interest in hobbies or other pleasurable pastimes	Intense, pervasive sense of guilt	Loss of confidence
Loss of appetite; weight loss	Feelings of hopelessness or helplessness	Weepiness
Unexplained or aggravated aches and pains; worsening of other physical symptoms	Loss of self-worth (worries about being a burden to others, feeling useless, feelings of worthlessness, self-loathing)	Social withdrawal and isolation (reluctance to be with friends, engage in activities, or leave home)
Difficulty falling asleep or staying asleep; early morning awakening	Thoughts of suicide or a preoccupation with dying	Increased use of alcohol or other drugs
Oversleeping or daytime sleepiness	Fixation on death; suicidal thoughts or attempts	Changes in behaviour – out of character, aggressive, bizarre, withdrawal
Loss of sexual interest	Delusion ideas (including Cotard's syndrome)	Dwelling on events from the past; getting things out of proportion
Restlessness and agitation	Auditory hallucinations (or hallucinations in other modalities e.g. visual)	
Slowed movement and speech	Memory problems or complaints	

Table 15.2 Differences between depression and dementia in relation to cognitive symptoms

Depression	Dementia
Onset relatively quick (weeks/months)	Onset relatively slow (months/years)
Tends to be orientated for time and place	Confused and disoriented; becomes lost in familiar locations
Poor concentration	Reduced short-term memory
Language and motor skills slowed, but maintained	Language and motor skills are impaired
Frequent 'don't know' answers, little effort	Will try to answer questions
Notices or worries about memory problems	Doesn't notice memory problems or seem to care
Upset by errors	Not upset by errors; minimizes mistakes or blames age
Low in mood with biological symptoms, diurnal variation of mood	Normal or blunted affect

erroneously diagnosed. Depression may be a co-morbid disorder in patients with dementia, in up to 20% of sufferers, and makes cognitive deficits worse. This is an important differential diagnosis to consider especially in the elderly presenting with cognitive problems or changes in behaviour. Given that the patient with dementia may not be able to give a coherent account of their mood, observations such as psychomotor slowing, apathy, agitation, loss of interest, reduced appetite, sleep disturbance or other behavioural changes are important to note. The difference between cognitive changes in depression and dementia are given in Table 15.2.

Anxiety disorders

Primary anxiety disorders developing in older people as a new diagnosis are uncommon. The majority of cases are generalized anxiety disorders and most are relapses or exacerbations of a previous anxiety disorder. Specific phobias may last into old age, but phobias arising in old age tend to be agoraphobic, often precipitated by a traumatic event such as a fall or mugging. Panic disorder is not common but panic episodes may be under-reported and may mimic physical disorders such as cardiac problems or cerebrovascular events. Only about 10% of anxiety disorders in older people are diagnosed as panic disorder, obsessive-compulsive disorder (OCD) or post-traumatic stress disorder (PTSD).

Management of anxiety disorders in older people is the same as for younger adults (see Chapter 7). If medication is required, benzodiazepines should not be used except for symptom control in severe attacks. Any underlying physical disorder, e.g. thyroid disorder, should be treated too to minimize any symptoms they may causing.

Anxiety is also a common feature in dementia. Patients may not show their emotions directly but may exhibit physical signs such as pacing and restlessness, agitation, fidgeting, sleep problems, handwringing or other stereotyped movements.

Obsessive-compulsive disorder

First-time onset is rare in older adults. For those with previous problems, changes to coping skills and patterns secondary to ageing, physical co-morbidities or changes in social circumstances may lead to deterioration and presentation, as symptoms of OCD can fluctuate over time. Depression may present with obsessive ideas or compulsions, or make underlying symptoms much worse. Dementia, anxiety and frontal lobe lesions may also cause symptoms. Prevalence rates are lower than in younger groups, but may be present in up to 1% of older adults

Post-traumatic stress disorder

PTSD is often under-diagnosed in older people and, like depression, may present as medical or somatic symptoms. It is often associated with anxiety and depression. Symptoms may have been present for many years before presentation but the patient may not have complained about them or used non-medical

methods of coping, particularly alcohol. Dissociative symptoms are much less frequent than in younger people, and PTSD in older people is linked to an increased risk of dementia.

Intellectual disabilities

People with intellectual disabilities are living longer (although people with learning disabilities are over 50 times more likely to die before the age of 50 than the rest of the population) due to improved medical care; hence, the number of older people with intellectual disabilities is growing. They will have to manage growing old as well as their underlying disabilities.

Most older people with intellectual disabilities live with their parents who are themselves older adults (40% live with a parent over 60 and 33% live with a parent over 70 years old) and have the associated problems of growing older, including carer-related issues. The number of older people using intellectual disability services is expected to double over the next 15–20 years. All people with an intellectual disability are at a greater risk of developing dementia as they get older compared with the general population (from about 15% in the 65–75 age group to 70% in the 85–95 age group) and it is recognized that people with Down syndrome are highly likely to develop Alzheimer's disease. Older people with intellectual disabilities have a higher risk of psychiatric disorder, which is two to four times more common than for other older people. Assessment should be no different than that for a younger person with intellectual disabilities as discussed in Chapter 11. Changes in behaviour may indicate an underlying psychiatric disorder including dementia, delirium or depression. Minimizing sensory impairments in sight and hearing helps communication. Management is similar to that in younger age groups with intellectual disabilities or older adults with mental health disorders. Psychological treatment will need to be adapted to the appropriate developmental level. Care should be taken with medication, particularly antipsychotics if used, due to the effects on seizure thresholds and interactions with antiepileptic agents, as a quarter of patients with intellectual disabilities have epilepsy. It should also be noted that there are many transferrable skills between assessing and managing patients with intellectual disabilities and those with dementia.

Assessment in older patients

The principles of assessment of older people are similar to those in younger adults. Assessment is more fully covered in Chapter 3 but some areas need specific mention in relation to older adults.

Enquiries about support, such as visitors, relatives, social groups and home care visits are essential. The patient's accommodation and living arrangements need to be assessed and whether the patient is managing the home. The effects of physical problems may be major factors to consider, so mobility and the ability to manage stairs or get to the toilet need to be ascertained.

Mental state assessment

A careful consideration of the patient's appearance can be helpful in the assessement of their mental state. Food stains or dirty clothes may indicate poor self-care, possibly due to depression or organic disorders. Spectacles and hearing aids may indicate sensory impairment, sticks or frames problems with mobility or pain. Signs linked to physical disorder may aid diagnosis, such as tremor and a mask-like face in Parkinson's disease or facial palsy following stroke. If the patient has hearing problems, hearing loops or amplifiers may be helpful (but make sure the hearing aid works and has a live battery too). Take visual and hearing impairments into consideration when undertaking cognitive assessments too (plus other physical disorders) – it is hard to copy a diagram if you cannot see it in the first place, or if you are disabled by a stroke. During the interview the patient may look to the informant they have with them for support. If they do this frequently, it may be an indication of cognitive problems. A full assessment for evidence of psychotic symptoms should be undertaken as patients may have these but may not volunteer them unless asked directly. Risk assessment including thoughts about suicide is essential.

Investigations

A focused physical examination is required and appropriate investigations should be organized. The use of clinical rating scales should be considered. For patients with depression, the geriatric depression scale is a useful tool and is given in Table 15.3.

Table 15.3 Geriatric Depression Scale (15-item version) (GDS-15)

Choose the best answer for how you have felt over the past week			
1.	Are you basically satisfied with your life?	YES	NO
2.	Have you dropped many of your activities and interests?	YES	NO
3.	Do you feel that your life is empty?	YES	NO
4.	Do you often get bored?	YES	NO
5.	Are you in good spirits most of the time?	YES	NO
6.	Are you afraid that something bad is going to happen to you?	YES	NO
7.	Do you feel happy most of the time?	YES	NO
8.	Do you often feel helpless?	YES	NO
9.	Do you prefer to stay at home, rather than going out and doing new things?	YES	NO
10.	Do you feel you have more problems with memory than most?	YES	NO
11.	Do you think it is wonderful to be alive now?	YES	NO
12.	Do you feel pretty worthless the way you are now?	YES	NO
13.	Do you feel full of energy?	YES	NO
14.	Do you feel that your situation is hopeless?	YES	NO
15.	Do you think that most people are better off than you are?	YES	NO

Note: Answers in PINK shaded boxes indicate depression. In clinical practice, scores > 5 points suggest depression – a full assessment is indicated. Scores > 10 are almost always depression.

Settings

Assessment in the patient's normal residence or home is often better as patients are more likely to behave in their usual ways and more comfortable and relaxed in their own environment. You can check on living conditions, get access to their medications and look for signs not possible in the clinic setting. For example, is there adequate food in the house? Is it in date? Are there safety issues? Is there evidence of incontinence or poor hygiene? Is there evidence of alcohol misuse? In 24-hour care settings, the staff can be questioned and concordance with medication checked via drug charts, and also behaviour charts checked.

Management

General principles

The treatments for mental health disorders are the same for older adults as they are for other adults. The full range of biological, psychological and social treatments should be considered but may need modification. Any underlying co-morbidities, both physical and psychological, particularly dementia, need consideration. If the patient lacks capacity, then decisions should be taken around the patient's best interests. Readers are directed to the relevant chapters for detailed information on treatments and management on specific disorders, though pharmacological principles for older patients are covered in the next section.

Pharmacology

There are significant changes that occur in the body as ageing occur, which need to be considered when prescribing. The amount of water and muscle reduce and the proportion of fat increases. Thus, water-soluble drugs, such as lithium and alcohol, may have a smaller volume of distribution, and higher serum levels compared with younger people given the same dose. For drugs that are more lipohilic, such as benzodiazepines, the elimination half-life will be increased, so effects and side effects will last longer. Reduced renal function will reduce the rate of excretion of some drugs (e.g. lithium)

increasing their risk of toxicity. Monitoring estimated glomerular filtration rate (eGFR) is thus more important in older people. Similarly reduced hepatic function and hepatic blood flow may affect the rate of metabolism of some drugs, particularly those subject to a high 'first-pass' effect. Reductions in numbers of neurones and numbers of neurotransmitter receptors (e.g. dopamine and acetylcholine) will often increase sensitivity to drugs, resulting in greater susceptibility to adverse effects.

Although the medication regimes are generally the same as for younger people, the changes outlined above mean that starting doses and effective therapeutic doses of drugs tend to be lower. The general principle for the use of drugs in older people is to start at a low dose (often at 50% or less than that for younger people) and increase the dose slowly, depending upon the response. Some drugs are rarely used in older people because of the risks of side effects, particularly clozapine (older people seem to be very susceptible to problems with clozapine). Duration of medication is usually longer in older adults, particularly with antidepressants. Discussions around which drug to use, as well as the risks (including side effects) and benefits of each therapeutic option should be discussed with the patient. If they lack the capacity to decide, the decision about their treatment should be considered after a discussion with their carers in the best interest of the patients.

As many older people take multiple medications consideration of drug interactions is important. The positive psychiatric effects with any potential interactions either with medications or the co-morbid disorder need to be balanced, e.g. antipsychotic use in dementia, lithium in renal impairment and vice versa e.g. renally excreted drugs (especially thiazide diuretics and NSAIDS) on patients with lithium. Medication for psychiatric disorders should not be stopped suddenly or without appropriate psychiatric consultation unless there is an acute medical necessity.

Concordance should always be a consideration in older people, especially in those with cognitive problems or those who live alone, and may be a significant factor in any apparent lack of efficacy. Psychiatric disorders may cause poor concordance, e.g. lack of insight in schizophrenia or memory loss in dementia. The use of concordance aids – dossette boxes, blister packs, automated dispensers or even monitoring and/or administration by care staff may all enhance concordance. Liaison with pharmacy colleagues may be helpful here.

Graduates from other psychiatric services

With improvements in the care of people with enduring mental health problems, many patients move from adult to older age psychiatric care. The histories may be long and complex with greater susceptibility to the longer term side effects of their medication, so the dose required to maintain their mental health may need to be adjusted. They may no longer be able to tolerate some of the medications and will need changing onto different medications. Discussion of the risks and benefits of adjusting needs to be undertaken with the patient. Patients on lithium may require different mood stabilizers or need augmentation with anti-epileptic agents such as valproate or lamotrigine. For those with schizophrenia who have grown older, their schizophrenia is known to be an independent risk factor for cognitive impairment.

Mental Health and Mental Capacity Acts

Older people are still subject to the terms of the Mental Health Act 1983 and require the same conditions to be met in order to be detained under the Act. Its use should be considered where appropriate.

As capacity is time and decision specific, capacity for consent for admission needs to be considered – patients may understand and decline admission to a psychiatric hospital even with dementia. Patients willing to be admitted but lacking the capacity to consent for their admission cannot be admitted voluntarily, and need to be detained.

Capacity for decisions is frequently an issue in older people, especially those patients with cognitive impairment or a diagnosis of dementia. The principles of the Mental Capacity Act apply to older people in just the same way as to any other age group. While this Act is an English and Welsh statute, the principles are similar to other jurisdictions. Patients without the capacity to decide should be treated in their best interests, which should be determined on a case by case basis, in consultations with other professionals and carers. If a patient lacking capacity needs to be deprived of their liberty (for example a delirious patient trying to leave hospital or a patient with dementia who tries to leave permanent 24-hour

care), then a Deprivation of Liberty Safeguards order (known as a DoLS) authorization should be instituted. If the situation requires this to be done urgently, an urgent order may be put in place by the institution caring for the patient (this may be a care home or hospital). This lasts for up to 7 days (and may be renewed in certain circumstances for a further 7 days only), but a standard order must also be applied for. A standard DoLS authorization may last for up to 1 year, with rights of appeal. Standard authorizations are undertaken by trained DoLS assessors, usually social workers employed by local authorities.

Fuller details about the Mental Health Act and Mental Capacity Act and their use are given in Chapter 5.

Care settings and services

When planning management strategies including social interventions, the care setting needs consideration. Many older people may be living alone. Management should aim to keep patients in their preferred accommodation if at all possible with support as required. Liaison with primary care, e.g. community matrons and district nurses, and local social services teams, e.g. rehabilitation teams, is also essential. Input from home care services, including meal delivery or preparation, needs to be considered for those patients whose disorder affects their ability to look after themselves. For those patients who cannot remain at home, there is a full range of alternative accommodation, from supported living (where there may be a visiting warden or services) and sheltered accommodation (with on-site wardens and group areas) to 24-hour care (residential or nursing care). Patients from the hospital may require short-term placements in other care settings before returning home, particularly if there are co-morbid physical problems. Many of those staff looking after older people are not trained to high levels so when planning care, the qualifications and competencies of carers needs to be considered, especially in 24-hour care settings.

The majority of carers, however, are family members such as spouses, siblings or children. Caring for someone with mental health disorders can be difficult and carers need support as they have increased rates of mental health problems, particularly depressive disorders. Their needs warrant assessment. This is particularly the case where the carer may have a pre-existing mental health disorder, e.g. dementia.

Box 15.3 Reflective exercise

- Without the support of other services, a general practitioner (GP) or psychiatrist for older people will not be able to manage their patients optimally. You should have a working knowledge of what support is accessible to patients and their carers where you are practicing or are on placement.
- What services are available for older people with mental health disorders and their carers in your area?
- In primary care settings?
- In secondary (specialist community) or tertiary (specialist hospital) care?
- In general hospital settings?
- In the voluntary sector?
- From social services and other local authority organisations?
- What are the services available specifically for carers?
- What are the mechanisms and pathways for referral into these organisations?
- What are the procedures for safeguarding adults?

Without good carer support, care of the patient may break down leading to increased morbidity and admissions.

Elder abuse and safeguarding

As discussed in Chapter 3, those working with elderly patients and especially those with mental health disorders need to be aware of the possibility of elder abuse which is defined as 'A single or repeated act or lack of appropriate action, occurring within any relationship where there is an expectation of trust, which causes harm or distress to an older person.'

Summary

In this chapter, we have highlighted that psychiatric disorders in the elderly are common, and present differently compared with disorders in younger adults. When assessing older adults presenting with mental

health problems, the effects of physical disorders need to be considered, either directly (delirium and dementia – see Chapter 12) or indirectly as co-morbid conditions that precipitate or worsen disorders in older adults, in particular depression. Depression, suicide risk and alcohol use in older people should not be overlooked. Although treatment and management are similar to that in younger people, consideration of all the biological (including pharmacological changes), psychological and social factors, including the risk of elder abuse, is vital to ensure successful recovery and maintenance of quality of life.

Further reading

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CASE STUDY 15.1

A 79-year-old woman presents to her general practitioner asking for medication to help her sleep. She cannot get to sleep due to pain in her back. She denies feeling low but on direct enquiry she reveals a 6-month history of reduced energy, poor appetite and mild weight loss. She lives with her husband who has Alzheimer's disease. She has a history of breast cancer 10 years ago.

What diagnoses should you consider?

Although physical symptoms predominate, depression is a major differential diagnosis. Underlying physical illnesses, e.g. thyroid disease, an undiagnosed recurrence of breast cancer or another malignancy may have precipitated depression and should be excluded. The chronic back pain may be a precipitant, but may be made more unbearable by depression. Co-morbid depression and physical disorder should be considered. If there is no organic disease or disorder, depression is the most likely diagnosis. Risk factors for depression include her age, gender, poor sleep, chronic pain and her role as a carer.

CASE STUDY 15.2

An 84-year-old man is referred because he has told his general practitioner he can hear his neighbours talking about him and plotting to poison him by putting gas through his letter box at night. He reports that this has been occurring daily over the past 6 weeks. He has a diagnosis of schizophrenia, which is treated with a monthly depot injection at the surgery, though he forgets to come for his appointments. He has been stable for several years with no hallucinations. When you do a home visit, he is dishevelled and there is evidence that he has urinary incontinence.

What diagnoses should you consider?

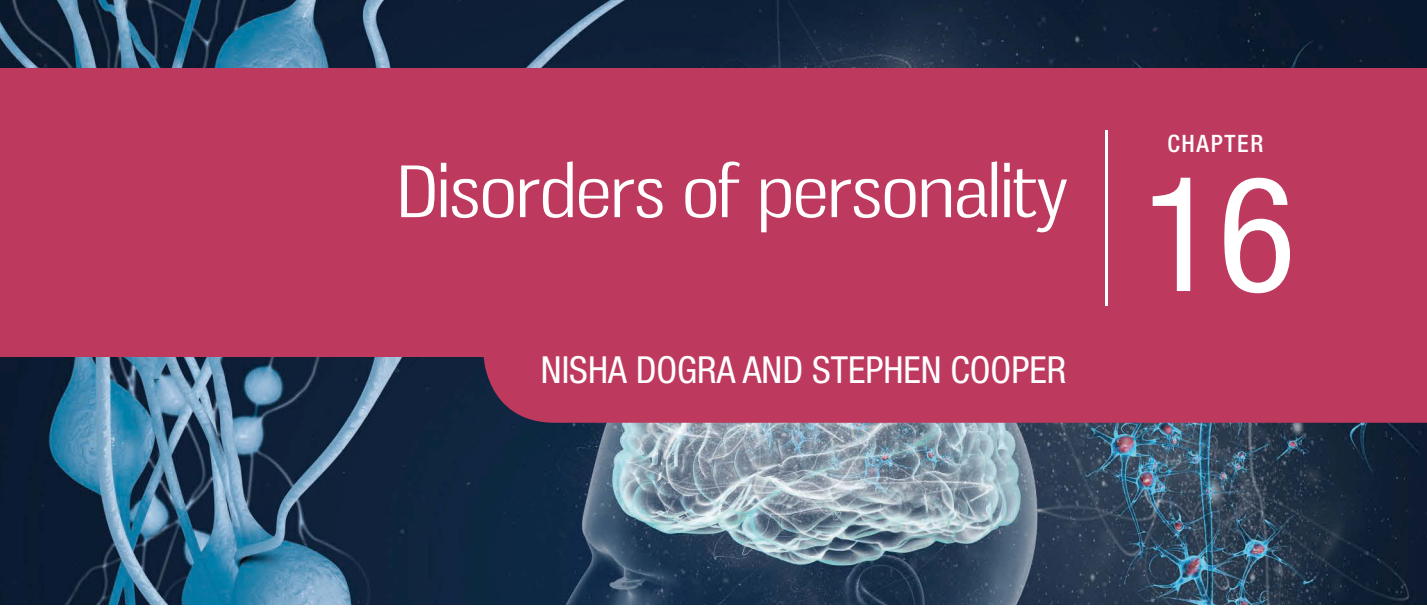
Again, organic disorders should be thought about. The apparent recent onset and incontinence point to a possible delirium, possibly due to a urinary tract infection. An underlying dementia may also be present, as he misses appointments. His changed mental state might indicate an acute deterioration and relapse of his underlying schizophrenia. The negative nature of his beliefs and poor self-care might also indicate depression.

CASE STUDY 15.3

You are asked to see a 75-year-old woman who is refusing to leave her home. Her husband reports this has been getting worse since they returned from living in Portugal a year ago after losing their retirement home in a property scam. He reports that she has become forgetful and confused at times. The patient says she doesn't enjoy life like she did in Portugal. During your visit, you notice a washing basket of empty brandy bottles.

What diagnoses should you consider?

Again, think about organic conditions first. The forgetfulness and confusion may indicate an underlying dementia. A thorough alcohol history should be taken (though the empty bottles may not be all hers). Her episodes of confusion might be intoxication, and her memory problems may be related to alcohol-related cognitive impairment. An alcohol-related physical disorder may also be precipitating her symptoms. Depression again should be considered, especially given recent life events. Agoraphobia is unlikely to present as a primary disorder and is more likely to be an anxiety symptom secondary to her depression. Her alcohol consumption may be a coping mechanism for her anxiety, but may be exacerbating her depression. A depressive episode may be responsible for her cognitive symptoms. She may also have had previous anxiety disorder, made worse by the social changes and adjustment problems related to the move.



Disorders of personality

CHAPTER 16

NISHA DOGRA AND STEPHEN COOPER

KEY CHAPTER FEATURES

- Definition and prevalence of personality disorders
- Characteristics of the subtypes and their presentation to services
- Impact of having a personality disorder
- Management of personality disorders

Introduction

Issues relating to different traits and types of personality, how these may be factors in increasing risk of mental illness and how they may modify its presentation have been discussed in Chapter 2. There is no clear dividing line between the point where particular features suggest someone has a particular personality trait and the point where similar features suggest a diagnosis of personality disorder. Indeed, some would argue that the concept of a disorder of personality is inappropriate for something that may perhaps be seen as relating to traits on a continuum from mild to severe. There is also considerable debate about how far 'treatment' is effective and indeed whether it is appropriate to attempt to change what is a person's essential personality. Indeed, in Soviet Russia in the mid-twentieth century, those with dissident ideas, who disagreed with the communist state, were sometimes described by the authorities as suffering from some form of mental disturbance, which could range from personality disturbance to mental illness, and were subsequently placed in mental institutions. The argument has become political in more ways than one. Successive UK government Home Secretaries have sought to make responsibility for people with 'dangerous and psychopathic personality disorders' the domain of mental health services. They wish us, as mental health professionals, to assess risk, provide treatment and protect the public from such individuals. In the mind of the general public, it is often the disturbed, impulsive, aggressive psychopath who seems to be *mentally ill* rather than those we as doctors may regard as much more ill.

Box 16.1 ICD classification of personality disorders

F60 Specific personality disorders

Paranoid – includes previous categories such as sensitive and querulant personality

Schizoid – not the same as schizotypal disorder (related to schizophrenia)

Dissocial – more used term is antisocial, psychopathic or sociopath

Emotionally unstable – either impulsive or borderline type

Histrionic (was hysterical)

Anankastic (was obsessive)

Anxious (also called avoidant)

Dependent (was asthenic, inadequate or passive)

Other specific personality disorders, including narcissistic personality disorder

F61 Mixed or other personality disorders

Thus, we cannot avoid having to face the issues involved. Perhaps the most useful way to try to define the point where a personality trait becomes a disorder is as follows:

> *It is where maladaptive patterns of behaviour, modes of thinking and relating to oneself, the environment and other people result in impairment of social functioning for the individual or others affected by their behaviour.*

In general, the features defining an individual's personality disorder are present during adolescence and are persistent throughout their life. *International Classification of Diseases*, 10th Revision (ICD-10) defines 10 main personality disorders (Box 16.1) as well as a category of enduring personality changes, where features do not easily fit one of the 10 specified categories.

Why is this relevant for you?

As students you will only encounter a few people with clearly established personality disorder, but some of those encounters may be the most challenging that you experience. Knowing something about the types of disorders can help you understand how different types of people may access help and present to services and how to ensure that the care you provide is professional despite the challenges. It may also help you in understanding their perspectives and being more objective if you are aware of the potential problems. It is also important because there can be a tendency to label people by their major trait and overlook other possibilities on the basis that they are just anxious or suchlike.

Assessment and diagnosis of personality disorder

Diagnosis of any particular personality disorder needs to be made carefully. First, it is often difficult, as individuals sometimes have features present in more than one type of disorder. It also depends in part on gaining a good account of an individual's general demeanour and behaviour from an informant (family member, friend), as one cannot rely solely on the person's own account. (We do not always see ourselves as others see us.) Finally, given the variability in how any of us behaves over time, it is important to be able to establish that the features being used to make the diagnosis are consistently present. The evidence for having a personality disorder is that the person consistently displays evidence of behaviours, cognitions and emotions that cause problems for him or her and others around them. These difficulties are evident in a number of different situations and appear to have persisted throughout life and begun in childhood.

A diagnosis of personality disorder may coexist with the diagnosis of another mental disorder. Sometimes other disorders are secondary to the personality disorder, arising because the personality disorder results in conflicts between the individual and the world around them, leading to, for example, depression or anxiety. Sometimes the disorders are coincidental. As with personality traits, it is best to view a diagnosis of personality disorder along a separate axis to the diagnosis of another mental disorder and to list one as the main diagnosis and the other as a secondary diagnosis rather than as another differential diagnosis.

Young people under the age of 18 years cannot be diagnosed as having a personality disorder, and the term emerging personality disorder may be used. However, given that adolescence is a stage of life when

young people are establishing a sense of who they are, some of the behaviours, especially those of the emotionally unstable type, may be transient.

Types of personality disorders

As outlined in Chapter 2, and shown in Box 16.1, ICD-10 recognizes several personality disorders. It is perhaps simplest to classify people into one of three broader categories as follows:

- Odd/eccentric types (paranoid, schizoid)
- Emotional, erratic types (dissocial, emotionally unstable, histrionic)
- Anxious/fearful types (anankastic, anxious, dependent)

It is perhaps worth being aware that in the next 2–3 years there are likely to be fundamental changes in how we classify and describe different presentations of people with personality disorder. While ICD-10 outlines what may appear to be specific disorders, the reality is that it can be difficult to clearly determine which category a particular individual falls within, nevermind whether or not their difficulties fall sufficiently outside the normal range of functioning to be described as a personality disorder. There are overlaps between each category in terms of certain personality traits and behaviours. In the future it is likely that an essential set of features will be regarded as a key to making a diagnosis, and the individual will then be regarded as having these to a mild, moderate or severe degree based largely around impairment of social, occupational and interpersonal functioning. Further description will then involve ascribing one or more of five different 'domain' personality traits to give more specific description of the presenting behaviours. Thus, for example, the problem of a crossover of features between say an anankastic and an anxious personality will be avoided.

The essential diagnostic features are described in Box 16.2. Even within the current ICD-10 system there are core features required for a diagnosis of any individual type of personality disorder.

We will not describe all of the different personality disorders in detail but talk about them generally in their clusters: Cluster A (odd eccentric types), Cluster B (emotional, erratic types) and Cluster C (anxious fearful types). However, two Cluster B types, dissocial/antisocial and emotionally unstable personality disorders, merit some detail given their impact on the community and health services.

Box 16.2 Essential features of personality disorders

- A pervasive disturbance in how an individual experiences and thinks about the self, others and the world, manifested in maladaptive patterns of cognition, emotional experience, emotional expression and behaviour.
- The maladaptive patterns are relatively inflexible and are associated with significant problems in psychosocial functioning that are particularly evident in interpersonal relationships.
- The disturbance is manifest across a range of personal and social situations (i.e. is not limited to specific relationships or situations).
- The disturbance is relatively stable over time and is of long duration. Most commonly, personality disorder has its first manifestations in childhood and is clearly evident in adolescence.

(These are as proposed for ICD-11 and taken from Tyrer et al. (2015). This paper is useful for further reading.)

How common are personality disorders and what are their features?

A recent World Health Organization (WHO) survey across 13 countries (developed and less developed) suggested a population prevalence for all types of personality disorder of 6.1% (Huang et al., 2009), which is within the range found in previous smaller, within-country studies. However, though the questionnaires used are internationally accepted, it is still not clear if they are equally applicable across widely varying sociocultural situations. Overall, most studies find those with a personality disorder to be more likely to be male and unemployed and also more likely to suffer from other forms of mental disorder than the general population, but other associations vary with type of disorder. Many other people have personality traits that increase their risk of developing mental health problems, but not to the extent of having a personality

disorder that of itself is likely to cause significant impairment of ability to function in life.

The aetiology of personality disorders is unclear, but it is likely that as with other types of mental disorders the aetiology is multifactorial. Having a predisposition to develop an antisocial disorder is more likely to materialize in the context of being raised in an aggressive, violent and abusive background. Being 'mollycoddled' and having anxiety traits may make avoidant personality disorder more likely. Other predictive factors for a diagnosis of personality disorder in adulthood include maternal reports of anxiety or depressive symptoms and conduct difficulties.

Before we discuss the potential impact of having a personality disorder, we will discuss the broad characteristics of the key groups. Dissocial personality disorder is more frequently found in men, whereas emotionally unstable personality disorder (EUPD) is higher in women in many surveys. As well as biological differences, this may also reflect societal expectations and gender roles.

Odd/eccentric types (Cluster A)

People with paranoid personality are by their nature paranoid; they are suspicious and mistrustful of others often believing themselves to have been unfairly treated or maligned. They tend to be very negative in their views about the external world, and when these negative ideas prove themselves to be true, they feel justified in their original views. They are unable to see how their own behaviour may lead to the pattern of events. There is little, if any, evidence to support their suspicions about others. They are more likely to be involved in legal actions as recourse for their perceived injustices, as reasoning with them can prove to be difficult as they do not trust others. They tend to have difficulty developing close and warm relationships.

People with schizoid personality are loners who struggle to make appropriate social relationships. They tend to be emotionally detached and have little involvement with others and the external world in socially acceptable ways.

Schizotypal personality, which is classified under schizophrenia rather than personality disorder, is different in that not only are these people socially and emotionally detached (as are those with schizoid personality) but also they can often show oddities of thought, perception and communication. Some individuals who go on to develop schizophrenia are thought to have this type of personality, although most with this disorder do not develop schizophrenia.

It is important, as with schizophrenia, not to confuse this type of disorder as 'split personality'. These people do not have many different types of personality but are socially withdrawn and isolated.

Emotional, erratic types (Cluster B)

The two major ones are discussed in some depth, as they can present significant challenges to healthcare providers. Also both tend to stabilize and/or diminish with age.

Dissocial/antisocial personality disorder

This is the disorder that is most often in the news headlines, because it is more likely to lead to violent or extreme behaviour. The most severe form of this is psychopathy which makes up about 20% of this group. The main features are listed in Box 16.3 and the diagnosis is made more often for men than women.

The history of many of these people may reveal a number of features that should make you consider the possibility of such a diagnosis. Typical are bullying

Box 16.3 Features of dissocial personality disorder

- More common in males
- Evidence of conduct disorder in childhood (required for *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) diagnosis of antisocial personality disorder but not for ICD-10 dissocial personality disorder)
- Callous unconcern for the feelings of others and no remorse for behaviour
- Gross and persistent irresponsibility and disregard for social rules, norms and obligations
- Unable to form stable or enduring relationships although has no difficulty in establishing them
- Low tolerance to frustration and easily becomes angry or aggressive
- Incapacity to experience guilt or profit from experience
- Marked proneness to blame others for difficulties
- Often behaves impulsively
- May have a criminal record

behaviour at school, leaving school early, convictions for assault, poor employment record, substance misuse, cruelty to animals and fire-setting.

In recent years, there has been increased interest in looking for underlying biological factors in these individuals. For example, there is evidence that impulsivity in some circumstances may relate in part to disturbances of serotonin function. Some functional magnetic resonance imaging (MRI) studies suggest possible areas of brain dysfunction. However, most theories relating to all forms of personality disorder focus on psychological and developmental factors that are beyond the scope of this book.

As would be expected from the clinical features, they are at increased risk of engaging in criminality and violence. They are also at increased risk of death from suicide, accidents and violence.

A diagnosis of dissocial personality disorder does not absolve an individual of responsibility for any criminal acts that they commit. The criminal justice system (police, prisons, courts and probation service) frequently manages such individuals (although they may not necessarily be labelled as having this disorder). It is important to maintain clear boundaries and rules when treating these patients. Any associated criminal offending is often best managed by the criminal justice system rather than mental health services.

Emotionally unstable personality disorder

Borderline personality disorder is a commonly used term for EUPD. It is a condition characterized by impulsive actions, rapidly shifting moods and chaotic relationships. There are two types (impulsive and borderline type) and in both there is:

1. A marked tendency to act impulsively without considering the consequences of these actions, for example, engaging in unprotected sex or substance abuse
2. An inability to plan ahead, coupled with a lack of self-control and outbursts of intense anger, which can lead to violence and other extreme behaviour, especially if impulsive acts are challenged or prevented by people around them

The impulsive type is characterized by emotional instability and an inability to control impulses, with episodes of threatening behaviour and violence occurring particularly in response to criticism by others. Women are more likely to receive a diagnosis of this disorder than men.

In addition, people with this type of personality disorder may experience severe doubts about their self-image, aims and sexual preferences which cause upset and distress. The main features are a chronic feeling of emotional emptiness, emotional instability, a pattern of forming very intense but usually unstable relationships, outbursts of anger, acts of self-harm and threats of suicide. When these individuals do experience emotions it is often in a very intense way, but this rapidly turns to an inability to cope with these emotions, leading to some of the features above. They are liable to become involved in intense but unstable relationships that can cause them continual emotional crises which they will endure to avoid being abandoned. Pseudo-hallucinations may be described by some in states of acute crisis. It is more common in females, and a history of sexual abuse is often elicited. Completed suicide occurs in around 8%–10% of individuals with this disorder, and acts of self-mutilation (e.g. cutting or burning), suicide threats and attempts are very common. Recurrent job losses, interrupted education and broken marriages are common.

VIDEO 16.1

"I just want some help…": borderline personality traits – https://vimeo.com/20662274

Co-morbidity with mood disorders, substance misuse, eating disorders (usually bulimia) and post-traumatic stress disorder is common. Very stressful or chaotic childhoods are commonly reported (e.g. physical and sexual abuse, neglect, hostile conflict and early parental loss or separation). For this reason, multiaxial classification systems can be helpful in designing interventions. This often means that the features leading to the presentation are longstanding, so long-term therapeutic interventions may be helpful.

Some of these individuals present frequently to Emergency Departments, to their general practitioners (GPs) and to crisis teams in the community. There are often strong pressures for their admission to hospital, but this is rarely helpful in the long term as it leads to increased dependence on others and further failure to develop coping mechanisms.

The terminology 'borderline' stems from the idea that borderline disorder lay somewhere between the

older notions of neurotic disorders and a psychosis. This is not the case, hence the move towards EUPD.

Anxious fearful types (Cluster C)

Cluster C has different types of personality disorder within it, with three types being most prominent. Individuals with avoidant personality disorder worry about doing anything new for fear of being rejected for failing. They are desperate for acceptance and approval, yet at the same time can be crippled by fear of rejection or being disappointed. They actively seek relationships and want them. Thus, an added component is frustration with the fact that they are unable to make successful relationships or be the person they might actually want to be.

Individuals with dependent personality type have difficulty in taking any responsibility for themselves and almost need to be given direction. They lack any ability to fend for themselves. A point worth bearing in mind is that in some cultural contexts (and even the broader UK context less than a generation ago), dependency by women on men is actively encouraged, as 'cultural norms' deemed women unable to make decisions regarding their own future. A trait is not a personality type if it is within a cultural norm.

An individual with obsessive–compulsive personality is preoccupied with order and control. They often seek perfection and thereby are great non-finishers, as the end product is never judged to be good enough. However, as they take their responsibilities seriously they can become very stressed, especially as they are generally indecisive and inflexible. People with obsessive–compulsive disorder (OCD) differ from those with obsessive–compulsive personality disorder in that those with OCD experience anxiety related to specific preoccupations which are perceived as threatening. They then carry out rituals in an attempt to dispel that threat. Unlike individuals with OCD, those with obsessive–compulsive personality generally do not have repeated, unwanted and/or ritualistic behaviour.

Impact of having a personality disorder

Clearly, from the above descriptions it will be evident that some disorders have more obvious social impact than others. However, personality disorders do share some commonalities and these relate to how the individual with a disorder relates to others and the external world. Many people with a personality disorder do not recognize that they do indeed have a personality disorder, although this may not be the case when the disorder is used to absolve responsibility for one's own behaviour. Different disorders carry different degrees of risk, but people with personality disorders are at a high risk of undertaking behaviours that do not conform to expected societal norms. They may be particularly vulnerable to developing specific psychiatric problems, as they do not tend to have a range of coping skills and at times of stress may be even less adaptable than usual. They may revert to their key trait and become even less functional. Developing and maintaining relationships may be particularly challenging and not just for those with dependent personality disorder or emotionally unstable personality disorder (EUPD). If an individual is an odd, eccentric person and is also suspicious of others, it becomes clear that relationships are likely to be strained, as they may well be in someone who constantly seeks reassurance. In particular, relationships with healthcare providers can be especially fraught. This may be true for all the personality types. Those in the odd/eccentric cluster may be frustrated by the needs of others to get them to access help, when this is not something they feel they need. The more dramatic personality types can frustrate healthcare providers by their behaviours and recklessness. The anxious types can be demanding on health services and yet never satisfied with the responses they receive. It can also be an issue that individuals with personality disorders do not do as well as they might expect and then become frustrated. An example of this may be the obsessive type of person who becomes stuck in tasks but never actually finishes them because of circularity in their thinking. However, with these negative sequelae the individual is likely to become more entrenched and thereby exacerbate the problem.

Not only are individuals with personality disorder at risk of developing mental health problems, but their personality can have a detrimental impact on their ability to parent and thereby be a factor in their children's mental health. A parent with an anxious personality type may because of their own fears restrict the child's exposure to the external world and thereby instil in the child a sense of fear rather than sense of mastery. For reasons that relate to their personality, parents with personality disorder may be inconsistent, detached, over-intrusive, over-permissive and/or inflexible. All of these may be important aetiological factors in whether children develop problems or not.

Individuals with a personality disorder may be more likely to use a more limited range of defence mechanisms than others. The defence mechanism of projection (attributing one's own feelings or thoughts to others) is typical of Cluster A type personalities and type B when under pressure. Splitting is typical of EUPD and often used with clinical staff. Acting out (expressing an internal impulse through an external behaviour) is characteristic of type B personality types.

Management of personality disorders

Most people with personality disorders, especially dissocial personality and EUPD, are often challenging to manage and can evoke strong negative feelings in others (for example, through repeated self-harm, or through aggression). Recognizing these feelings (countertransference) is important and helps in establishing and maintaining a professional relationship to ensure that they still receive any care they require. Despite many efforts, treatment for personality disorders is often unsatisfactory.

The most appropriate approaches are generally forms of psychological therapies. A few 'therapeutic community' treatment programmes have been successful for a proportion of patients. The aim of these is to provide a supportive and trusting environment in which the patient can learn better ways to cope. However, these all require a considerable commitment from the patient – something which is often lacking in the above two types of more severe disorders. It is also worth noting that psychological therapies for disorders such as depression have been found to be less successful if a patient has a coexistent personality disorder.

Evidence for the effectiveness of psychotropic medications is poor. Where depression of mood is a significant symptom a selective serotonin reuptake inhibitor (SSRI) may be worthwhile, but effectiveness tends to be less than in patients without coexistent personality disorder. Trials of SSRIs, second generation antipsychotics and mood stabilizers have been carried out for impulsivity and of mood stabilizers for the emotional instability seen with EUPD, but results are inconsistent and these treatments cannot be recommended on a routine basis.

Summary

There are three major subtypes of personality disorders and two that can present with significant challenges to health services. Although personality disorders have a prevalence rate of about 6%, the traits are more common and may still lead to problems. As future doctors, it is helpful to be aware of the potential presence of a disorder, as it may help in how you relate to the patients to provide them the care that they require.

Further reading

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McMain S, Pos AE (2007). Advances in psychotherapy of personality disorders: A research update. *Current Psychiatry Reports,* 9: 46–52.

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CASE STUDY 16.1

A 32-year-old man presents to casualty having got into a fight with a neighbour. He accused the neighbour of spying on him. He is known in the neighbourhood as being someone who keeps to himself but every now and again there are incidents when altercations occur.

What would you do as a doctor working in the Emergency Department? What might your differential diagnosis be?

The first action needs to be to treat any injury that might have occurred in the fight. The man is likely to be argumentative and suspicious, so it is important to be aware of this when treating him. Once the injury has been treated, it is important to take a history and see if you can establish what might be making him suspicious at this point. Before a diagnosis of personality disorder is considered, you need to ensure that a psychotic illness has been excluded.

CHAPTER

17 Commonly used psychological treatments

IAN COLLINGS AND NISHA DOGRA

KEY CHAPTER FEATURES

- Common components of psychological treatments
- Features of counselling, behavioural therapy, cognitive behavioural therapy, family therapy, group therapy, interpersonal therapy, psychodynamic psychotherapy and psycho-education
- Clinical indications for these types of therapy

Introduction

Psychiatrists do not just prescribe pills! We have a further armoury to call on, that of the psychological therapies. These therapies usually involve an interaction between an individual or group of individuals and a therapist or therapists, although, increasingly, psychological treatments are being delivered with limited therapist input. Examples include guided self-help, where an individual is given written or computer materials to follow with only limited therapist contact, or pure self-help, where the individual does not interact with a therapist. Over the years different types of psychological treatment have been developed, all of which share some important features as summarized by Jerome Frank (Box 17.1). In this chapter only the commoner forms of psychological treatment will be considered. In addition to these therapies psycho-education has been included, which although not a psychotherapy in its purest form is an increasingly important type of therapy, which is widely delivered to patients including those with schizophrenia and bipolar disorder.

All of the therapies discussed in this chapter can be used in any age group dependent on development levels. At times, therapies will require adaptation for use with particular groups or not be suitable for them (e.g. very young children and individuals with severe learning disabilities).

Psychotherapies may be as much about helping someone to be able to manage the life they have as about changing aspects of it. There is also often an impression that 'talking therapies' do not carry any

Box 17.1 Common features of psychological therapies

- Listening and talking
- Release of emotion
- Giving information
- Providing a rationale
- Restoration of morale
- Suggestion
- Guidance and advice
- Therapeutic relationship

risk, after all no potentially dangerous medicines are used. However, therapy that is poorly carried out can be damaging. Therapy that is mistimed may lead to the patient suffering more if their coping mechanisms (albeit that they may be maladaptive) are stripped away but they are left with nothing to replace them.

Psychodynamic therapies

Although there are various types of psychodynamic treatment, it is commonly agreed that many were developed out of the original work of Sigmund Freud and others. Freud developed a technique known as psychoanalysis, which often took place over many years, with an individual meeting their analyst five or more times a week. This form of psychotherapy is often referred to in the media and popular culture. Think of the patient lying on the couch, the caricatured therapist analysing their dreams or shouting out random words and asking the patient to respond with the first word that comes to mind. These techniques of dream analysis and free association were classic components of Freud's psychoanalysis. This form of therapy is still used today, although less commonly than in the past. Psychodynamic therapies can be a difficult concept for many students, as the processes taking place are often less transparent than in other therapies. It can also appear to be rather unstructured and random. It is also more dependent than some of the other therapies on the relationship between the patient and therapist. Some students struggle to identify how the therapist evaluates change. As with other interventions there should over time be a reduction in symptoms or their impact on the individual. It might be useful to try and think about the principles that have arisen out of psychotherapy that are applied in everyday clinical practice, such as taking a patient-centred approach and understanding the patient perspective better through exploring what meaning the issues have for them. It may also be useful for you to think at a more basic level about how unresolved conflict with a friend or partner leaves you feeling and how it impacts on you the next time the same situation arises. The approaches are based on the work of many people not just Freud's original work.

EXERCISE 17.1

Do this with your colleagues if possible so you can discuss your responses.

What frightens you? Think about something that makes you feel scared or anxious. It might be a spider or a rat. How do you think you might get over that fear? Talk it through with your colleagues.

You probably know how to explain a physical treatment to a patient, such as a medication or surgical intervention. Think about how you might explain a psychological treatment to a patient. What words would you use? How would you explain its mode of action?

Answer

Think of the psychology you learnt in your first or second year at medical school. Remember Pavlov and his dogs? Every time a bell was rung the dogs would salivate because originally the bell was rung at the same time as the dogs got food. The food was called the unconditioned stimulus and the salivation the unconditioned response. The bell was rung so many times at the same time as the food was given that it became the conditioned stimulus. Removing the food and ringing the bell, Pavlov noticed that the bell still produced the salivation. However, continued ringing of the bell without the food resulted in the response dying out; this is called extinction.

Now think of that thing that scares you. Why does it scare you? Have you had a bad experience in the past? Maybe if you continue to face the object of the anxiety either all at once (flooding) or in steps (systematic desensitization) the anxiety associated with it will eventually die out. These are two important psychological treatments; both remain in use today although the second is more commonly used.

Brief forms of psychodynamic psychotherapy are now more widely used and usually take the form of once weekly sessions (or fewer) with a psychotherapist. The therapist is aiming to help the individual explore and understand their unconscious motivation. Various defences are used to deal with intrapsychic conflict, and the aim is to give the individual insight into their presentation as discussed in Chapter 3. Some of these defences are reiterated for convenience below (Box 17.2). An unskilled therapist can let transference (unconscious redirection of feelings onto the therapist from the patient) and countertransference (unconscious redirection of feelings onto the patient from the therapist) issues affect the therapy in a detrimental way; care should be taken to prevent this from happening.

Psychodynamic therapy tends to focus on unconscious processes as they are manifested in a person's present behaviour. The goals of psychodynamic therapy are to raise the patient's self-awareness and understanding of how events in the past may be influencing current behaviour. In this way the patient is able to explore past unresolved conflict (which is what leads to the current behaviour that is causing them problems or symptoms) such as dysfunctional relationships, unresolved feelings. In doing so, the current problems can be better understood and enable more effective change. For example, a female who may have had a difficult relationship with her father may have a tendency to enter relationships with males that lead her to feeling bad about herself and view this as 'normal'. Through therapy she may gain insight that these relationships have come about because she has unresolved issues regarding her father. Although this is a fairly simplistic example, it indicates the way that psychotherapy tries to work. Through therapy the woman may recognize the defence mechanism she uses to try and deal with the internal conflicts that the relationship with her father has left.

The main forms of psychodynamic treatment now last from around 12 sessions through to 40 sessions, but for some patients the process is much longer.

Brief psychotherapy is felt to be most appropriately suited to highly motivated individuals of above average intelligence who are psychologically minded. There is some evidence that brief psychodynamic psychotherapies can be helpful, but the evidence base is far less developed than that of other brief psychotherapies, such as cognitive behavioural therapy (CBT) or family therapy.

Box 17.2 Defence mechanisms

Repression: Pushing away of unacceptable ideas or thoughts.

Denial: Denying external reality of unwanted information.

Displacement: Uncomfortable emotions or thoughts are moved from a bad object to a more acceptable one.

Projection: Unacceptable ideas and thoughts are transferred on to another person.

Regression: Moving to a lower level of complexity when under stress.

Reaction formation: Taking the opposite attitude to oppressed wish.

Rationalization: Explanation of things in a logical or ethical way.

Sublimation: Creative activities which are motivated and driven from sexual instincts and drives.

Identification: Attributes of others are taken on to oneself.

Cognitive behavioural therapy

There are various forms of CBT. Aaron Beck is considered to be the originator of cognitive therapy. He developed a therapy for depression, arguing that individuals develop basic or core beliefs/schemas from early experiences, for example, feelings that they are not good enough; perhaps they experienced harsh paternal criticism, or considered people to be judgemental. This leads to conditional dysfunctional assumptions, for example, 'If I get anything wrong I will be punished'. Many negative events lead to negative automatic thoughts, for example, 'It is my fault. I'm a failure; things will never go right for me', which then result in the development of depressive symptoms. It is argued that these negative thoughts maintain the depression and a vicious cycle is established. The main function of therapy is to break this cycle by getting individuals to recognize how their thoughts and feelings impact on each other. In cognitive therapy, it is common to use dysfunctional thought records to challenge these views. Individuals are asked

to rate their emotions, what they were thinking about at the time and the thoughts that were running through their mind, i.e. the negative automatic thoughts. They are asked to challenge these automatic thoughts with alternative, more positive views and then rate the outcome according to how far the individual now believes the original thoughts. By continually doing this, it has been shown that individuals are able to challenge their thoughts and feel brighter as a result.

This cognitive approach is often coupled with a behavioural approach. The key behavioural feature of depression is reduction in usual activity, often as a result of loss of interest, motivation and pleasure. Behavioural techniques encourage an individual to re-engage with activities, often referred to in therapeutic terms as 'behavioural reactivation'. This is often done by encouraging individuals to keep a weekly activity schedule and list exactly what they do, including rating how much pleasure they gain from it and how much of an achievement that was. This can also be used to help individuals plan what they are doing and to set goals that are achievable for individuals.

In addition to CBT for depression, it has been developed and used for a wide variety of other disorders, including most anxiety disorders, stress-related disorders, eating disorders and somatoform disorders. It has also been used in the treatment of schizophrenia in an attempt to help individuals deal with hallucinations and delusions, although the evidence for this is less convincing at present than for CBT for depression or anxiety disorders. Of particular research interest during recent times is the use of CBT to help patients manage symptoms of chronic physical disease. Research suggests that CBT can help patients deal with chronic pain and life-altering diagnoses such as human immunodeficiency virus (HIV) infection and cancer.

Trauma-focused CBT is a specific treatment used for the management of post-traumatic stress disorder. This involves an individual focusing on the trauma. For example, individuals are often asked to develop a detailed present tense account of exactly what they have been through, make a recording of this and listen to it over and over again. It is believed that individuals are helped by the repeated exposure to what happened, which allows the mind to habituate to the experience.

Mindfulness-based cognitive therapy (MBCT) is a psychological therapy that was first developed in the United States primarily to treat depression. The therapy has components of CBT in addition to elements that are derived from Buddhist meditation. In addition to CBT activities, the mindfulness component involves breathing exercises that enables the person to focus the mind on the here and now and attempt to push any distressing thought or emotions away. MBCT is increasingly recognized as a useful treatment for many mental health problems including anxiety disorders and depression. MBCT can also be a useful way to manage stress. If you are preparing for exams and reading this book, maybe you should give it a go. There are very useful websites or apps that can guide you step by step through MBCT.

Eye movement desensitation and reprocessing

This treatment involves an individual with post-traumatic stress disorder focusing on the worst picture of the trauma, coupling that with a thought, for example, 'I am out of control' and their feelings at the time, for example, anxiety and the part of the body where the anxiety is felt, for example, in the pit of the stomach. While holding these four separate things together, bilateral stimulation, for example, through asking the patient to follow the fingers of the therapist from side to side or alternating hand tapping, the individual is asked to allow their mind to go where it wants to, i.e. a form of free association. It is not known how eye movement desensitization and reprocessing works, but randomized controlled trials have shown that it is an effective treatment for post-traumatic stress disorder (The National Institute for Health and Care Excellence [NICE] guidelines: https://www.nice.org.uk). Some individuals argue that it works by a similar mechanism to trauma-focused CBT, whereas other individuals believe that there is a different as yet unknown mechanism.

Other forms of behavioural therapy

Relaxation training

This is a useful form of therapy for people with stress or anxiety disorders. Here patients are asked to use techniques such as progressive muscle relaxation,

where the individual moves through different muscle groups tensing and relaxing them, or guided imagery, when at times of stress or anxiety the individual learns to take themselves off into a situation they find relaxing, such as walking in a meadow or alongside a stream on a warm summer's day.

Systematic desensitization

In the treatment of phobic anxiety disorders, systematic desensitization is often used. In this an individual is gradually exposed to more stress-inducing situations on a hierarchy they have developed in conjunction with the therapist regarding their phobia. The following list shows a graded hierarchy for an individual with a fear of spiders:

1. Discussion of a small spider
2. Picture of a small spider
3. Picture of a bigger spider
4. Film of a small spider
5. Film of several spiders
6. Real life exposure to a small spider in a jar
7. Exposure to spider out of jar
8. Exposure to bigger spider in jar
9. Exposure to bigger spider outside jar
10. Visit to zoo to see spiders through window
11. Entry into spider exhibit within zoo
12. Progress to contact with spiders

The aim of a graded desensitization programme is to gradually move through the steps, asking individuals to rate their level of anxiety at each step, often using diaries similar to those described earlier for depression. As the levels of anxiety reduce, i.e. the individual habituates, they are asked to move up to the next level of the hierarchy. This has been shown to be a very effective treatment of phobic disorders if individuals are able to fully engage with it.

Exposure with response prevention

In this type of therapy, which is particularly used for obsessive–compulsive disorder (OCD), the patient is asked to think of a hierarchy of situations that may fuel a particular ritual and expose themselves in a controlled way without engaging in the ritualistic behaviour. It is anticipated that if the patient can hold off engaging in the ritual for upwards of an hour the anxiety and drive to do it will eventually habituate and die out.

Interpersonal therapy

Interpersonal therapy was developed to treat individuals with depression but is now also used for the treatment of other conditions, such as anorexia nervosa. There are numerous positive studies showing the effectiveness of this form of therapy in depression. This therapy is based on interpersonal theory from the work of Adolf Meyer, Harry Stack Sullivan and later John Bowlby and other individuals. The theory argues that life events occurring after the formative years influence psychopathology. The focus is on an interpersonal problem, for example, a complicated bereavement, relationship difficulty or an interpersonal deficit, using techniques from different psychotherapies. It has some overlap with psychodynamic therapy and CBT, and deals with four interpersonal problem areas: grief, role dispute, role transitions and interpersonal deficits.

Problems in a patient's life are split into one of these four areas and strategies are developed to help the patient to cope with difficulties or to think about them in a different way. As with CBT, homework is often used to enable the patient to experiment and try out these strategies in their day-to-day lives.

Group therapies

Group therapies often use components of other therapies, for example, psychodynamic groups and cognitive behavioural groups. It has been argued that there are many advantages to working in groups, making it a superior form of treatment to individual therapy, although most research studies do not convincingly show group therapy to be better than individual therapy. Groups may be closed and run for a specified period with the same members, or open with members coming and going depending on their needs. Irvin Yalom described a list of seven factors associated with the effectiveness of group therapy. There are universality, altruism, instillation of hope,

cohesiveness, corrective recapitulation of the primary family experience, self-understanding/interpersonal learning and catharsis as discussed in the following.

Universality

Universality describes the shared experiences among clients. It helps the clients overcome their sense of isolation, validates their experiences and raises self-esteem. For many, it may be the first time they feel understood and like other people.

Altruism

As members are able to help each other, they identify their own strengths but also learn from each other, thus improving their own self-esteem, and develop better coping styles and interpersonal skills. By being able to help others their own sense of worth is likely to increase, and for many it might be the first time they have felt useful or as if they have something to offer.

Instillation of hope

The group members are able to learn from each other and gain hope through the experience of others. As each member in a therapy group is inevitably at a different point on their therapy (this would only be the case for open groups), watching others cope with and overcome similar problems usually instils hope and inspiration. New members or those in despair may be particularly encouraged by others' positive experiences.

Cohesiveness

Cohesiveness is often referred to as the most therapeutic factor from which all of the other factors flow. Humans have an instinctive need to belong in groups, and some personal development can only occur in interpersonal relationships. Cohesiveness brings belonging, acceptance and validation to the counselling process.

Corrective recapitulation of the primary family experience

Members of a group will often unconsciously identify other members of the group as similar to their own immediate family. In this way through the therapy process, they can deal with these unresolved issues and address unhelpful patterns in their relationships. For example, a dominant member of the group may remind them of their authoritarian father and their need to rebel. Through this identification they may be able to address their feelings in a more appropriate way than, for example, with substance misuse.

Self-understanding/interpersonal learning

Members of group counselling may achieve a higher level of self-awareness through interactions and observations of others. They may also achieve greater levels of insight about the origins of their own problems and their maladaptive coping strategies.

Catharsis

Catharsis is defined as the experience of relief from emotional distress by expressing emotion. This is often a very positive part of the group therapy process, and relating their experiences and not being judged by them can be a way of helping people move forward.

Group therapies may be particularly useful for problems people have with relationships. Because of the group situation an important caution in group therapy would be any form of anxiety, particularly associated with a group setting such as social phobia.

Counselling

Counselling is a widely used term that has been defined as 'the means by which one person helps another to clarify his or her life situation and decide further lines of action'. In fact, various forms of counselling are used.

The humanistic or client-centred approach was developed by Carl Rogers, who emphasized the importance of recognizing that individuals already have the ability to work through their problems and that a counsellor's role is to facilitate this process by providing conditions of warmth, empathy and unconditional positive regard.

Other forms are more therapist led. For example, problem-solving counselling purely focuses on identifying and formulating problems, setting clear and achievable goals, generating alternatives for coping

and then allowing an individual to problem-solve as necessary. Problem-solving counselling is very much recommended for many types of less severe mental illness such as mild forms of depression and anxiety.

Other forms of counselling use techniques developed separately as therapies, for example, cognitive behavioural counselling and interpersonal counselling, which would be virtually indistinguishable from CBT and interpersonal therapy. Psychodynamic counselling is another form of counselling that uses psychodynamic theory, and one of the most widely recognized forms of counselling is grief or bereavement counselling, which is widely used to help individuals with the loss of a loved one to help them work through the different stages of grief (denial, anger, bargaining, depression and acceptance).

Family therapy

Most children, adolescents and young adults live within families. Families are an environmental factor that can influence the presentation and maintenance of mental health problems, so it is perhaps unsurprising that there is a treatment modality which is specifically aimed at working with families. A child mental health assessment should include a brief assessment of family functioning, which will identify any need for more detailed exploration.

Family therapy is one of the most effective therapies available, with good evidence for its use. It is the first-line treatment for several disorders of childhood, including eating disorders. However, it also has a place in anxiety, bereavement disorders, conduct problems, substance misuse, chronic illness and psychosomatic disorders. The success of family therapy in the classic sense is less proven in substance misuse and conduct problems, but in conjunction with other therapies it can be useful.

Family therapy was developed on the basis that there was a 'right' way for families to function and family therapy helped the family negotiate this right way. Over time, the approach has changed and there is a greater focus on collaboration and seeing family therapy intervention as a way of helping families find their own solutions. However, in practice, family therapy remains about helping families find better ways of being or functioning.

Principles of family therapy

There are clear principles at work for most of the models:

- Children need parents (or carers) who love and care for them, are emotionally responsive to their needs but also set clear and appropriate boundaries (this is culturally and socially dependent).
- Most people take a fairly pragmatic approach to defining a family and let the family decide for itself who is part of their family.
- Parents take on adult roles and responsibilities and set limits and boundaries. Where the adults fail to do this, intergenerational boundaries are considered too fluid. Where there is no understanding of a need to be flexible, the boundaries are considered too rigid.
- The family is an interdependent unit with flexibility to adapt to changing life stages and circumstances.
- There is a healthy balance between individuals in the family caring about each other but also having relationships outside of the family (depending on age).
- Families in which members are overly dependent on each other without a sense of individual autonomy are described as 'enmeshed'. It is mindful to be aware of cultural expectations, but even families that subscribe to a collective sense of self have individuation within a context.
- It is important not to attach blame but help families understand that they function the way they do for a host of reasons, and family work helps unpack some of these reasons and allow for improved family functioning.

Psycho-education

As a medic, it is important to educate our patients or their families about a particular mental illness or treatment. This is referred to as psycho-education. Psycho-education can be a conversation between a doctor and a patient about the importance of

compliance with medication for example, or it could be a formal training package that can be delivered face-to-face or online to help patients or families understand about a particular mental illness. There are numerous psycho-education packages that can be delivered to people with schizophrenia and bipolar disorder. The packages consist of a number of teaching sessions that educate the patient or family about the condition, medication compliance, relapse prevention and a variety of other topics. The overarching aim of psycho-education is to improve patients self-management of their condition and thus reduce the chances of future relapse.

Summary

This chapter described the common components of psychological treatments and outlined the features of behavioural therapies, CBT, counselling, family therapy, group therapy, interpersonal therapy, psychodynamic psychotherapy and psycho-education. The clinical indications for these types of therapy have been explained.

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CASE STUDY 17.1

David is a 35-year-old doctor who has suffered with two episodes of depression in his life. The second one started 2 months ago. He has low mood, decreased energy, sleep problems and appetite loss. This has impacted on his work and he is currently taking sick leave. He was commenced on an antidepressant by his general practitioner (GP). There has only been a partial response and his GP has suggested a course of CBT.

Explain the term CBT to David.

Advise him how it will work to improve his depression and any side effects of the treatment.

CBT is a structured form of talking therapy that has been proven to be very effective in treating a number of mental health problems including depression. It works on the premise that when you are suffering from mental illness your thinking pattern is different. Cognition is another word for thought. The different thinking pattern is characterized by a number of cognitive distortions. For example, if you are depressed you may minimize all the positive things in your life and maximize (blow them out of proportion) the negative things.

As a consequence of this different pattern of thinking there is an emotional response (feeling low in depression or anxious with a phobia), and this in turn leads to a behaviour associated with the disorder. In the case of depression the negative thought pattern will lead to feeling down, which may lead to not going out or seeing friends, which perpetuates the difficulties.

CBT looks at altering the thought patterns that are seen in the mental illness in a hope to change the emotional response and subsequent behaviours. By challenging the negative thoughts and offering behavioural exercises like activity planning this can improve your depression.

CBT rarely has side effects. It does demand a lot of work on the part of the patient though (it is not as easy as swallowing a pill every day!). There are usually homework assignments and the individual has to continue with the techniques they have learnt long after the formal therapy has finished.

CASE STUDY 17.2

Marianne is a 26-year-old barrister. All her life she has had an irrational fear of pigeons. This started as a child when she was taken to Trafalgar Square by her parents to feed the pigeons and two of them landed on her head and pecked her. Now she gets panicky even if she sees pigeons on the television. The phobia has never been a problem as she lives in the country and went to university in Cambridge, where there were not too many pigeons; she could always detour around them. However, in the past month she has been offered a job with a prestigious law firm in the city of London. She is terrified about encountering a flock of pigeons and wonders whether she can take the job or not. She does not want this irrational fear to prevent her career progression. She has heard that there may be some help available for her in the form of a talking therapy.

What kind of therapy might be useful for Marianne?

Devise a structure to her treatment. Marianne may well respond positively to a systematic desensitization approach which is often used to successfully treat phobic anxiety disorders. Below is a hierarchy that may be used to address her pigeon phobia.

A systemic gradual desensitization approach includes:

1. Discussion of pigeons
2. Picture of a pigeon
3. Picture of a flock of pigeons
4. Film of a pigeon
5. Film of a flock of pigeons
6. Real-life exposure to a pigeon at a distance
7. Exposure to a flock of pigeons
8. Exposure to a flock of pigeons flying
9. Close exposure to a flock of pigeons
10. Pigeons eating seed out of her hand
11. Pigeons landing on her head

You probably know how to explain a physical treatment to a patient such as a medication or surgical intervention. Think about how you might explain a psychological treatment to a patient.

What words would you use? How would you explain its mode of action?

We all explain medications to patients routinely every day, why not use the same template as we use when talking about medication? Talk about modes of action, side effects, how long you have to take the treatment for. It is not as difficult talking about psychological treatments as you may think. Remember do not use jargon or big medical words. Give the patient an opportunity to ask questions and if you do not know the answer to the questions, just say so and tell the patient you will find out.

Here are some words and phrases that you may be able to use:

- Talking therapy
- Thoughts
- Feelings
- Behaviours
- Relaxation
- The mode of action of psychological treatments may include words like:
- Listening and talking
- Release of emotion
- Giving information
- Providing a rationale
- Restoration of morale
- Suggestion
- Guidance and advice
- Supportive and therapeutic relationship

CASE STUDY 17.3

Alexa is an 11-year-old girl referred by her GP for features of obsessive – compulsive disorder (OCD). The history from the mother reveals that over the last 4 months Alexa has developed counting rituals and also has to complete some activities a specific number of times. The rituals do not appear to be present at school, but it is unclear if she is just better at hiding them there. Alexa appears to be constantly angry and not interested in anything. Alexa herself believes something bad will happen if she does not complete the rituals. The background is that Alexa has suffered from chronic airways disease for several years. She has at times become cyanosed and needed emergency oxygen. She is currently using a continuous positive airway pressure machine to prevent her from becoming cyanosed when asleep because of sleep apnoea. Alexa heard the paediatrician say that she requires further operations and this has caused some anxiety. She does not appear to understand her medical condition except that it is worrying for the adults. Alexa presents as a very anxious girl. She also expressed some distress around family arguments related to her tantrums.

What is likely to be the primary diagnosis?

The primary diagnosis is likely to be anxiety, and the OCD features are a way of managing her anxieties rather than OCD per se. Having rituals helps emotional containment.

What interventions might be helpful in this case and why?

The intervention involved individual work with Alexa to help her think about what her worries were and how they might be addressed. The work with her parents involved helping them to help each other as a couple contain their anxieties about Alexa's medical problems. In doing this, they were able to be firmer and clearer with Alexa when she had a tantrum. During the tantrum they focused on ensuring they remained firm and consistent. However, they were also able to identify times when they could be responsive to Alexa and discuss her worries in an appropriate way.

CHAPTER

18 Psychopharmacology and physical treatments

STEPHEN COOPER

KEY CHAPTER FEATURES

- Outline of the pharmacological mechanisms of action of the main drugs used in the treatment of mental illnesses.
- Description of the clinical use of these drugs and issues surrounding the choice of drug to prescribe.
- Description of the main adverse effects and drug interactions.
- Summary of key issues relating to ECT and some other non-drug physical treatments.

Introduction

The general aim of this chapter is to give you an understanding of the mode of action and adverse effects of those drugs commonly used in the management of psychiatric illnesses. The drugs described under this heading are often termed 'psychotropic drugs'. Alcohol and a wide variety of drugs of misuse and abuse, such as cannabis and cocaine, also have potent effects on brain neurotransmitter systems and these may be termed 'psychotropic agents'. However, this chapter will focus on therapeutic psychotropic drugs.

The drugs to be discussed achieve their effects through alteration of function in neuronal systems in the brain. The main mechanisms by which they achieve this are as follows:

- Alteration of neurotransmitter availability or release
- Antagonism or agonism at neurotransmitter receptors
- By having an effect on second messenger systems in neurones or directly at ion channels

Various aspects of the pharmacokinetics, mechanisms of action, adverse effects and drug interactions are discussed for each group of drugs. Understanding these basic issues helps you understand how to use these drugs in practice and the information you need to give patients

about their treatment. Good communication with patients about their treatment, and potential adverse effects, improves adherence (compliance) with treatment and hence the likelihood of good clinical response.

Antidepressants

Antidepressants are used for the treatment of patients with moderate and severe depressive disorder (Chapter 6) and also some forms of anxiety disorder (Chapter 7 and the 'Axiolytics and hypnotics' section of this chapter.). Since the development of the first antidepressant drug in 1957, there have been considerable advancements in our understanding of the probable mechanisms of action of these drugs. This has resulted in the development of drugs with greater safety in overdose and profiles of adverse effects that most patients find easier to tolerate. There are now over 20 drugs licensed for use as antidepressants in the United Kingdom. The main subgroups are described in Table 18.1. There is little consistent evidence to suggest that for the majority of patients one antidepressant might be more effective than another for the initial treatment of a first episode of depressive illness. Box 18.1 provides an outline of how to approach the initial phase of pharmacological treatment of depression. Remember that as well as being guided by the clinical presentation, choice of medication must also be partly guided by risk of suicide and patient preference in terms of adverse effects profile. The more cardiotoxic drugs, such as tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs) should be avoided where possible in patients with a significant suicide risk as they may take an overdose of their medication.

Though 50%–60% of patients may have a significant response to treatment, it is estimated that at the end of the initial phase of treatment only 35%–50% of patients will have achieved complete remission compared with around 25% on placebo. (For those patients with a

Box 18.1 Initial pharmacological treatment of moderate to severe depression

- A selective serotonin reuptake inhibitor (SSRI) is the drug of 'first choice' when starting a patient on an antidepressant and can usually be commenced at full therapeutic dose. Ideally, clinical review should be every 1–2 weeks. Approximately 50%–60% of patients are likely to have a significant response.
- If psychotic symptoms are present (only the most severely ill) an antipsychotic should be added, e.g. quetiapine.
- If there is little or no clinical response after 4–6 weeks, consider a switch to a second drug. This can be either a different SSRI or one of the other newer types of antidepressant: noradrenalin a reuptake inhibitors (NARIs), serotonin and noradrenalin reuptake inhibitors (SNRIs), a receptor antagonist, or agomelatine. (*Note:* Only 20% of those with no response at 4 weeks will go on to respond to their first drug by 12 weeks.)
- If the patient is more severely ill, then one of the following may be the best choice: venlafaxine, escitalopram, sertraline, mirtazapine or a tricyclic antidepressant. (Some guidelines would recommend these as second-line treatment for such patients but other guidelines would advise them as first-line for severely ill patients.)
- Occasionally, if there is a partial response after 4 weeks, an increase in dose of the antidepressant, above the usual dose, may be considered, if adverse effects are minimal, but evidence for effectiveness of this strategy is limited.
- If there is no response to the initial two antidepressants then a tricyclic antidepressant (TCA) should be considered, if not previously prescribed.
- For patients who still do not respond after a third antidepressant a number of strategies can be considered depending on the exact nature of the symptoms and clinical presentation. Some of these are listed below:
  - Augmentation of the antidepressant with lithium
  - An SSRI / TCA or an SSRI / NARI combination
  - Electroconvulsive therapy (ECT)
  - An MAOI if there are prominent anxiety symptoms

partial response it is sometimes appropriate to consider augmentation of their antidepressant drug with a second-generation antipsychotic drug or lithium.) After the phase of acute treatment of a first episode of depression, prophylaxis is required for at least 6 months, and ideally 12 months, to reduce the risks of relapse or recurrence and increase the likelihood of complete remission of symptoms. Usually prophylaxis should be with the dose of antidepressant to which the patient's acute episode responded. Those with recurrent episodes require prophylaxis over many years.

Neurochemical mechanisms in depression

Current theories of the biological mechanisms underlying depressive illness are largely derived from observations of the neurochemical effects of effective antidepressant drugs and electroconvulsive therapy (ECT). Altered function of the serotonin or 5-hydroxytryptamine (5-HT) and/or noradrenalin (NA) systems are thought to be important (Chapter 2) in explaining depression. This may result from disturbance of signal transduction and gene expression within neurones. Other evidence demonstrates disturbance of the corticosteroid system and potentially important interactions between this and other neurotransmitter systems. Where the initial disturbance lies is not yet clear, and indeed pathways may differ between patients. The aim of antidepressant treatment is to normalize the function of these systems.

Classification of antidepressants

Antidepressants are classified according to the principal pharmacological effect they cause rather than by specific chemical structure (Table 18.1). The exceptions to this are the TCAs, which share a common chemical structure and also common pharmacological effects. The other group of drugs used in the treatment of mood

Table 18.1 Classification of antidepressant drugs with examples (for an exhaustive list see the British National Formulary [BNF])

Group	Principal pharmacological effects	Examples
Tricyclic antidepressants (TCAs). (Now should probably be called non-selective monoamine uptake inhibitors.)	Inhibition of reuptake of 5-HT and NA in synapse. Most have weak effects on DA reuptake. Cholinergic receptor antagonism.	Amitriptyline Clomipramine Imipramine Lofepramine
Selective serotonin reuptake inhibitors (SSRIs).	Inhibition of reuptake of 5-HT in synapse.	Citalopram Fluoxetine Paroxetine Sertraline
Noradrenalin reuptake inhibitors (NARIs).	Inhibition of reuptake of noradrenaline (NA) in synapse.	Reboxetine
Serotonin and noradrenalin reuptake inhibitors (SNRIs).	Inhibition of reuptake of 5-HT and NA in synapse. Lack other effects of TCAs that give rise to many of their adverse effects.	Venlafaxine Duloxetine
Receptor antagonists. (Sometimes called atypical antidepressants.)	Have some amine uptake inhibition but main effect is to antagonize one or more of 5-HT_2, alpha$_1$-NA, alpha$_2$-NA receptors, resulting in increased amine availability in the synapse.	Mianserin Mirtazapine Nefazodone Trazodone
Monoamine oxidase inhibitors (MAOIs).	Inhibition of MAO types A+B and thus reduce metabolism of 5-HT and NA. (These older drugs are non-selective and the effects are irreversible.)	Isocarboxazid Phenelzine Tranylcypromine
Reversible inhibitors of MAO-A (RIMAs).	Inhibition of MAO-A in a competitive and reversible manner.	Moclobemide
Miscellaneous.	Agonist at melatonin (MT_1 and MT_2) receptors and an antagonist at 5-HT_{2c} receptors. Increases plasma level of 5-HT precursor. May augment effects of MAOIs but probably no antidepressant effect on its own.	Agomelatine L-tryptophan

disorders are the mood stabilizers (lithium compounds and some anticonvulsants), which are discussed in the section 'Mood stabilizers'.

The selective serotonin reuptake inhibitors (SSRIs) are the most widely prescribed antidepressant drugs in the United Kingdom. Their development during the 1980s and 1990s represented one of a series of steps in the wider development of antidepressant drugs with more specific pharmacological effects and less adverse effects. Prior to this, the TCAs had been the mainstay of antidepressant treatment, but they had a wide spectrum of pharmacological effects and were associated with many adverse effects. They remain in use for patients whose illness is more resistant to treatment. The MAOIs were another of the first generation of antidepressant drugs. They also have problematic adverse effects, though some newer and safer versions have been developed. To understand the popularity of the SSRIs it is useful to have knowledge of what preceded them.

Tricyclic antidepressants

TCAs were so named because of the three-ring structure that is central to all of these drugs. Their principal therapeutic pharmacological effects are inhibition of 5-HT and NA reuptake into the presynaptic terminal, thus enhancing the effects of these monamine neurotransmitters at the postsynaptic receptors. All TCAs inhibit reuptake of both 5-HT and NA, but they vary in their potency of effect for each. For example, clomipramine is about 1,000 times more potent in its inhibition of reuptake of 5-HT than NA, whereas lofepramine is about 1,000 times more potent in its effects on NA reuptake than 5-HT. Most of these drugs will also inhibit dopamine (DA) reuptake to some degree and are potent antagonists at the muscarinic cholinergic receptor, the H_1 histamine receptor and $alpha_1$-adrenoceptors.

Reuptake inhibition occurs immediately on commencing these drugs, but experimental studies in laboratory animals and humans demonstrate a number of other pharmacological effects occurring with longer term (in the experimental context) use. There is down-regulation of postsynaptic $5\text{-}HT_{2A}$ receptors, presynaptic and cell body $5\text{-}HT_{1A}$ receptors, presynaptic $alpha_2$-adrenoceptors and $beta_1$-adrenoceptors. This means that the increase in synaptic concentration of neurotransmitter results in an adaptive response by the neuron. Given the interactions in the central nervous system (CNS) between the 5-HT and NA systems, the result of these effects is difficult to determine but is thought to be an overall enhancement of 5-HT function (which is reduced in depression) and a stabilization of NA function (which is probably dysregulated in depression). It was originally thought that onset of the therapeutic effect did not occur until the aforementioned adaptive changes had occurred, after about 10–20 days, but evidence now suggests that 35% of eventual symptom improvement may be seen by the end of the first week of treatment in those who ultimately respond.

Pharmacokinetics and dosing

TCAs are all lipid soluble, well absorbed from the gut and widely distributed in the body. Peak plasma concentrations are reached 2–6 hours after a single oral dose, and elimination half-life is between 8 and 36 hours, generally allowing once daily dosing. Most have active metabolites, also with relatively long half-lives. They are highly bound to plasma proteins (75%–95%) and undergo extensive hepatic metabolism.

There is no evidence for a clear relationship between plasma concentration and clinical response. An approximate 80–180 μg/L 'therapeutic window' has been suggested for imipramine, with levels below this being unlikely to induce response and levels above being unlikely to induce additional improvement. However, routine use of plasma levels is not of value in monitoring treatment, unless poor patient adherence with treatment is suspected.

TCAs are usually prescribed as a single nighttime dose because of their sedative effects and long elimination half-life. The dose is usually increased gradually to allow some development of tolerance to the adverse effects. If the dose required is large, a twice daily dose regime may be appropriate.

Adverse effects and toxicity

Sedation is common, especially early in treatment, and is largely related to antihistamine and antiadrenergic effects of the TCAs. Weight gain may also be related to antihistamine effects. Some recent evidence suggests there can be impairment of psychomotor function, but in the treatment of patients with depression this must be balanced against improvements in function likely to occur in conjunction with improvement in the illness.

Blockade of muscarinic cholinergic receptors by TCAs results in what are often the most

troublesome adverse effects (commonly referred to as their anticholinergic effects): dry mouth, blurred vision, constipation and (mainly in older males) urinary hesitancy and retention. Pupil size, and hence risk of acute closed angle glaucoma, is determined by a balance between anticholinergic (mydriatic) and sympathomimetic (miotic) effects, which varies between drugs. Sympathomimetic effects (from NA reuptake inhibition) and antimuscarinic effects can cause a sinus tachycardia. Postural hypotension may occur as a result of sympatholytic alpha$_1$-adrenoceptor antagonism.

TCAs have direct effects on cardiac tissue, causing impairment of cardiac conduction and decreased myocardial contractility. They are *extremely dangerous in overdose* because of these direct cardiac effects and their anticholinergic effects. They are responsible for 10%–20% of deaths from drug poisoning in England and Wales. This risk of overdose is shared with MAOIs and some other antidepressants, particularly venlafaxine. They should be avoided if possible in patients with cardiac disease and are contraindicated after a recent myocardial infarction.

Impaired libido and sexual function can occur in both males and females. While TCAs can cause raised prolactin levels, this is not a common effect, is usually asymptomatic and not related to sexual dysfunction.

These drugs lower the seizure threshold and can occasionally result in convulsions. In patients with bipolar affective disorder (manic depressive illness) mania can be precipitated, possibly partly related to the inhibition of DA reuptake. There is no specific evidence of teratogenicity toward the fetus but, like most drugs, they should be avoided if possible during pregnancy. Care is required in elderly people as they are more sensitive to the adverse effects, have a lower rate of metabolism and excretion and are more likely to have concurrent physical disease (e.g. cardiac disease).

Drug interactions

TCAs potentiate the pressor effects of directly acting sympathomimetic amines, such as adrenalin or NA, to cause hypertension. Small amounts of these, such as that present in dental anaesthetics, can be dangerous. TCAs will inhibit the antihypertensive effects of the older antihypertensive drugs, such as adrenergic neurone blocking agents (e.g. guanethidine), alpha-methyl-DOPA and clonidine. Interactions with some anti-arrhythmics are possible.

Taken with alcohol, they potentiate the sedative effects and impairment of psychomotor performance. Hepatic enzyme induction by anticonvulsants or nicotine may reduce plasma levels. Cimetidine, beta-blockers and calcium channel antagonists may increase TCA levels by inhibition of hepatic enzymes. Some antipsychotic drugs may compete for similar metabolic pathways, resulting in altered plasma concentrations of both.

Co-administration of TCAs with MAOIs will result in increased adverse effects, particularly antimuscarinic effects, and can lead to a dangerous toxic interaction, 'serotonin syndrome' (see 'Drug interactions' in the section 'Selective serotonin reuptake inhibitors'). Although in the past such a combination might have been used in hospital for very treatment-resistant patients it would be very rare to consider it now.

Selective serotonin reuptake inhibitors

SSRIs are the treatment of first choice for someone presenting with onset of a new episode of depression, essentially because of their safety in overdose and their relative lack of adverse effects compared with TCAs. These are all inhibitors of reuptake of 5-HT with no significant effect on reuptake of NA. They are well absorbed and elimination half-lives for most people are in the region of 15–40 hours. There are active metabolites but, except for fluoxetine, these do not contribute greatly to the therapeutic effect. Dosing is once daily in the morning and SSRIs can usually be started at full therapeutic dose because of the relative lack of severe adverse effects.

Adverse effects

Nausea and sometimes vomiting may occur because of activation of 5-HT$_3$ receptors by SSRIs. Diarrhoea and headache may also occur. Tolerance generally develops to all of these effects within 7–10 days and explanation of this to patients will often aid treatment adherence. Sexual problems are quite common, principally these include delayed ejaculation and anorgasmia and reduced libido. The frequency of these is difficult to determine as patients often fail to report these spontaneously and they can also be a symptom of depression.

Antimuscarinic effects are rare. SSRIs increase the risk of gastrointestinal bleeding, particularly in the elderly, probably due to alteration of platelet

function. Care is required in patients on warfarin, aspirin or non-steroidal anti-inflammatory drugs (NSAIDs).

A syndrome of restlessness or agitation, which seems very similar to the akathisia sometimes induced by antipsychotic drugs, may occur early in treatment in 5%–10% of patients. This may be accompanied by increased suicidal ideation. It is an indication to stop the SSRI immediately and seek advice from a more senior doctor. Some studies have suggested that increased suicidal ideation per se, in isolation from the above restlessness, occurs in some patients, particularly adolescents. As a result, careful clinical monitoring is required early in treatment. For depressed patients under 18 years of age only fluoxetine is licensed, as current evidence suggests it carries less risk in adolescent patients. It should be noted that controversy surrounds this area with analysis of reports from certain types of clinical trials suggesting an increase in thoughts of suicide but other epidemiological studies not suggesting a link with suicidal behaviour. Despite this uncertainty, SSRIs should still be considered, but prescription must be followed by careful monitoring and clinical care.

Sedation is uncommon and many patients find that these drugs prevent them getting to sleep, which is why the dose is best taken in the morning. Effects on psychomotor function can occur but are uncommon compared with TCAs. Epileptic convulsions can occur but are rare and much less common than with TCAs. There is some evidence for potentiation of ECT-induced seizures. Reduction in plasma sodium can occur, particularly in elderly people, and needs to be borne in mind.

A recent study has shown that citalopram and escitalopram may have a dose dependent effect on QT_c interval on ECGs. As a result, the Medicines and Healthcare Products Regulatory Agency (MHRA) has reduced the maximum daily dose for patients over 65 years of age and advised that these two SSRIs should not be prescribed to patients with known pre-existing QT prolongation and be preferably avoided with other medications that may prolong QT interval. Otherwise effects of SSRIs on the cardiovascular system are rare.

Drug interactions

Co-administration of SSRIs with other drugs acting to enhance 5-HT function can result in development of the serotonin syndrome. This almost invariably occurs if co-prescribed with MAOI antidepressants. Serotonin syndrome can occur with TCAs, lithium and L-tryptophan, but combination with these drugs for treatment-resistant patients is acceptable as long as they are carefully monitored in the early stages of treatment. In serotonin syndrome, the patient is initially restless and may have nausea or diarrhoea. Hyperthermia, rigidity, tremor, myoclonus, autonomic instability and convulsions may develop with fluctuating levels of consciousness. Death may ensue.

SSRIs, particularly fluoxetine and paroxetine, can inhibit some hepatic enzymes and thus may affect metabolism of opiates, many antipsychotic drugs, TCAs, beta-blockers and some anti-arrhythmic drugs.

Withdrawal syndrome

Sudden cessation of an SSRI antidepressant can result in unpleasant symptoms that constitute a withdrawal syndrome, the symptoms of which are listed in Box 18.2. Recovery from these symptoms usually occurs within 7–14 days. These symptoms are probably due to a sudden change in 5-HT function following cessation of the antidepressant. Their frequency and severity vary considerably between SSRIs with over 60% of patients stopping paroxetine experiencing these, but less than 30% with other SSRIs. This difference is largely due to the relatively short elimination half-life of paroxetine resulting in much faster washout of the drug.

Similar withdrawal symptoms have been reported with almost all antidepressant drugs but are less frequent and severe with TCAs and MAOIs because these drugs have quite long half-lives. However, the fact that

Box 18.2 SSRI withdrawal symptoms

Neurological	Dizziness Vertigo	Headache Paraesthesiae
General somatic and GI	Nausea Anorexia Sweating	Emesis Diarrhoea
Neuropsychiatric and psychological	Fatigue Anxiety Hallucinations Impaired concentration	Irritability Mood change
Sleep disturbance	Insomnia	Vivid dreaming

this occurs indicates that when any antidepressant is to be stopped the dose should be gradually tapered down over a period of 2–4 weeks.

Monoamine oxidase inhibitors

The enzyme monamine oxidase (MAO) exists in two forms: MAO-A (intestinal mucosa and intraneuronally in the brain) and MAO-B (platelets and mainly extraneuronally in the brain). 5-HT is preferentially metabolized by MAO-A, and NA, DA and tyramine by both forms. The original MAOI antidepressants (phenelzine, tranylcypromine and isocarboxazid) inhibit both MAO-A and MAO-B and are thought to work through increasing the availability of 5-HT and NA in the synapse – with longer term adaptive effects occurring as for the TCAs. These MAOIs are 'irreversible', that is they permanently inactivate MAO. Thus, recovery of activity after stopping the drug occurs slowly, over days, as new MAO molecules are synthesized.

These drugs are rapidly absorbed and achieve peak plasma level 2 hours after a single dose. Although elimination half-lives are generally short, the irreversibility of the effect on MAO makes this largely irrelevant. Patients who are slow acetylators (approximately 50% of the Caucasian population) may develop toxic plasma concentrations.

Recently, drugs selective for each form of MAO as well as drugs that are reversible inhibitors of each form have been developed. The aim of these strategies is to develop drugs less prone to the tyramine interaction ('cheese effect') and which can be withdrawn or changed to another drug more rapidly. Of these, the only drug in regular use is moclobemide, a reversible inhibitor of MAO-A (RIMA).

Tyramine interaction (the 'cheese effect')

The clinical effect of a tyramine interaction (cheese reaction) is a hypertensive crisis: flushing; severe throbbing headache; severe hypertension; tachycardia; pallor. This is a dangerous condition and there is a risk of cerebral haemorrhage. Treatment is with an alpha$_1$-adrenoceptor antagonist (phentolamine or chlorpromazine), which is usually given by slow intravenous injection.

Tyramine acts as an indirect sympathomimetic to cause release of catecholamines from nerve terminals. Tyramine is found in several foods: mature cheeses, yeast extracts, some red wines, hung game, pickled herrings and broad bean pods. Normally MAO-A in the intestinal mucosa will metabolize tyramine absorbed from the gut. In patients on the older MAOIs, considerable amounts of tyramine will enter the circulation (because mucosal MAO-A is inactivated) and will act to release the increased amounts of catecholamines stored in nerve terminals – increased because of the effects of the MAOI in preventing their metabolism. For patients on the RIMA drugs, high concentrations of tyramine can compete for MAO-A, thus mitigating some of the effects, and MAO-B is still available to metabolize NA. MAO-B, however, has relatively much less effect on 5-HT and thus 5-HT function is still enhanced.

Adverse effects and toxicity

MAOIs are best avoided in patients with cerebrovascular, cardiovascular and hepatic disorders. Some sympathomimetic effects may occur, mainly mild tremor and occasionally cardiac arrhythmias. Apparent 'anticholinergic' effects may also occur but these are mainly the result of sympathetic potentiation in tissues with dual cholinergic/adrenergic innervation, e.g. pupil. Sympatholytic effects can also occur, principally postural hypotension, because of synthesis of relatively inactive 'false' transmitters (e.g. octopamine) in nerve terminals following inhibition of MAO and activation of alternative metabolic pathways.

Other adverse effects noted are restlessness, insomnia, peripheral oedema and sexual difficulties. These drugs carry similar risks of cardiac toxicity in overdose to the TCAs.

Drug interactions

Tyramine-containing foodstuffs must be avoided. Indirectly acting sympathomimetic amines, such as phenylpropanolamine or phenteramine, that are used as nasal decongestants or bronchodilators also pose a risk. These may also be found in over-the-counter cough mixtures and will release the enhanced neuronal stores of pressor amines. There can also be interactions with a number of antihypertensive drugs.

Combination with SSRIs carries a high risk of serotonin syndrome. Use with TCAs carries some risk of this and should be avoided except for occasional inpatient treatment of the most treatment-resistant patients. MAOIs also potentiate the effects of opiates, particularly pethidine. This is in part a pharmacokinetic interaction and may result in a syndrome of excitement, hyperpyrexia,

muscle rigidity, sweating, respiratory depression and hypotension. If opiates must be given to a patient on an MAOI the initial dose should be 10% or less of the usual dose.

Other antidepressant drugs

This chapter does not detail all of the pharmacological effects of the antidepressants in the noradrenalin reuptake inhibitors (NARI), serotonin and noradrenalin reuptake inhibitors (SNRI) and receptor antagonist groups. Their principal pharmacological actions include inhibition of reuptake of NA, 5-HT or both and they generally have some additional direct effects on specific receptors, which in theory should assist their therapeutic action. For example, mirtazapine has alpha$_2$-adrenoceptor antagonist properties that should enhance NA activity and also has a secondary effect of enhancing 5-HT activity through adrenergic effects on the serotonergic raphe nuclei. Venlafaxine enhances NA and 5-HT reuptake but lacks the antimuscarinic effects of the older TCAs. The adverse effects of these drugs are relatively similar to those of the TCAs but tend to be much less frequent and severe. Similar drug interactions may occur but are generally less likely. Venlafaxine can cause elevation of blood pressure in some patients and for that reason blood pressure should be checked before starting and over the initial weeks of treatment.

Agomelatine is an agonist at the melatonin (MT_1 and MT_2) receptors and a 5-HT_{2c} receptor antagonist and thus has a very different mode of action to other antidepressants. Adverse effects include sedation, nausea and sometimes insomnia but are generally less than for most SSRIs. Agomelatine has minimal effects on sexual function. In 1% of patients, disturbance of hepatic function may occur so serum transaminase levels must be monitored before and at 6, 12 and 24 weeks after commencing treatment.

L-tryptophan, the amino acid precursor of 5-HT, has been used as an antidepressant as its administration may enhance 5-HT function. There is doubt whether on its own it is an effective antidepressant but it has been used successfully to augment the effects of other antidepressants, particularly MAOIs, in treatment-resistant patients. Mild sedation is the main adverse effect. Some years ago, there were concerns that L-tryptophan might be associated with an eosinophilia-myalgia syndrome, so it is wise to monitor eosinophil count initially after starting L-tryptophan.

Mood stabilizers and prophylaxis

Patients who have experienced an episode of hypomania, or full mania, have an 80% risk of a further episode of the illness – which may be either of mania/hypomania or depression. Similarly, patients who have had a first episode of severe major depressive disorder with associated somatic symptoms and/or psychosis have an 80% risk of recurrence at 3 years. Following a second episode of either type of illness, the risk of further recurrence rises to well over 90%. Thus, prophylaxis is very important for both bipolar and unipolar affective disorder and is more or less mandatory if a second episode occurs within 2 or 3 years of the first. Immediately following the first episode, prophylaxis is necessary for at least 6–12 months and further continuation will depend on the clinical situation and patient views.

For patients with bipolar affective disorder (manic depressive illness; Chapter 6) lithium, usually in the form of lithium carbonate, has been the main prophylactic agent for the last 50 years and remains the drug of first choice. However, during the last 25 years, consistent evidence for the value of certain anticonvulsants (carbamazepine, lamotrigine and valproate) has emerged. Antipsychotic medications are required in the treatment of acute episodes of mania, and sometimes hypomania, but can also be useful in the prophylaxis of bipolar disorder. It is important that antidepressant drugs are *not* prescribed for long-term use in patients with bipolar disorder as these increase the risk of relapse with mania/hypomania. Box 18.3 provides a summary of how to approach the immediate and long-term pharmacological treatment of bipolar disorder.

A small proportion of patients suffer from rapid-cycling bipolar disorder in which four or more episodes may occur per year. Some of these patients will respond to lithium but some respond better to valproate.

For patients with recurrent depressive disorder, prophylaxis should initially be through continuation of the antidepressant which was effective for the acute

Box 18.3 Pharmacological management of bipolar disorder

Phase of illness	Types of medication used	Specific medications	Comments
Acute mania	Antipsychotics (Usually required for acute mania.)	Haloperidol-olanzapine, quetiapine, risperidone	Which one is the drug of choice depends on concerns about particular adverse effect profiles and response in any previous episode of mania.
	Mood stabilizers (May be required in addition to an antipsychotic.)	Lithium or valproate	Continue any mood stabilizer if the patient is already prescribed one. If not already on one then decision depends on which is likely to be prescribed for subsequent prophylaxis.
Hypomania	Mood stabilizers	Lithium or valproate	This may be sufficient in milder cases.
	Antipsychotics	As for mania	Required for more severe cases or if poor response to mood stabilizer.
Prophylaxis	Mood stabilizers	Lithium valproate or carbamazepine lamotrigine	Most effective. Drug of first choice. These are less effective than lithium. Teratogenicity in pregnancy. Useful if majority of episodes are depressive but otherwise less effective than lithium.
	Antipsychotics (Sometimes needed in addition to a mood stabilizer.)	Quetiapine Olanzapine	These can also be used alone if a mood stabilizer is not tolerated or is not practicable (e.g. risk of pregnancy). Quetiapine more protective against episodes of depression than olanzapine.

episode (at the effective dose) but if this is not sufficiently effective then lithium can be tried on its own or in combination with an antidepressant.

Lithium

Lithium carbonate (e.g. as Priadel® or Liskonum®) and lithium citrate (e.g. as Camcolit®) are the most commonly used compounds. Lithium has effects on cation transport, on individual neurotransmitters (including 5-HT) and on intracellular second-messenger systems. It may also have neuroprotective effects. Among these the mode of action that is key to its therapeutic efficacy is not entirely clear but as for the antidepressant drugs, the net effects seem to be to enhance 5-HT function and stabilize the NA system.

Once lithium treatment is established, it is very important that it is not suddenly stopped as this may result in rebound hypomania in patients with bipolar disorder and increased risk of suicide in patients with all types of mood disorder.

Pharmacokinetics and dosing

Lithium is rapidly absorbed. Peak serum concentrations are reached in 2–3 hours. It is excreted unchanged by the kidney at a rate proportional to the glomerular filtration rate (GFR). It is best given as a single daily dose around 22.00 hours and steady-state serum levels are reached after 5–7 days of dosing, with the elimination half-life being around 10–24 hours for most people. Most formulations of lithium are in the form of a slow-release preparation. There can be variations in kinetics between different proprietary brands and it is best for individual patients to remain on the same brand.

Monitoring lithium treatment

Lithium has a narrow therapeutic range. The gap between minimum effective serum concentration (0.4 mmol/L) and that at which toxicity may begin to appear (1.2 mmol/L) is low and serum lithium concentrations can rise quite rapidly with even small changes in dose within this range. It is therefore

important to monitor serum lithium regularly. When establishing a patient on lithium treatment, or restarting treatment after a gap of more than a few days, monitoring must be done weekly until the final dose is established and steady-state serum lithium has been achieved. Blood samples for serum lithium should be taken 12 hours after the last dose (known as the '12-hour serum lithium'). For patients on regular dosing, serum concentrations are stable from around 12 hours after the last dose. Fluctuations of ±0.1 mmol/L between tests are acceptable but greater than this are a cause for concern. A common reason, however, is the patient varying the time of dosing or omitting a dose. Patients well established on lithium require a serum level approximately every 3–4 months, unless they show signs of lithium toxicity (Table 18.2 and Box 18.5).

Renal function must be assessed before starting lithium for two reasons. First, long-term treatment with lithium may sometimes impair renal function. Second, lithium is excreted entirely via the kidney, and patients with any impairment of function require greater care in their management. Assessment is now usually by serum creatinine and estimated glomerular filtration rate (eGFR), which is calculated automatically and is reported along with standard laboratory electrolyte and renal function screen values by most laboratories. If eGFR is not available, then a 24-hour creatinine clearance should be carried out. Serum creatinine and eGFR then require monitoring twice annually. Thyroid function (T_4 and thyroid stimulating hormone, TSH) must also be assessed before treatment and every 6 months, as lithium can induce hypothyroidism in 10%–15% of patients. Box 18.4 outlines an appropriate monitoring schedule.

Box 18.4 Monitoring of lithium therapy

- Before initiation: renal function (eGFR, electrolytes); thyroid function (T_4 and TSH); ECG if history of cardiovascular problems; pregnancy test
- During initiation: 12-hour serum lithium after 5 days and then every 5–7 days after each dose increase
- Long-term: 12-hour serum lithium every 3–4 months; eGFR and electrolytes every 6–12 months; thyroid function every 12 months. Frequencies will depend on symptoms and stability of results over time

The total daily dose of lithium required to achieve a therapeutic serum concentration is usually between 400 and 2000 mg. Initiation of therapy should usually be with 400 mg per day. Not all patients will achieve clinical response at the lower end of the therapeutic range and in the treatment of acute mania a higher concentration may be required to speed response. However, serum concentrations greater than 1.2 mmol/L must be avoided due to the risk of developing lithium toxicity. It should also be remembered that toxicity can occur in a few patients at concentrations less than 1.2 mmol/L and that identification of lithium toxicity is always a clinical diagnosis – it is simply much more likely to occur above 1.2 mmol/L and almost invariable above 1.6 mmol/L.

Adverse effects and lithium toxicity

Adverse effects of lithium toxicity are best considered in three groups (summarized in Table 18.2): 'minor', often occurring at the beginning of treatment with tolerance frequently developing; persistent and requiring monitoring during treatment; lithium toxicity, requiring 'immediate cessation of lithium' and 'urgent medical intervention' (Box 18.5).

Normal use of lithium may result in permanent renal impairment in a small proportion of patients. Polyuria occurs in 20%–40% and is due to inhibition of the action of antidiuretic hormone by lithium. It usually resolves on cessation of lithium as do any effects on glomerular function. Evidence suggests that once-daily dosing is preferable with regard to renal function, unless the daily dose is very high. There is evidence that a few patients may develop renal tubular damage. If eGFR is found to be falling less than 60 mL/minute, and continuing to fall over time, then a renal physician should be consulted and consideration given to stopping lithium.

Interference with thyroid function is due to inhibition of the action of TSH and is managed by prescription of thyroxine. Lithium is contraindicated during pregnancy (may cause major vessel anomalies in the fetus) and during breastfeeding.

Drug interactions

Thiazide diuretics and angiotensin-converting enzyme (ACE) inhibitors considerably reduce renal clearance of lithium and should be avoided if possible. Loop diuretics, such as furosemide, seem to have less likelihood of such effects, but any drug affecting fluid

Table 18.2 Adverse effects of lithium preparations

Minor effects to which tolerance generally develops	More persistent effects, some of which require to be monitored	Signs of lithium toxicity which require urgent intervention
Mild GI upset (nausea, vomiting, diarrhoea)	Polyuria and polydipsia	Severe GI upset
Fine tremor	Hypothyroidism	Worsening of existing side-effects
Metallic taste in mouth	Lethargy	Coarse tremor
	Weight gain	Ataxia
	Persistent tremor	Dysarthria
	T wave flattening on ECGs	Cardiovascular collapse
	Mild cognitive impairment	Impaired consciousness, coma
	Change in hair texture	Epileptic seizures
	Mild leucocytosis	
	Exacerbation of psoriasis	

Box 18.5 Lithium toxicity

- Lithium toxicity is a life-threatening condition and suspicion of this requires immediate medical attention. Patients and their families should be educated about common causes and symptoms.
- The common signs of this are described in Table 18.2. Appearance of any one of these should prompt suspicion.
- Lithium toxicity is a clinical diagnosis and can sometimes occur with serum lithium levels in the therapeutic range.
- Common causes of lithium toxicity are as follows:
  - Drug interactions (non-steroidal anti-inflammatory drugs [NSAIDs], thiazide diuretics, angiotensin-converting enzyme [ACE] inhibitors)
  - Dehydration (holiday in a hot climate, acute physical illness, perioperative fluid restriction)
  - Renal failure
  - Wrong dose of lithium (patient error, incorrect prescription)
- Management of lithium toxicity is done as follows:
  - Immediate cessation of lithium therapy (not waiting for result of serum lithium test)
  - Admission to hospital (usually to an acute medical ward)
  - Ensuring good hydration (unless in renal failure) as lithium is excreted via the kidney
  - Monitoring serum lithium, electrolytes, creatinine and eGFR
  - Monitoring cardiac function (risk of arrhythmias) and giving anticonvulsants if epileptic seizures occur
  - Haemodialysis occasionally if required, but often necessary if serum lithium >3.0 mmol/L or renal failure

and electrolyte balance should be used with care and with careful monitoring.

NSAIDs inhibit prostaglandins and can therefore reduce sodium and lithium excretion. This may result in lithium toxicity and patients need to be warned about use of NSAIDs, as these can be obtained over the counter without medical prescription. Paracetamol is safe.

There are reports of interactions between lithium and carbamazepine, haloperidol, digoxin and verapamil, resulting in a variety of neurotoxic and cardiotoxic effects. These are not common interactions but clearly indicate a need for caution if such drug combinations are unavoidable. Prolonged apnoea has been reported when patients on lithium were given succinylcholine or pancuronium during anaesthesia.

Anticonvulsants

Valproate is a generic term used to describe different formulations of valproic acid. While the particular formulation prescribed may not matter, it is important that the same formulation be used consistently for any particular patient as the dose levels used to achieve the necessary plasma concentration of valproate (between 50 and 125 μg/mL) differ between formulations. Valproate is a little less effective than lithium in the prophylaxis of bipolar disorder, but it will work for some of those who do not respond to lithium and its adverse effect profile may be more suitable for some patients (e.g. those with persistent tremor or who develop low eGFR on lithium). There is a significant risk of fetal abnormalities. It should be avoided in females during childbearing years unless in exceptional situations and with clearly effective contraception.

The principal adverse effects are nausea, weight gain, hair loss, thrombocytopenia and, for 5%–10% of patients, a non-pathological elevation of blood alanine aminotransferase (ALT) levels. However, a few patients may develop significant, potentially fatal, hepatocellular damage and jaundice. This can occur without elevation of ALT or with elevation of a wider pattern of enzymes. Some patients also develop hyperammonaemia, without evidence of hepatic damage, which can result in fluctuating confusion. Anaemia, leucopenia and increased bleeding time can occur. Hence, liver function should be monitored before and during the first 6 months of treatment and a full blood count before starting. Whether or not to monitor these on an annual basis will depend on the clinical situation.

The efficacy of carbamazepine is similar to that of valproate but its adverse effect profile makes it less useful. It is a potent inducer of hepatic enzymes and thus may affect the plasma concentrations of other medications, including antipsychotic drugs, In addition to the less severe adverse effects of nausea, sedation, skin rash, dizziness and ataxia it can cause aplastic anaemia, agranulocytosis and Stevens–Johnson syndrome in a few patients. There are risks of fetal abnormalities in pregnancy.

Lamotrigine is less effective than other mood stabilizers for prevention of episodes of mania/hypomania. However, it is more effective than valproate and carbamazepine for prevention of episodes of depression and can be of value for those patients for whom lithium is not adequately effective. Nausea, headache, skin rash and a hypersensitivity syndrome (which may progress to Stevens–Johnson syndrome) can occur. There are also risks of fetal abnormalities.

Anxiolytics and hypnotics

In the few situations where acute problems with anxiety or sleep disturbance require pharmacological treatment a benzodiazepine (BDZ), or related compound, is the most effective short-term treatment. However, because of risks of addiction and dependence, BDZs must not be used indiscriminately. If medication is required for patients with long-term anxiety disorders (Chapters 7 and 8) prescription of an SSRI is more appropriate. BDZs are inappropriate for long-term prescription in chronic disorders. The following section describes the mechanism of action of BDZs, important clinical aspects of BDZ use and the place of a number of other drugs, particularly antidepressants, in the treatment of anxiety.

Gamma-aminobutyric acid–BDZ receptor complex

BDZs act as agonists at the GABA–BDZ receptor complex. Gamma-aminobutyric acid (GABA) is the principal inhibitory neurotransmitter in the CNS and acts via $GABA_A$ receptors to increase the opening of chloride ion channels. An influx of Cl^- ions results in hyperpolarization of the postsynaptic neurone, making an action potential more difficult to achieve. $GABA_A$ receptors are linked to benzodiazepine receptors in the GABA–BDZ receptor complex, which is made up from five macromolecular subunits. These subunits are arranged around the chloride ion channel (Figure 18.1). Opening of the ion channel requires binding of two GABA molecules to the two GABA receptors. When a BDZ binds to its receptor site on the complex, the ion channel opens for a longer period. Thus, BDZs enhance the inhibitory effects of GABA. Points to note are: (1) BDZs act only to enhance the effect of GABA and will do nothing in the absence of GABA or if the GABA receptor is blocked; (2) the binding sites for GABA and BDZs are separate; and (3) the presence of a BDZ enhances the binding of GABA to its receptor.

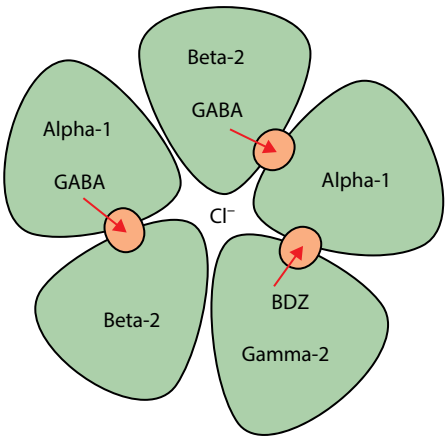

Figure 18.1 Diagrammatic representation as though looking down onto that part of the receptor complex that is on the outside of the neuronal membrane. It shows the arrangement of the five subunits (in this case two alpha-1, two beta-2 and one gamma-2) around a chloride ion (Cl^-) channel. The two GABA binding sites are at the interface between the alpha-1 and beta-2 subunits and the benzodiazepine (BDZ) binding site is at the interface between the alpha-1 and gamma-2 subunits. Opening of the Cl^- channel depends on the binding of two molecules of GABA. Binding of a BDZ molecule will increase the amount of time GABA maintains the opening of the ion channel.

(There are eight families of subunits with between one and six subtypes in each family. Each receptor complex contains subunits from either two or three families. Thus many variants of this receptor complex are possible though only some actually exist in human brain. Figure 18.1 shows a commonly occurring variant.)

Although the typical BDZ drugs, such as diazepam, act as full agonists at the receptor, having sedative, anxiolytic and anticonvulsant effects, other compounds may have different effects. (Full inverse agonists have the opposite effect, being pro-convulsant and anxiogenic. Such compounds are of experimental interest only.) Neutral competitive antagonists block the effects of both agonists and inverse agonists and have no intrinsic activity of their own. Flumazenil acts as such a drug in most situations and is thus used to reverse the effects of overdoses of BDZs. Partial agonists and partial inverse agonists occupy the receptor but have only some of the effects of the 'full' agonists/inverse agonists. An example is clonazepam which is a partial agonist with full anticonvulsant effects but little anxiolytic effect.

There is evidence for a number of endogenous GABA–BDZ receptor ligands in the CNS. However, these are only present in small amounts and do not have any clear physiological significance.

Older drugs

Before BDZs became available, barbiturates were the most widely used drugs for anxiety and sleep disturbance. (Barbiturates, first synthesized around 1900, were used as anaesthetic induction agents until the late 1990s.) They had potent anxiolytic and hypnotic effects, but also caused significant impairment of psychomotor function. This potency, the rapid development of tolerance to their effects and marked induction of their own metabolism led to escalation in dose in many patients as the beneficial effects would appear to wear off. Dependence occurred in a high proportion of patients. Barbiturates can also cause marked respiratory depression and are thus extremely dangerous in overdose. At normal doses, they act at a receptor site on the GABA–BDZ receptor complex to enhance the effects of GABA (like BDZs). However, in overdose they open the chloride ion channel independent of GABA, causing marked and prolonged neuronal depolarization and thus marked CNS depression. This does not occur in an overdose with BDZs (whose effects are always dependent on GABA) making them relatively much safer.

Benzodiazepines

The greater safety of BDZs in overdose and the fact that, when used properly (Box 18.6), they are less

prone than barbiturates to dependence, resulted in them rapidly supplanting the barbiturates following their introduction in the early 1960s. Death or serious morbidity from overdose of BDZs alone is rare and is only likely to occur if they are combined with other CNS depressants. Following over-prescription of BDZs during the 1960s and 1970s it was recognized that dependence could occur and prescribing has been reduced. However, they remain one of the most effective and safe treatments for symptoms of severe 'acute' anxiety.

Pharmacokinetics

Diazepam is the most widely used BDZ and its pharmacokinetics are summarized in Table 18.3. Absorption is good with oral use but is erratic and slow with intramuscular administration. Lorazepam, which has greater water solubility, is the BDZ recommended for intramuscular use. The principal active metabolite, desmethyldiazepam, has a very long elimination half-life ($t_{1/2}$), and gradual accumulation of active metabolites is one of the main reasons for many of these drugs having 'hangover' effects throughout the following day when given as hypnotics. The elimination half-life of diazepam is age dependent, resulting in greater hangover effects in older people. Some BDZs undergo Phase I metabolism, which results in production of active metabolites, and some only Phase II metabolism, resulting in inactivation. This and other pharmacokinetic properties of some of the commonly used BDZs are summarized in Table 18.4.

Physiological and adverse effects

The relative potencies of different BDZs as agonists at the GABA–BDZ receptor complex correlate with their potencies as anxiolytics and hypnotics. The inhibitory effects in the brain then cause the anti-anxiety, sedative and anticonvulsant effects. At normal therapeutic doses, BDZs have little effect on cardiovascular, respiratory or autonomic function. Respiratory depression and reduction in systolic blood pressure may occur, but principally with intravenous

Table 18.3 Pharmacokinetic properties of diazepam

Bioavailability	Almost complete with oral dosing
Peak concentration	30–90 minutes
Renal excretion	Negligible for unchanged drug
Elimination half-life	Young adults: 20 hours
	Elderly people: 30–100 hours
Hepatic metabolism	Active metabolite:
	Desmethyldiazepam: half-life 30–90 hours

Table 18.4 Comparison of the pharmacological properties of common anxiolytics and hypnotics

Drug	Absorption	Metabolic phases	Parent drug $t_{1/2}$ (h)	Important active metabolites $t_{1/2}$ (h)
Diazepam	Rapid	I+II	20–100	30–90
Chlordiazepoxide	Intermediate	I+II	5–30	30–90
Lorazepam	Intermediate	II only	10–20	None
Nitrazepam	Intermediate	I+II	24	30–90
Temazepam	Slow	II only	10	None
Zaleplon	Rapid	II only	1	None
Zolpidem	Rapid	II only	2.5	None
Zopiclone	Rapid	I+II	3–6	3–6

Note: $t_{1/2}$, elimination half-life.

Table 18.5 Adverse effects of benzodiazepines

Common	Occasional	Rare
Drowsiness	Ataxia	Amnesia
Dizziness	Headache	Restlessness
Psychomotor impairment	Reduced blood pressure	Skin rash
	Dry mouth	
	Blurred vision	
	Gastrointestinal upset	

administration or overdose. Leucopenia and eosinophilia are rare. In the 1980s there was a suggestion of increased risk of breast cancer but a large case–control study refuted this.

The more common adverse effects are listed in Table 18.5. Sedation is common and occurs with all BDZs. Tolerance to this effect generally occurs. Psychomotor impairment is related to sedation and also impairment of central information-processing ability. Research is equivocal on whether or not tolerance develops over time to these impairments. The effects of BDZs on memory were first noted in 1965: when used as premedication before general anaesthesia, it was possible to demonstrate amnesia for new information learnt by patients just before surgery. This appears to be an anterograde amnesia, with no impairment of previously stored information. Sedation also plays a part in this.

It seems likely that different variants of the GABA–BDZ receptor complex may mediate different actions of these drugs. The different variants have different anatomical distributions in the brain, but may overlap in different brain regions. However, the currently available BDZ drugs are not particularly specific for individual receptor variants and therefore influence a number of functions.

Tolerance and dependence

The phenomena of tolerance and dependence are perhaps best illustrated by the effects of BDZs on rapid eye movement (REM) sleep. In the first week of taking BDZ hypnotics, REM sleep is suppressed from around 25% to around 10% of total sleep time. Over the following 14 days, adaptation occurs (due to resetting of receptor sensitivity) and REM sleep returns to around 25% of total sleep time. Thus, tolerance has developed to the suppression of REM sleep. However, if the BDZ is now suddenly stopped a rebound increase in REM sleep, to around 40% of total sleep time, occurs because the receptor has reset its sensitivity to GABA. This results in increased awakenings through the night and thus a tendency for the patient to restart and continue the BDZ believing their original sleep problem remains, i.e. the patient has developed a degree of dependence on the drug. If the patient remains off the drug, the receptor sensitivity gradually returns to normal and the REM sleep percentage returns to normal over about 6 weeks.

Although tolerance appears to develop to the effects of BDZs on REM sleep, it is not clear that it occurs for the hypnotic, or sleep-inducing, effects of these drugs. There is certainly evidence, using subjective measures, of effectiveness being maintained for up to 6 months. With regard to the other effects of BDZs, there is evidence from human and laboratory animal studies for the development of tolerance to the sedative, muscle relaxant and anticonvulsant properties. Evidence is equivocal with regard to tolerance to the anxiolytic and psychomotor effects. However, some patients with anxiety disorders will begin to escalate their doses over time because tolerance has developed to the anxiolytic effects and they thus become dependent on the drug.

Withdrawal syndrome

In the late 1970s, it also became clear that a withdrawal syndrome occurred after sudden cessation of normal therapeutic doses of BDZs used for 6 weeks or more and not just following prolonged use of high doses. The appearance of withdrawal symptoms will often lead to the patient recommencing the drug, suggesting that the patent has become dependent on/addicted to the BDZ. The symptoms of this are listed (Table 18.6), the most common being symptoms similar to those of

Table 18.6 Benzodiazepine withdrawal symptoms

Anxiety type	Disturbance of perception
Anxiety	Hypersensitivity to stimuli
Tremor	Abnormal bodily sensations
Dysphoria	Abnormal sense of movement/body sway
Sleep disturbance	Depersonalization
Muscle pains	Visual disturbance
Headache	**Severe but rare**
Nausea, anorexia	Paranoid psychosis
Sweating	Depressive episode
Fatigue	Seizures

the anxiety disorder for which the drug was probably first prescribed. Thus, withdrawal of a BDZ should be gradual, particularly if the person has been on it for more than 4 weeks. A dose reduction by 25% every 1–2 weeks is recommended for very long-term users. A beta-adrenoceptor antagonist, e.g. propranolol, may reduce the intensity of the symptoms of withdrawal but does not actually improve success in coming off and remaining off BDZs.

During the 1960s and 1970s BDZs were very widely, and almost indiscriminately, prescribed (31 million general practitioner [GP] scripts per year across England and Wales in 1979). In the early 1980s, this evidence that dependence and addiction could occur, even with what was at that time 'normal' use of BDZs, led to a campaign to ensure more careful use and around a 50% drop in prescribing. Box 18.6 provides guidelines for their prescription based around those in the British National Formulary.

Clinical use

BDZs are used where moderate to severe anxiety symptoms arise due to acute stress, but should only be considered if symptoms are disabling in terms of daily functioning. In such situations, their use should be short-term and preferably for no longer than 2 weeks. Similarly, they are also used for acute exacerbations in patients presenting with generalized anxiety disorder (GAD). This is often a fluctuating condition with exacerbations at times of stress and chronic, milder symptoms on a longer term basis. Use of BDZs for GAD should only be for short periods, maximally 4 weeks, with the dose tailored to the severity of symptoms. Almost inevitably, some of these patients use BDZs chronically with risk of dose escalation over time. BDZs can also be useful as a short-term adjuvant to cognitive behaviour therapy for phobias but are of limited use for panic disorder and obsessive-compulsive disorder (OCD), except when patients are very acutely anxious.

BDZs are commonly used to assist withdrawal from alcohol, where a long elimination half-life drug is best. Chlordiazepoxide is the most widely used. In acute psychotic states short-term use of a high-potency drug, such as lorazepam, can be helpful in managing acute anxiety, agitation or aggression (Chapter 19).

Box 18.6 Prescribing guidelines for benzodiazepines

- Where possible, identify causes of anxiety or insomnia and treat these appropriately.
- Benzodiazepines are indicated for the 'short-term' relief of anxiety that is severe or disabling or causing the patient unacceptable distress. The use of benzodiazepines to treat short-term mild anxiety is inappropriate.
- Benzodiazepines should only be used to treat insomnia when it is severe, disabling or causing the patient extreme distress.
- Use the lowest effective dose.
- Ideally only prescribe for 2 weeks and at most 4 weeks.
- Avoid 'repeat' prescriptions as far as possible.
- Warn patients about the possibility of dependence.

Hypnotics

Insomnia (difficulty in initiating or maintaining sleep) is reported by 20%–40% of the population each year and is more common in females and the elderly. However, only 15% of those affected may actually require treatment beyond alteration of lifestyle, nighttime routine or reduction in use of alcohol or caffeine. Hypnotics are drugs that help to induce sleep. BDZs are useful in the treatment of insomnia, particularly for initial insomnia, when patients have difficulty falling asleep. As in the treatment of anxiety, these should

be used only for short periods, as prolonged use will result in dependence and difficulties in withdrawal, although it is relatively rare for patients to escalate the dose of hypnotic they use. Since 1980, prescribing of hypnotics has been stable at around 12 million scripts per year across England and Wales.

They will generally assist induction of sleep within 30–60 minutes. Some of the original BDZ hypnotics had active metabolites with long elimination half-lives that caused significant 'hangover' effects the next day. However, some have no active metabolites (e.g. temazepam) and thus have advantages.

During the last 20 years several new hypnotics have become available (zaleplon, zolpidem and zopiclone) that are not chemically benzodiazepines but which nevertheless act via the GABA–BDZ receptor complex. They generally have shorter elimination half-lives (Table 18.4), reduced risk of tolerance and dependence, and reduced psychomotor and hangover effects and are thus now the most usually prescribed hypnotic agents. Induction of sleep occurs approximately 20–30 minutes sooner than with placebo. A NICE technology appraisal concluded that variation in the clinical trial methods used to compare these drugs with standard BDZ hypnotics and with each other made it impossible to say that one was better than another or even better than a short half-life BDZ. There is some evidence to suggest that zopiclone may carry greater risk of dependence. Some patients find that the very short half-life of zaleplon (1 hour) results in a rebound awakening in the early morning as the drug is rapidly eliminated from plasma.

One important issue to be aware of is that older patients prescribed hypnotics have an increased frequency of falls and fractured neck of femur and hypnotics should be avoided in the elderly unless absolutely necessary.

Flumazenil

Flumazenil is a competitive antagonist of BDZs at the GABA–BDZ receptor and is used to reverse the CNS depressant effects of BDZs taken in overdose. It can only be given intravenously and has a short elimination half-life of 1 hour. Thus, its action to counteract the potentially life-threatening effects of an overdose may well wear off before there is a large fall in the plasma level of the BDZ taken in overdose. It is thus important to maintain the patient under observation until it is likely the drug taken in overdose has been washed out.

Other drugs used for anxiety

Antidepressant drugs, of all types, have been subject to clinical trials in anxiety disorders. Beneficial effects are thought to be mainly due to their actions on the 5-HT systems with possibly some contribution from effects on the NA systems. Those antidepressants acting principally via the 5-HT system seem to be particularly useful for the symptoms of OCD. SSRI antidepressants have become the medication of first choice for patients with persistent anxiety disorders (Chapter 7). Not all SSRIs have been subject to clinical trials for all types of anxiety disorders, but there is clinical trial evidence for at least one of the various SSRIs for most of the individual types of anxiety disorder. The most consistent evidence is for benefits in GAD, panic disorder and OCD. There are also successful clinical trials for NARIs in some forms of anxiety disorder

The advantages of SSRIs over BDZs are that they can be maintained long-term without problems of tolerance and dependence and are safer in overdose. Compared with TCAs and MAOIs they have a better adverse event profile and again are safer in overdose. However, clinical trials have shown benefit for TCAs in GAD and, specifically for imipramine, in panic disorder. Phenelzine, an MAOI, is effective in panic disorder.

During the first 7–10 days of treatment with an SSRI some patients with anxiety disorders, and particularly with panic disorder, may experience a worsening of symptoms before sustained clinical improvement occurs. This is probably due to the initial increase in availability of 5-HT before longer term neurotransmitter receptor adaptations occur. Patients should be warned about this.

In the late 1960s, beta-adrenoceptor antagonists, particularly propranolol and atenolol, were shown to be effective for the physiological symptoms of anxiety, particularly in situational anxiety and GAD. It is generally thought that their effectiveness is due to a primary reduction in peripheral symptoms, such as tremor and tachycardia, and that reduction in the unpleasant experience of these symptoms then reduced 'negative' feedback to the individual making it easier for them to manage the psychological aspects.

Buspirone, an azaspirodecanedione, an agonist at 5-HT_{1A} receptors, also has anxiolytic effects. Initially it probably has no net effect as it acts to stimulate postsynaptic 5-HT_{1A} receptors but also to reduce 5-HT release through its effect on cell body 5-HT_{1A} receptors. However, over 2–3 weeks the effect on the cell body receptors wears off and the net effect becomes an increase in 5-HT transmission. It is less potent than the BDZs and the effects take up to 3 weeks to become evident. There is high first-pass metabolism and a considerable proportion of the effect is due to a metabolite (1-PP). The principal adverse effects of buspirone are nausea, gastrointestinal upset and headache. Because of its low potency, it is not widely used.

Pregabalin binds to a subunit of voltage dependent calcium channels and reduces the release of a number of neurotransmitters. It was originally introduced for the treatment of epilepsy and neuropathic pain. Subsequently, clinical trials have shown its efficacy for treatment of GAD, for which it was approved in 2007. Sedation, dizziness and weight gain are some of the more common adverse effects. There is no hepatic metabolism and it is excreted unchanged via the kidney.

Antipsychotics

These drugs are used for the treatment of psychosis in whatever context: schizophrenia, mania, psychotic depression, drug-induced psychosis and other organic disorder-induced psychoses (e.g. through Huntington's disease or toxic confusional state). In schizophrenia, it is only the 'positive' symptoms which show consistent, significant response to antipsychotics. Antipsychotics are useful for treatment of agitation in the context of severe depressive disorder. There is some evidence to support the use of second-generation drugs, such as risperidone, as adjuncts to antidepressants in a few patients with severe, treatment-resistant anxiety but this use should be reserved for care supervised by a psychiatrist. Sometimes very challenging or aggressive behaviour can occur in children, young people and adults with either autism or learning disability. If psychological or social interventions have not been effective, or if the behaviour(s) are very severe, use of antipsychotics may be effective but must be initiated at low doses and supervised by a psychiatrist. Antipsychotics are never indicated for the core features of autism itself.

They have often been prescribed for the management of agitation occurring in a variety of situations in elderly people. However, there is evidence that all antipsychotics in elderly people may increase the risk of vascular events such as stroke. Such situations should be approached in other ways (see the section 'Dementia – Management' in Chapter 12). There are only a few situations for people with dementia where use of antipsychotics may be appropriate (e.g. where acute psychosis is the cause of the agitation) but, even in these, use should be for less than 3 months. They should be completely avoided in Lewy body dementia which they will make worse.

Mechanism of action

All effective antipsychotic drugs antagonize DA receptors (principally the D_2 subtype) and this led to considerable investigation into possible abnormalities of DA systems in schizophrenia (Chapter 2). In vivo functional brain imaging does not support earlier claims, from studies of post-mortem brain tissue, of an increase in DA receptor density, but does support a subtle alteration in DA turnover in the brain. There is also more recently consistent evidence of a (possibly) primary disturbance in glutamate function, which has secondary effects on the function of GABA and DA systems in the brain. Hence, even if a DA disturbance is not primary it is still an important way to manipulate many of the symptoms. This more recent knowledge is opening the way to the development of new drugs for the treatment of schizophrenia. There is also evidence that 5-HT systems have a part to play in some aspects of schizophrenia, which may be partly why some of the second-generation antipsychotic drugs have benefits over the first-generation drugs.

Positron emission tomography studies of D_2 receptor occupancy in patients demonstrate that clinical response is normally achieved with 50%–70% occupancy of these by the antipsychotic drug. There is no additional clinical benefit to >70% occupancy, but at >70% occupancy extrapyramidal side effects (EPSE) become much more prevalent (Box 18.7). Clozapine and the available second-generation drugs rarely achieve occupancy greater than 70%, even at maximum doses, whereas this is relatively easy to achieve with the first-generation drugs

at medium to high doses, making them more likely to induce EPSE.

Classification of antipsychotics

The first antipsychotic drug was chlorpromazine, introduced in 1952. This and others are listed in Table 18.7. Chlorpromazine is moderately potent in the antagonism of D_1 and D_2 types of DA receptors and also antagonizes a wide variety of other receptors – serotonergic, adrenergic, cholinergic, histaminergic – resulting in a wide variety of adverse effects. Other first-generation (often called 'typical') antipsychotics also had a wide spectrum of effects beyond the desired antipsychotic effect. Among the most problematic adverse effects of these drugs were the EPSE (Box 18.7) related to DA receptor antagonism in the caudate–putamen region: drug-induced pseudo-parkinsonism, acute dystonia, tardive dyskinesia and akathisia. Designing of drugs to eliminate these problems is difficult because the antipsychotic effect is primarily due to the DA receptor antagonism as well.

However, there are now a number of second-generation (previously called 'atypical') antipsychotics which have a much reduced propensity to induce EPSE. These drugs differ from the first-generation antipsychotics in two main ways: (1) most are less potent in their antagonism of DA receptors (see 'Mechanism of action', above) and (2) they have a high ratio of 5-HT_2 receptor antagonism to DA receptor antagonism (often referred to as a high 5-HT_2:D_2 ratio). Both (a) and (b) seem to contribute to the reduction in EPSE. In addition, these properties may also result in patients having fewer 'negative' symptoms when taking these drugs. (Some aspects of what are sometimes clinically assessed as 'negative' symptoms, using rating scales in clinical trials, probably reflect aspects of side-effects, such as psychomotor slowing and akinesia as part of EPSE, rather than true 'negative' symptoms.) The second-generation drugs differ from each other in chemical structure and in their propensities to antagonize other neurotransmitter receptors. Thus, they have profiles of adverse effects that are sometimes quite individual to a specific drug and cannot usefully be classified into various subgroups.

Around 30% of patients with schizophrenia show either no, or very poor response, to the antipsychotic drugs described earlier or may have unacceptable adverse effects. Around half of these patients will achieve a better response with clozapine (see Table 18.7). This antipsychotic carries a 5%–10% risk of neutropenia and 1% cent risk of agranulocytosis. Hence, its use is restricted to patients who are treatment resistant to other antipsychotics. It is mandatory to monitor total white cell and neutrophil counts in

Table 18.7 Classification of antipsychotic drugs with examples (for an exhaustive list see the BNF)

Drug group	Examples (in alphabetical order)
First-generation	
Phenothiazines	Chlorpromazine, trifluoperazine, fluphenazine
Thioxanthines	Flupenthixol, zuclopenthixol
Butyrophenones	Haloperidol, droperidol
Diphenylbutylpiperidines	Pimozide
Substituted benzamides	Sulpiride
Second-generation	Amisulpride, aripiprazole, lurasidone, olanzapine, paliperidone, quetiapine, risperidone, sertindole, zotepine, asenapine (only licensed for treatment of mania), ziprasidone (available in the United States and many European countries but not the United Kingdom)
Clozapine	Considered separately because it is more effective than all other antipsychotic drugs for treatment-resistant patients. It is not regarded as a second-generation drug but has been the prototype for the development of many of the second-generation drugs. It has modest dopamine receptor blocking effects but a very wide range of other pharmacological effects. The key pharmacology conferring its unique clinical effects is not clear.

patients on clozapine, initially weekly and then every 4 weeks in the long term.

Clinical use

Meta-analyses comparing the efficacy of first- and second-generation antipsychotic drugs find no major differences between them for the main symptoms of schizophrenia. Hence, decisions about first-line treatment for an acute episode of psychosis depend largely on the patient in relation to the adverse effect profile of the available medications and any pre-existing physical health problems. Second-generation oral medications are most commonly chosen. The adverse event profile of these usually allows a rapid titration to an effective dose. For first-generation drugs, the problems of sedation, anticholinergic adverse effects and the development of EPSE can limit the speed of dose increase and limit the final dosage.

A clinical response will begin to be seen within the first week and a poor response by 2 weeks is an indication that the particular medication may be ineffective for that patient. Lack of response by 4 weeks should trigger a change of medication. Around 50%–60% of patients will have some degree of response to either the first or second antipsychotic medication prescribed. There is no evidence that giving very high doses of antipsychotic drugs (above BNF recommended upper limits) will improve the response rate but it will result in more side-effects. Over the course of the first 3–4 months of treatment around 30% of patients will be found to be treatment non-responsive or poorly responsive (often termed 'treatment resistant'). All these patients should be offered a trial of treatment with clozapine unless there is some very specific reason not to do so (e.g. a previous history of white blood cell disorders). Approximately 50% of these patients will respond to clozapine. Combining two different antipsychotics for a treatment-resistant patient is only occasionally effective and such polypharmacy should be avoided if at all possible.

Long-term antipsychotic treatment, in patients with established schizophrenia, considerably reduces rates of relapse. For example, at 1 year on placebo around 60%–80% will relapse compared with 20%–40% of the patients on active treatment. However, longer term outcomes vary considerably with only around 25%–30% of patents remaining relapse free at 5 years. Up to 50% of patients will not adhere fully to their treatment regime. Where adherence is a problem the medication is often given in one of the slow-release depot or long-acting injectable formulations, usually administered at a frequency between 1 to 6 weeks, depending on the clinical situation and medication being used.

Pharmacokinetics

Antipsychotics are generally rapidly absorbed from the gut reaching peak serum concentrations in 2–4 hours. The elimination half-lives of most of the first-generation drugs are quite variable between individuals but are generally quite long – in the region of 20–30 hours but up to 100 hours in some individuals. Many have active metabolites, which have much longer elimination half-lives, that are usually of lower potency at the DA receptor but contribute to some of the effects of the drug. Some, such as chlorpromazine, have a number of active metabolites. This makes meaningful interpretation of serum concentrations difficult. It is almost impossible to demonstrate other than very broad relationships between serum level and clinical response. Therefore, serum level monitoring is of no value in routine clinical practice. The second-generation drugs are also readily absorbed. Quetiapine and ziprasidone have short elimination half-lives (<10 hours) but the others have half-lives in the region of 20–30 hours.

Active metabolites generally do not contribute significantly to the therapeutic action of these drugs except for chlorpromazine and risperidone.

Adverse effects and toxicity

The number of different drugs and variety of adverse effects makes it impossible to comprehensively describe these. Table 18.8 lists the more important effects and the drugs most likely to cause them. Note that all of the antipsychotic drugs can give rise to virtually all of these adverse effects, but particular adverse effects are more common with certain drugs.

Most antipsychotics can cause some prolongation of QT_c interval on ECGs and some carry specific recommendations regarding ECG monitoring. Although it can be difficult in clinical practice, NICE recommends that an ECG should be recorded before

Table 18.8 Adverse effects of antipsychotic drugs

Adverse effect	Drugs most likely to cause the effect
Pseudo-parkinsonism, dystonia, and tardive dyskinesia	First-generation antipsychotics, especially haloperidol
Akathisia (an unpleasant syndrome of mental and motor restlessness)	First-generation antipsychotics, especially haloperidol Aripiprazole, lurasidone and clozapine
Sedation	First-generation antipsychotics Clozapine, olanzapine, quetiapine, zotepine
Anticholinergic effects (dry mouth, blurred vision, constipation, urinary retention)	First-generation antipsychotics
Considerable weight gain	Chlorpromazine, thioridazine Clozapine, olanzapine, zotepine
Little weight gain and occasionally weight loss	Aripiprazole, haloperidol, lurasidone, ziprasidone (The other antipsychotics not mentioned here often cause a small or medium degree of weight gain.)
Postural hypotension	Chlorpromazine Clozapine, quetiapine
Prolongation of QTc interval on ECGs (thought this may indicate risk of arrhythmia and sudden death)	All may cause some prolongation but for most it is <10 ms The following are likely to cause >15 ms: thioridazine*, pimozide, sertindole* and ziprasidone (*No longer in routine use because of this)
Elevated serum prolactin (PRL), galactorrhoea and altered menstrual cycle (PRL may sometimes be increased tenfold)	Varying degrees with all antipsychotics except olanzapine and clozapine. Most marked with sulpiride.
Sexual dysfunction	Can occur with most. Reported least in clinical trials with olanzapine and quetiapine.
Cholestatic jaundice, skin pigmentation, skin rashes, photosensitivity	Principally seen with phenothiazines and mainly chlorpromazine

initiation of an antipsychotic drug in the following cases:

- If it is specified in the summary of product characteristics (SPC)
- A physical examination has identified specific cardiovascular risk (such as diagnosis of high blood pressure)
- There is a personal history of cardiovascular disease (or a family history before the age of 60 years)
- The service user is being admitted as an inpatient

A rare, but potentially fatal, idiosyncratic adverse effect is neuroleptic malignant syndrome. This can occur with any antipsychotic drug. The symptoms are rigidity, hyperthermia, autonomic lability and reduced level of consciousness. Massively elevated creatine kinase is usually found. Prior to 1984, the mortality rate was around 20% in unrecognized cases but improved early recognition has considerably reduced this. The cause of this is unknown. Management of this is described in Chapter 19.

As mentioned in Chapter 9, patients with schizophrenia have a greater risk of physical health problems. Life expectancy is reduced by 20%, and 60% of this excess mortality is due to cardiovascular disease. Risk for this is largely driven by smoking (over 50% of patients), as well as excessive weight gain and obesity, development of dyslipidaemia and, in a high proportion of cases compared with the general population, development of diabetes. An important, but not the only, factor in the development of these metabolic problems is the effect of many antipsychotic drugs on appetite. For this reason, when patients are commenced on an antipsychotic drug, especially if for the first time, their weight and BMI should be monitored. Patients can gain over 5% of

their initial body weight even in the first 12 weeks of treatment. If rapid weight gain is occurring, consideration should be given to a weight management programme and possibly a switch to a drug less likely to induce weight gain.

Extrapyramidal side effects

EPSE include disturbance of motor function and were extremely common with the first-generation antipsychotic drugs (pseudo-parkinsonism affected 40% of patients and tardive dyskinesia 60% of those on long-term treatment). Patients found them particularly unpleasant and they were a common cause of poor compliance with treatment.

One of these, acute dystonia is a particularly painful and unpleasant condition and patients may often present with it as an emergency to an emergency department or a GP. It can occur within a few days of commencing treatment, and particularly on the first occasion a patient is prescribed an antipsychotic medication.

VIDEO 18.1

Acute dystonia – https://vimeo.com/17084871

Drug interactions

Pharmacokinetic

Antacids reduce absorption and enzyme-inducing drugs may decrease serum levels of antipsychotics. The enzyme inhibitors cimetidine and propranolol

Box 18.7 Extrapyramidal side effects and their management

Extrapyramidal side effects (EPSE) may occur secondary to antipsychotic medication (see Table 18.8) and are related in different ways to DA receptor antagonism by the antipsychotic drug in the caudate putamen region (though not entirely clear for TD). As it is usually not possible to stop the antipsychotic other approaches have to be used.

Symptom and time course of onset	Key features	Management
Acute dystonia Occurs within a few days (More common in young males)	Muscle spasms lasting from minutes to hours. These are often painful and functionally disabling. Spasmodic torticollis (spasm of neck muscles). Facial grimacing and trismus (spasm of muscles of the face and jaw). Oculogyric crisis (sustained upward deviation of the eyes). Opisthotonous (spasm in muscles of the trunk).	Anticholinergic drugs. In acute situations these may require to be given IM or IV, either of which require careful post-injection monitoring.
Pseudo-parkinsonism May take 3+ weeks to appear	Increased muscle tone (lead-pipe or cog-wheel). Tremor, as seen in Parkinson's disease. Akinesia (slowness and poverty of movement).	Anticholinergic drugs.
Akathisia May occur within a few days or after many weeks	An inner feeling of restlessness and agitation. Motor restlessness (e.g. constantly shuffling feet; cannot sit still). 'Dysphoric' mood (mixture of anxiety/frustration/low mood).	Anticholinergics often help. Beta-blockers or low dose diazepam may also work.
Tardive dyskinesia (TD) Takes months or years to present	Choreo-athetoid movements (irregular contractions and writhing movements combined). Oro-lingual-facial movements most common.	Reduce dose of antipsychotic or switch to different drug. Made *worse* by anticholinergics.

both increase serum levels. There can be competition for metabolic pathways by some TCAs and SSRIs (especially fluoxetine), which may increase serum levels. Omeprazole can reduce clozapine levels.

Central nervous system

Phenothiazine-type antipsychotics will potentiate the CNS depressant action of many drugs, including opiates and alcohol, and will potentiate the effects of general anaesthetic agents. All antipsychotics will antagonize the effect of L-dopa in Parkinson's disease, making management of patients with psychosis and Parkinson's disease difficult.

Cardiovascular

Many antipsychotics are alpha-adrenergic receptor antagonists and may thus enhance the effect of antihypertensives, including ACE inhibitors. In contrast, the effects of the older adrenergic neurone blocking type of antihypertensive drug may be antagonized. Combination of antipsychotics with other medications that may prolong QT_c interval should be avoided if possible.

Drugs for attention deficit hyperactivity disorder (ADHD)

Available drugs and mechanisms of action

The medications currently licensed in the United Kingdom are simply grouped according to their mechanisms of action:

- Monoamine releasing agents (increase release of DA and NA): D-amphetamine and lisdexamfetamine, which is a pro-drug metabolized to active dexamfetamine.
- Monoamine (principally DA and NA) reuptake inhibitors: methylphenidate
- Selective NA reuptake inhibitor: atomoxetine

Current evidence suggests that their therapeutic benefits (improved cognitive function and attention; reduced distractibility and hyperactivity) occur through their effects on brain NA systems rather than DA systems. Effects on both the systems mediate many of the potential adverse effects.

Adverse effects common to all of these drugs are: nausea, tachycardia, effects on blood pressure, reduced appetite and effects on growth. D-amphetamine, methylphenidate and lisdexamfetamine carry risks of abuse, which are thought to be least with the last of these, and may cause insomnia. Atomoxetine may cause somnolence but does not have potential for abuse.

Clinical use

For children and adolescents with ADHD, the NICE guideline recommends drug treatment for those with severe ADHD or moderately severe ADHD non-responsive to psychological treatments. Treatment should continue for as long as it seems to confer benefit, which means that some of these patients will continue on their medication into adulthood. For patients first diagnosed as adults, medication is recommended as first-line treatment. For both children and adults, methylphenidate is usually the first choice and atomoxetine used if the first is found ineffective. Slow release formulations are preferred, if available, and the dose should be titrated up gradually over 4–6 weeks.

Pre-treatment assessment requires a full physical examination, including height, weight, heart rate, blood pressure and exercise syncope. If there is a history of cardiac disease, or family history of cardiac disease or sudden death of a young person, then an ECG should be recorded. There should also be an assessment of any risks of abuse of the medication by the patient, or other family members, as this can influence choice of drug. Growth and cardiac parameters should be monitored during treatment. Parents and children should be warned of the possibility of increased suicidal thinking and hepatic problems with atomoxetine.

Psychotropic drugs and vehicle driving

All of the drugs described earlier, with the exception of lithium, can cause sedation. All, including lithium, may impair concentration or psychomotor function and thus impair the ability of someone to drive a vehicle, operate complex machinery or carry out complex tasks. These effects will be more common with those drugs that cause the most sedation and are something

that all patients commencing such drugs should be warned about. For many patients and most drugs, these effects are likely to wear off over a few weeks but patients should be warned that if they feel sedated or that their concentration is impaired, they should not drive, use machinery or attempt complex tasks.

An acute mental illness can also by itself affect the aforementioned abilities. Thus, the aforesaid considerations must be added to the general guidance regarding driving and mental illness (https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/457961/aagv1.pdf). The guidance indicates that there must be a 3-month period of stability for those recovering from an episode of psychosis or mania/hypomania and an unspecified period of stability depending on medical reports after an episode of depression.

Since April 2015 new regulations regarding driving and the use of various controlled drugs came into force (https://www.gov.uk/government/collections/drug-driving#information-for-healthcare-professionals) – this website has links through to a further useful document, 'Guidance for healthcare professionals on drug driving'. The list of controlled drugs includes a number of 'medicinal' drugs, which includes some BDZs, as well as other 'illegal' drugs and provides a table of threshold blood concentrations above which driving would be considered an offence. However, if a patient has been prescribed one of these drugs by a doctor and is taking it according to instructions, then they are allowed to drive, irrespective of blood concentration of the drug, as long as their driving is not impaired by the drug. In other words, the patient has a 'medical defence' that allows them to drive when taking one of these drugs as long as they are not impaired by it – but this does place an onus on the patient to be sure they are not impaired. One effect of this regulation is that patients are advised to carry documentary evidence that they have been prescribed the drug for a recognized medical condition and thus they may seek such documentation from the prescriber.

Patients who have had a course of ECT must also be advised not to drive during a course of ECT and for some weeks afterward. This is because the effects of anaesthesia (no driving for 48 hours after a general anaesthetic) and the cognitive effects of ECT may be significant for at least 2 weeks post-ECT and may remain for many patients for up to 6 weeks or even longer.

Electroconvulsive therapy

Although this is not a drug, it seems appropriate to include it here. Most students will probably be aware that its use has been controversial over the years. In part, this is related to a public perception that it was frequently given without anaesthesia and was misused in the rather frightening manner as depicted in films such as *One Flew Over the Cuckoo's Nest*. The development of safe, short-acting anaesthetic techniques during the 1940s and 1950s consigned the rather terrifying nature of the early approach to administration to history. Good clinical observation and the well-devised clinical trials of the 1970s and 1980s clearly defined the disorders for which ECT can be effective. It continues to be used for very severe depressive disorder, usually when resistant to drug treatment, catatonia and a very small number of patients with mania and acute exacerbation of schizophrenia who do not respond to antipsychotic drugs.

The idea that induction of a seizure might be useful derived from the observation that some patients with both psychotic illness and epilepsy often showed improvement in their psychosis after having a seizure. The use of convulsions was pioneered by Meduna in 1934, when he used chemical means (IM camphor) to induce seizures. This and other chemicals were unreliable in their effects and in 1938 Cerletti and colleagues developed electrical means – ECT. The lack of safe short-acting anaesthesia at that time meant this was given with only physical restraint of the patient to prevent injury. Use of general anaesthesia became routine during the 1950s. Two large clinical trials (random allocation, but not blinded) in the 1960s confirmed efficacy in depressive illness, and six placebo controlled, double-blind, random allocation studies between 1978 and 1985 further demonstrated this. The precise mode of action of ECT is not understood but it has a number of similar effects to antidepressant drugs on relevant neurotransmitter systems.

Administration of ECT

The patient must give written consent for ECT treatment and have had an explanation of the potential benefits, adverse effects and risks of not having the treatment. There are only very occasional situations where a patient is extremely unwell, lacks the capacity

to make a decision but is detained under the appropriate mental health legislation that ECT can be given against the patient's wishes. Such a decision always requires an independent second opinion. A link to the type of information that might be given to a patient can be found in the list of references.

Before administration of the electrical stimulus, the patient is anaesthetized. The prime reason for anaesthesia is to allow muscle relaxants to be used, usually suxamethonium chloride, in order to prevent injury from muscle contractions during the seizure. Good oxygenation of the patient immediately prior to the stimulus is important both for anaesthetic safety and to reduce the seizure threshold.

The electrical stimulus given needs to be at least double the patient's seizure threshold and a standard formula based on age and gender is generally used to determine this. Seizures are required to be at least 20 seconds in duration to be effective and usually between five and eight are required, given twice weekly. Failure to have an adequate seizure should be followed, if anaesthetic conditions permit, by one re-stimulation with an electrical stimulus 50% greater than that first used. However, if a seizure is prolonged (>60 seconds) then intravenous diazepam is used to abort the seizure.

The electrodes are first soaked in a suitable electrolyte solution to improve electrical contact with the skin. For bilateral ECT they are applied bilaterally in the fronto-temporal region, to an area of the scalp 4 cm above the mid-point of a line joining the external auditory meatus and the angle of the orbit, which should be dry and grease free. For unilateral ECT the electrodes are applied to the non-dominant hemisphere with the second electrode placed over the parietal region about 9 cm superior and posterior to the fronto-temporal electrode. Unilateral ECT causes less immediate post-ECT cognitive impairment but some studies suggest it may be less effective. The decision regarding which approach to use depends on whether there may be particular problems surrounding the short-term post-ECT impairment. If possible, BDZs should not be prescribed as part of the patient's medication regime as these will raise the seizure threshold and shorten seizures.

Adverse effects

ECT should be avoided if possible within 3 months of a myocardial infarction or a cerebrovascular accident. At least 30% of patients complain of headache for a few hours post-ECT. Figures for mortality reported in the 1970s suggest one death per 22,000 treatments. This is probably most often due to cardiac arrest following excessive vagal inhibition and is similar to mortality from general anaesthesia for minor surgery. There is no evidence of structural damage to the brain. Impairment of short-term memory may occur and two studies suggest this improves to normal over the 6 weeks following ECT. However, some patients do describe persistent memory difficulties that are difficult to disentangle from the effects of their depression.

Other physical treatments

Three other types of non-drug physical treatment are available and are described briefly as follows.

Transcranial magnetic stimulation

This is a technique for stimulating small areas of cerebral cortex by application of high intensity magnetic fields of brief duration. These changing fields may cause neuronal depolarization and also affect local cortical blood flow. Two different techniques exist for doing this (transcranial direct current stimulation and repetitive transcranial magnetic stimulation) and the placement of the coils providing the magnetic fields can vary between centres. Research is still being carried out regarding the value of this technique, which can be used as an alternative to ECT, for patients with depression who have not responded to drug treatment. The available clinical trials suggest it is beneficial for some patients but there is no clear consensus yet on the best way to deliver the treatment. Headache is the most common adverse effect but occasionally an epileptiform seizure can be induced.

Vagal nerve stimulation

This treatment was first introduced for some types of intractable epilepsy but has also been found useful for a small number of patients with more severe treatment-resistant depression. It is not clear how it works but afferent fibres of the vagal nerve project to the nucleus solitarius, which then has projections to a number of mid-brain structures, including the locus coeruleus, which controls aspects of brain NA function. A stimulus generator is implanted in a pouch in

the left upper chest and an electrode clamped to the left vagus nerve via an incision in the neck. A recent large trial shows that 27% of otherwise treatment-resistant patients respond and that at least half of these maintain this response. However, the effect may take up to 6 months to become evident.

Neurosurgery for mental disorder (sometimes called psychosurgery)

The use of neurosurgical intervention on the brain, for treatment of severe mental disorders, began in the 1930s at a time when there were no effective treatments. The procedure, then known as a leucotomy, involved dividing fronto-limbic connections via a burr hole in the temporal region of the skull. It was used for a wide variety of diagnoses where symptoms and associated behaviours were very severe or distressing, including schizophrenia, severe depression, OCD and severe, chronic anxiety. Adverse effects were common and severe, such as intellectual impairment, seizures, aggressive outbursts, loss of self-control and incontinence. Following initial enthusiasm, it was recognized that outcomes were often poor and its use declined by the late 1960s.

However, greater knowledge of brain circuitry and the function of specific pathways as well as improved neurosurgical techniques, using stereotactic instruments, led to some resurgence of interest in the 1970s. The technique was more or less exclusively used for patients with severe, intractable OCD and severe, intractable depression. The existence by then of more effective psychological and drug therapies for these conditions meant that patients referred to neurosurgery for mental disorder (NMD) had now had prior trials of a number of potentially effective treatments. A variety of different sites have been used for generating lesions but common ones involve the cingulate gyrus and the subcaudate area. Adverse effects still occur but are less common and generally less severe with the more sophisticated modern techniques.

Clearly, this is a controversial area of medicine. Every operation in England has to be approved by a panel from the Care Quality Commission or, in Scotland, the Mental Welfare Commission for Scotland. NMD patients must be over the age of 20 years and it is only carried out with the patient's informed consent. Only two centres in Cardiff and Dundee now perform this and less than 10 patients per year undergo NMD in the United Kingdom (only four in 2014–2015). Patients considered for NMD will generally have been ill for a period of 5 years (but at least 3 years) before referral to NMD. For those with depression they would have received trials of at least five different antidepressants or combinations of antidepressants, at least one course of ECT, one course of an appropriate psychological therapy and are likely to have had a trial of vagal nerve stimulation. Treatment criteria for patients with OCD are similar with respect to drug treatment and psychological treatment trials.

Summary

At the end of this chapter, you should be able to initiate appropriate psychopharmacological treatment for patients with mental illnesses. You should also understand some of the limitations of these treatments and in particular, the issues relating to their adverse effects, which quite often continue to be a limiting factor. Nevertheless, progress in our understanding of the neurochemistry of severe mental disorders and in our understanding of the actions of effective treatments has led steadily toward more useful drugs and better outcomes for patients. Though effective and safe use of psychotropic drugs is very important, and is essential knowledge for doctors on mental health teams, this is usually only effective within the context of the wider range of supports and services provided. Medication will not achieve maximal effectiveness unless patients are within a structure that encourages good adherence with treatment and within which they can then develop their lives once key symptoms are suppressed or abolished.

Further reading

Anderson IM, McAllister-Williams RH (2015). *Fundamentals of Clinical Psychopharmacology,* 4th edition. London: Taylor & Francis/CRC Press.

King DJ (2004). *College Seminars in Clinical Psychopharmacology*, 2nd edition. London: Gaskell.

NICE Website. NICE guidelines relating to many of the conditions mentioned in this chapter can be found through this website. Available at http://www.nice.org.uk

Royal College of Psychiatrists. Information about electroconvulsive therapy (ECT). Available at http://www.rcpsych.ac.uk/healthadvice/treatmentswellbeing/ect.aspx.

CHAPTER

19 Psychiatric emergencies

BRIAN LUNN AND RICHARD DAY

KEY CHAPTER FEATURES

- Deliberate self-harm
- Acute behavioural disturbance (including acute tranquillization)
- Delirium tremens (alcohol withdrawal delirium)
- Lithium toxicity
- Acute dystonia
- Neuroleptic malignant syndrome

Introduction

There are relatively few true psychiatric emergencies, but there are some important situations where prompt intervention is required. In this chapter, these will be discussed with an emphasis on recognition and basic management. These are also conditions that you may come across as doctors in other specialities, which is why we have included them here. In this chapter there will be some overlap with Chapters 12 and 18. However, to help your learning we have cross-referenced to reduce repetition but have also included information in this chapter to enable you to consider the particular issue in the context of an emergency.

Why is this relevant to you?

This chapter is more detailed since these are situations you are likely to be faced with and have to manage safely.

The two most common emergencies are deliberate self-harm (DSH) and acute behavioural disturbances, principally those where extreme emotional distress, acute agitation or threatening and/or actual aggressive behaviour are involved. Such behavioural disturbances may also occur in those who have self-harmed and sometimes various aspects of behavioural disturbance may occur in the same person all together. Patients with disturbed behaviour often have to be managed in the absence of a

clear diagnosis, because the very existence of the behavioural problems makes it difficult to establish a diagnosis unless they are already well known to services. Acute confusional states also often present as behavioural disturbances. Another group of emergencies are the potentially life-threatening unwanted effects of psychotropic medications that require prompt intervention: dystonia, neuroleptic malignant syndrome (NMS) and lithium toxicity. The longer term, less acute adverse effects are covered in Chapter 18. A further emergency that may require urgent psychiatric input is acute delirium. This is dealt with in Chapter 12. Of specific importance are acute withdrawal from alcohol and benzodiazepines, both of which will present to junior doctors on a regular basis and which are important to recognize early. As will become evident, good nursing care is an essential part of managing any of these emergencies.

Deliberate self-harm

Before we discuss self-harm and its management, it may be useful for you to consider what comes to mind when you think of this. There is good evidence that those presenting with self-harm often receive less than optimal care. There is no doubt that when dealing with other life-threatening emergencies, being asked to assess self-harm can feel frustrating. However, it is important to remember that the patient presenting may be distressed and lack effective strategies to manage at that time. A professional approach without judgement can have enormous benefits.

It is important to recognize at the outset that DSH is a behaviour rather than a diagnosis, and that the behaviour of DSH is influenced by a complex interplay of personal, psychological, health and illness, environmental and cultural factors. Greater understanding of these issues over the last 10–20 years has led to the terminology 'deliberate self-harm' being preferred over the previous commonly used terminologies 'parasuicide' and 'attempted suicide'. The significance of any act of DSH can only really be understood once you have tried to understand and integrate these different factors.

It is important to differentiate between the antecedents to and behaviours involved in DSH and suicide as they are often different and not part of a continuum, although there can be overlap. Some DSH is certainly an unsuccessful attempt at suicide. However, sometimes DSH unintentionally results in death, which was not an intended or anticipated outcome. It is important not to look at single demographic factors (such as gender or age) or characteristics of the attempt but to consider the picture as a whole. With suicide there generally tends to be a plan in place with the attempt having been planned. The patient anticipates that the action they take will lead to death. The attempt is made so that there is little, if any, chance that they will be discovered. There is often coexisting co-morbidity with mental illness and/or chronic physical health problems (especially painful conditions which impact negatively on quality of life). Elderly men recently widowed are at high risk of suicide but at low risk of DSH, whereas the reverse is true for young women.

There can be many other motives for an act of DSH. The commonest are as a form of escape, or even temporary distraction (e.g. from an intolerable situation), or an appeal (to mobilize support or express distress). However, it is preferable to avoid the term 'cry for help' as that tends to bring about unhelpful responses from health professionals.

Risk factors

DSH is a common phenomenon. UK rates are between 250 and 300 per 100,000. It is the commonest single reason for admission to hospital for women aged less than 65 years (in men aged less than 65 it comes second only to ischaemic heart disease). It must also be remembered that many acts of DSH do not come to medical attention at all. It is a behaviour that is most prevalent in young adults and becomes less common with advancing age. In the past there has been a clear female preponderance for DSH, but over recent years, in the United Kingdom there has been a significant increase in DSH among young men and the gender ratio is now almost equal. Risk factors are listed in Box 19.1. Only about 30% of DSH presentations are associated with a specific mental disorder, such as depressive disorders, adjustment disorders, personality disorders or drug and/or alcohol dependence.

Up to half of people who present with DSH have a history of previous DSH and up to one in five will engage in further DSH over the next year. There is also a significant minority (about one in seven) who have a history of more than five episodes of self-harm. This group of people who engage in repetitive DSH are

much more likely to have a diagnosis of personality disorder than people who only self-harm occasionally. Only a small minority of people who self-harm go on to commit suicide – about 1% in the year following an episode of self-harm and 2%–3% over 5 years.

Suicide

Suicide is much less common than DSH – the overall UK rate was 11.9 deaths per 100,000 in 2013. The male suicide rate was more than three times higher than the female rate, with 19.0 male deaths per 100,000 compared to 5.1 female deaths. It accounts for about 1% of all deaths and, after accidents, is the second most common cause of death in young men. The demographics for completed suicide are significantly different to those of nonfatal DSH. However, the single best indicator for those at risk of suicide is a past history of DSH – 20% of people who commit suicide have a history of DSH in the previous year and 50% have a history of previous DSH at some point in the past. Gender is another main risk factor for suicide – 75% of suicides are men (Figure 19.1). The relationship of rate of suicide to age has varied over time. Up to around 20 years ago, rates of successful suicide increased with age. In the United Kingdom, over the last 20 years, there has been a significant rise in suicide among younger men. At present, the highest rate of death by suicide is in the 25–44 years age group (Figure 19.2).

A diagnosis of mental disorder is a significant risk factor for suicide in that those people who are in contact with mental health services are 10 times more likely to die by suicide that the general population. Of these, about half have an affective disorder (bipolar disorder or unipolar depression), with schizophrenia, personality disorder, alcohol dependence and drug dependence being the other most common primary diagnoses.

Other risk factors for suicide are shown in Box 19.2.

Box 19.1 Factors that increase the risk of DSH

- Age (effect varies with gender)
- Low social class
- Unemployed
- Stressful life events in preceding 6 months
- Relationship difficulties
- Drug and alcohol misuse
- Impulsivity/hostility
- Physical/sexual abuse in childhood
- Psychiatric disorder

Clinical presentation

There are many ways that people can self-harm. Some methods are not associated with lethality: burning the skin with a cigarette; superficial lacerations to the arms, legs or other parts of the body; picking at healing scars. Others may be more dangerous: deeper lacerations that may involve arterial damage; overdoses of

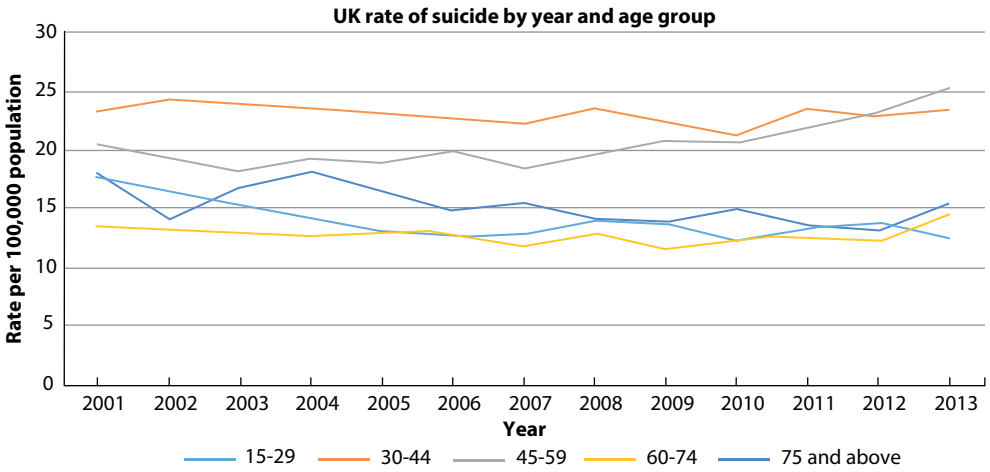

Figure 19.1 Age-specific suicide rate: males, United Kingdom, 2001 to 2013. (Data from Office for National Statistics, National Records of Scotland and Northern Ireland Statistics and Research Agency.)

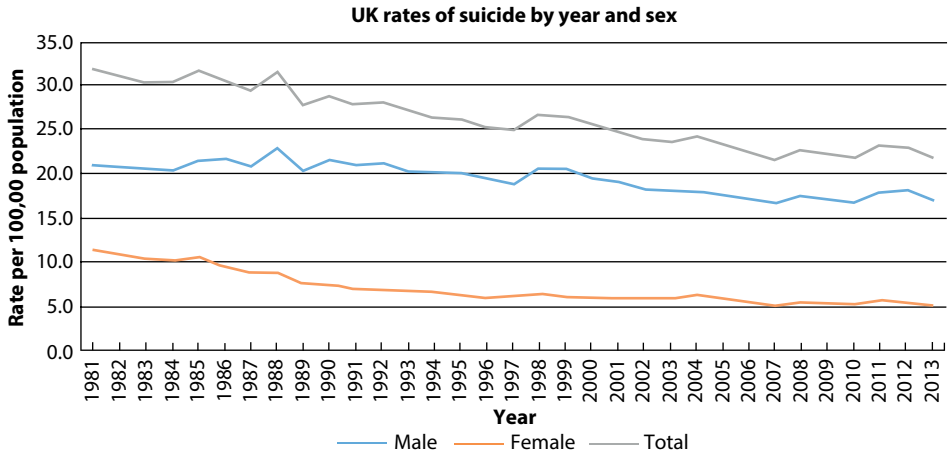

Figure 19.2 Age-standardized suicide rates: by sex, United Kingdom, 1981 to 2013 (Data from Office for National Statistics, National Records of Scotland and Northern Ireland Statistics and Research Agency.)

Box 19.2 Factors that increase the risk of suicide

- Previous DSH
- Psychiatric illness
- Male sex
- Age
- Social deprivation
- Living alone (divorced > separated > widowed)
- Chronic physical illness
- Recent adverse life event (e.g. bereavement in elderly people)
- Certain occupations: doctors, dentists, vets, farmers
- Springtime peak (possibly following on from winter depression)

VIDEO 19.1

"I can't stop thinking about it…": impulsive overdose – https://vimeo.com/39051388

over-the-counter, prescription or illicit drugs; carbon monoxide poisoning (e.g. with a car exhaust); hanging, drowning or jumping from a height. In the United Kingdom, hanging is the commonest method of suicide (40%), followed by self-poisoning (25%) and jumping (10%). Men are more likely to use more violent methods than women, which may mean that impulsive attempts are more likely to be fatal even when there was no actual suicidal intent.

The method of self-harm or suicide is influenced to some degree by availability. Thus, there are now limitations on the number of paracetamol and aspirin tablets that can be sold over the counter at one time, in an attempt to make it less likely that people will have large numbers of them readily available at home. Death by firearms is common in the United States (because of availability) but very uncommon in the United Kingdom. Deaths by carbon monoxide poisoning have reduced very significantly over the past 20 years since catalytic converters became universal on new motor vehicles.

People who have self-harmed may present themselves to medical services or may be brought, more or less reluctantly, by friends or family, or even may be found coincidentally by a stranger. There may be the added complication of alcohol or drug intoxication. The disinhibition caused by alcohol may have made the self-harm behaviour more likely to occur, or the individual may have used alcohol or illicit drugs to give them 'courage' to self-harm or to increase the perceived lethality of the self-harm. Intoxication also impairs judgement and may make the person feel less hopeful, which may lead to the self-harm.

VIDEO 19.2

Tina's story: impulsive overdose – https://vimeo.com/29043466

Box 19.3 Factors to consider in the assessment of DSH

- Medical safety and fitness
- Circumstances of the DSH
- Current suicidal ideation and mental state
- Presence of mental illness
- Options for immediate and future treatment

Box 19.4 Assessment of risk: typical questions in assessment of risk of harm to self

- Have you ever felt that life is not worth living?
- How long do those feelings last?
- Do they come and go or are they there all the time?
- Can you manage the feelings?
- Have you thought about acting on the feelings?
- Have you made any plans?
- How close have you come to acting on the thoughts?
- What stopped you doing anything?
- Have you tried anything before?
- How can I trust that you will be able to keep yourself safe?
- Do you feel unsafe?

If the feelings of self-harm are pervasive and there is an urge to act on them and plans have been made, the risk is high.

Assessment

There are a number of distinct but related aspects to consider in the assessment of DSH (Box 19.3). Box 19.4 summarizes the key features to consider when assessing the component risk in a DSH assessment.

Usually the first priority when someone is admitted with DSH is to ensure that they are medically safe and stable. Medical treatment will clearly be determined by the nature and severity of the method of self-harm. Unless the person is considered to be at high risk of a further attempt at suicide or is insisting on being discharged, assessment of the DSH itself is best left for a period of time, commonly until the following day, as admission with DSH is often in the evening or at night. This may allow what can be an unduly emotional situation to settle, and facilitates a clearer assessment of the underlying mental state and level of ongoing risk. In cases of self-poisoning or intoxication, this also gives a chance for the effects of drugs or alcohol to wear off.

As discussed in Chapter 5, various Mental Health Acts in different parts of the United Kingdom do not allow compulsory treatment except for an individual's mental disorder. Thus, if a patient refuses a treatment for their physical health, for example, acetylcysteine after a paracetamol overdose, they cannot be forced to accept this under the terms of the Mental Health Act. It is a common misperception among other doctors that somehow a psychiatrist can invoke legislation to permit such treatment. However, it is also important to recognize that such treatment can be given if a patient is assessed as lacking capacity to make proper decisions regarding their physical health. Assessment of capacity is something that will often require an opinion from a psychiatrist though is only rarely required in DSH.

Patients being assessed after DSH may be quite difficult to interview – the effects of self-poisoning or alcohol may make them irritable; some may be unhappy to be found alive and therefore resentful of medical attention, while others may be acutely embarrassed, feel foolish and just want to forget about the episode. In any case, after an episode of self-harm, the person may be experiencing a variety of distressing and conflicting emotions and so require extra care and empathy during the consultation. A patient's distress may also be exacerbated by staff who are dismissive of them or openly hostile, indicating that their time is for those who cannot 'help their illnesses'. Sometimes patients may be very reluctant to speak following DSH and may volunteer very little other than superficial information. They may, in particular, not wish to say much about how they are feeling, or may simply respond with very limited answers such as 'fine'. It is important to combine a detailed history (including corroboration from other sources where possible) with careful mental state examination and to consider how well the history and mental state correspond. For example, a patient who

VIDEO 19.3

Paul's story: deliberate overdose – https://vimeo.com/36829005

denies any recent symptoms of psychiatric illness, denies intent of suicide and now says that they feel 'fine' and 'just want to go home', but who cannot make eye contact, appears agitated and distressed, is low and unreactive in affect and/or has reduced amount and volume of speech should be assessed very carefully to decide whether discharge home is the best option.

Events/circumstances leading to self-harm

The history should concentrate initially on the circumstances of the self-harm. This means obtaining information about events leading to the act of DSH. It is helpful to know if any single incident (such as an argument with partner or parents) led to distress, which in turn led to impulsive self-harm. It is of greater concern in terms of risk if the person had thoughts of self-harm for a period of time, and cumulative factors led them then to act on these feelings.

The self-harm incident

The assessment then focuses on the means of self-harm and the effect the individual thought would result. Sometimes, staff will erroneously say a serious overdose has occurred because a large number of tablets were taken, but this may have been because the patient was unaware of the potential effects of these. A patient who has taken 20 mg of diazepam but thought this might be fatal may have much greater intent. The seriousness of the attempt may not correlate with the nature of the attempt – more important is what the patient *thought* could happen. Further, if a patient has deliberately self-harmed and did not expect to be found, this may indicate a greater intent. Leaving a note may also be significant.

After the self-harm

It is also important to establish what happened after the attempt. Many patients will almost immediately regret the incident and/or be overwhelmed by guilt, panic or fear, especially if the attempt was impulsive. Some may be very distressed that the attempt has failed or that they have been found. There may be anger and hostility, and it is likely that for many there is ambivalence about the situation in which they find themselves. A good risk assessment is an important part of assessment that all doctors should be able to undertake to some degree.

Management

There are two phases of management: (1) acute and (2) longer term. As mentioned above, the immediate aim is to ensure that the patient is medically safe and stable. Once this has been established it will be necessary to complete a full assessment. Given the risk factors discussed above, management will depend on:

- The assessed level of risk which, if high, needs to be managed through inpatient care or outreach support.
- Any co-morbidity, commonly anxiety, depression or substance misuse, which will require appropriate assessment and management.
- Was this an impulsive self-harm now genuinely regretted? Such cases do not usually warrant follow up in adults and non-vulnerable groups.
- Was this part of a pattern of episodes of repeated self-harm?

Such patterns may be indicative of personality disorder. The two personality disorders most frequently associated with completed suicide are (1) emotionally unstable personality disorder (EUPD) and (2) dissocial personality disorder (DPD). Both can be challenging to manage and shared care arrangements between accident and emergency departments and psychiatric services may need to be in place.

Acute behavioural problems

It is not uncommon for various types of psychiatric disorder to present with an extreme abnormality of behaviour rather than what you might expect to be the typical symptoms of that disorder. In general medical settings, a common reason for a patient to present with acute behavioural problems is the development of acute confusional states, characterized by concurrent disturbances of consciousness and attention, perception, thinking, memory, psychomotor behaviour, emotion and the sleep–wake schedule. The presentation can also include aggression and irritability that may put the patient and/or others at risk of harm. In settings such as emergency departments and acute psychiatric wards, acute intoxication with alcohol or illicit drugs, psychosis and agitation associated with extreme anxiety are common causes. An

assessment should enable the cause to be identified and managed appropriately. (This section should be read in conjunction with Chapter 12, in which delirium was discussed.)

Management of acute behavioural problems

In managing acute behavioural disturbance the management plan needs to consider both pharmacological and non-pharmacological strategies. In psychiatric intensive care units (PICUs), it is commonly found that skilled non-pharmacological approaches can resolve the majority of behavioural disturbances. In less ideal circumstances, such as emergency departments, where staff may not possess the same skill set as PICU nurses, the chances are that strategies will need pharmacological intervention as a key component. The goals of medication in the management of acute agitation are to quickly calm the patient without excessive sedation and to ensure the safety of the patient and sometimes staff, if the patient is violent. However, there is also a need to nurse the patient appropriately so that minimal medication is used.

Nonmedical interventions

Regardless of the environment in which the patient is encountered, there are several key elements to non-pharmacological interventions. These are summarized in Box 19.5. Other elements are much more environment dependent and are listed in Box 19.6.

Rapid tranquillization

There have been a number of guidelines for rapid tranquillization (RT) published by national as well as local bodies. In England, since the publication of National Institute for Health and Care Excellence (NICE) guidance, individual trusts should have an easy to understand algorithm in their RT protocols.

Two main classes of drugs tend to be used for RT. These are antipsychotic drugs and benzodiazepines. These, in the United Kingdom at least, tend to be primarily administered orally or intramuscularly. Very rarely intravenous use may be required. Only in very extreme circumstances would intravenous use be considered, and then only if good resuscitation facilities were available. This would only be carried out after discussion with a consultant.

Box 19.5 The first element of any non-pharmacological approach is to try and de-escalate the situation

- Communication needs to be clear, consistent, firm and non-confrontational.
- When speaking to the patient, one person at a time should do the talking.
- They need to use a slightly slower rate and lowered tone to aid clarity.
- Lighting needs to be bright enough to allow for the patient to see clearly and not misinterpret shadows etc.

Box 19.6 Environmental considerations in non-pharmacological management of acutely disturbed behaviour

- Remove items that confer risk for the patient or those around them. This can include cables to trip over or items that can be used as an improvised weapon.
- The amount of stimulation should be minimized.
- 'Time-out' away from the situation-provoking or situation-driving behaviour can have value.

Antipsychotics

In younger patients and those who are antipsychotic drug naive (that is, have not previously taken antipsychotic medications), NICE guidance recommends that the first-line antipsychotic treatment should be a newer generation drug such as olanzapine as this decreases the risk of acute dystonia (see below). If the patient will accept an oral antipsychotic, this should be the primary method of delivery. In patients where there is a need to intervene urgently or their behavioural disturbance is of a level that requires a rapid response, there may be no option but to give the medication intramuscularly.

When giving oral medication it is important to remember that a disturbed patient may attempt to secrete tablets. To address this, oro-dispersible forms of drugs and syrups can be used, although even with these vigilance is required to ensure medication is ingested.

When giving antipsychotic drugs it is important to remember there is a risk of significant extrapyramidal side effects (EPSEs), particularly dystonia and

akathisia, and that apart from the distress possible for the patient there is a risk of these fuelling the patient's agitated behaviour. Thus, if the antipsychotic has to be given intramuscularly, concomitant administration of an antimuscarinic medication should be considered. Where an intramuscular drug is required, haloperidol is probably the most widely used and has a good safety record as it has a relatively low propensity for sedation and hypotension compared with other intramuscular agents. However, it is more likely to cause EPSEs. Its effects will usually last for a period of between 6 and 18 hours. Chlorpromazine should never be given intramuscularly because of marked hypotensive effects. Intramuscular olanzapine and aripiprazole are alternatives to haloperidol.

In the setting of a psychiatric unit, and only for those few patients requiring repeated short-acting, intramuscular antipsychotics, intramuscular zuclopenthixol acetate, with effects lasting up to 72 hours, can be considered for patients with a known diagnosis and who are not overly sensitive to the development of adverse effects. (Do not confuse this with the depot preparation zuclopenthixol decanoate.) This should not be used in those already receiving a depot antipsychotic or as a first line in those who are neuroleptic naive. However, the long duration of action compared with the most commonly used drug, haloperidol, is a drawback in a situation where you may be uncertain of the diagnosis and potential risk factors might be masked. Depot antipsychotic drugs should never be used for RT as they do not act rapidly and are only slowly washed out.

Benzodiazepines

In some cases, particularly when the patient is already on a high dose of antipsychotics, has a previous history of serious adverse effects with antipsychotics, is antipsychotic naive or has a history of epilepsy, benzodiazepines may be given on their own. Lorazepam is the best benzodiazepine to use in this situation because of its high potency at the receptor and reasonably short elimination half-life. It is also better absorbed when given intramuscularly than other benzodiazepines. Lorazepam is said to have a similar efficacy whether given orally or intramuscularly, but that is only in ideal circumstances and when the patient is compliant. Thus, it may be given orally or intramuscularly in doses of 1–2 mg and titrated against patient response.

The clear advantage of benzodiazepines when given alone is that they are anxiolytic and sedative while not having the risk of EPSEs and cardiotoxicity associated with antipsychotics.

Combining an antipsychotic with a benzodiazepine is commonly required. The combination of a benzodiazepine and a typical antipsychotic (i.e. haloperidol) has been shown to be superior to monotherapy with either agent and may allow for decreased doses of the antipsychotic medication. The combination can cause excessive sedation. After treatment with intramuscular agents, clinical status and vital signs should be monitored at regular intervals. Allow adequate time for clinical response between doses. A typical combination is 5–10 mg of haloperidol with 1–2 mg of lorazepam, either orally or intramuscularly. In the patients mentioned above, where a newer generation antipsychotic is used olanzapine 10 mg can be given intramuscularly, but concomitant use of a benzodiazepine is to be avoided due to the increased risk of oversedation and severe hypotension.

If the initial treatment fails to produce an adequate response, options include:

1. Giving another dose of the same medication if partially effective, or a different medication if the first medication was ineffective
2. Giving a dose of lorazepam if the first medication was an antipsychotic
3. Giving a combination of the same antipsychotic and lorazepam (except olanzapine)

It is also worth noting that for elderly patients lower starting and maximum doses need to be observed.

Risks

There is a significant risk associated with acute tranquillization over and above the EPSEs highlighted already, and this is why guidelines and treatment algorithms have been introduced. The potential hazards are shown in Box 19.7.

As there are significant potential problems, patients who have been medicated in this way require close monitoring of their pulse, respiration, temperature and blood pressure in the period following medication administration. In those more profoundly sedated this may include monitoring their airway. Staff need to be trained to recognize and manage

Box 19.7 Risks of acute tranquillization

- Cardiac
  - Bradycardia
  - Tachycardia
  - Hypotension
  - Prolongation of corrected QT (QTc) interval
  - Arrhythmias Respiratory depression
- EPSEs and acute dystonia (see below)
- Local pain at injection sites
- Seizures
- NMS (see below)

these, and a ward should have flumazenil and oral and intramuscular antimuscarinics available.

Delirium tremens

In Chapter 12 the topic of confusional states due to alcohol withdrawal has already been mentioned in the context of considering possible causes of delirium. The confusional state caused by alcohol withdrawal occurs in around 5% of patients admitted to hospital. Onset is typically within 18–36 hours of stopping drinking but may be later. The clinical presentation is typically characterized by a triad of features:

1. Clouding of consciousness (confusion)
2. Hallucinations and illusions (in any sensory modality but classically visual)
3. Tremor

In addition to these signs patients may present with secondary delusions, which are typically paranoia, agitation, sleep disturbance/insomnia and autonomic overactivity. The confusional state may occur rapidly but more typically is preceded by the signs and symptoms of simple withdrawal. Seizures occur in about a third of cases and may be the presenting feature.

The confusional state fluctuates, typically being worse at night. This is in common with other causes of delirium.

Examination of patients will often show signs of autonomic overactivity such as pupil dilatation, tachycardia, pyrexia, hyperhidrosis and raised blood pressure. The examining doctor should be on the lookout for signs suggestive of Wernicke's encephalopathy (see 'Alcohol and other drugs' in the section 'Potentially treatable dementias' in Chapter 12). Dehydration in patients withdrawing from alcohol is not uncommon. Should hypotension be noted this is most often due to hypovolaemia and is a gravely concerning sign as it may precede circulatory collapse. Severe forms of delirium tremens carry a risk of death. This is often quoted in the literature as being of the order of 5%–15%. As such it is important that clinicians have a high index of suspicion, even where the patient or their family have denied alcohol use.

Management of delirium tremens

Patients suffering from delirium tremens need to be managed in a clinical environment where their physical needs can best be addressed. Typically, this is not a psychiatric ward but an acute medical unit. The best way to manage delirium tremens is preventatively. Addressing an alcohol withdrawal state early with sufficient medication, such as the benzodiazepine chlordiazepoxide, is important and it is better to err on the side of caution by ensuring the availability of additional doses if required than to under-medicate.

However, if a patient develops delirium tremens, then quite high doses of a benzodiazepine will usually be required (e.g. chlordiazepoxide 20–40 mg every 4–6 hours) to reduce symptoms and prevent epileptic seizures. Sometimes, even higher doses of benzodiazepine are required. (Note that antipsychotic medications are usually not appropriate for the symptoms of acute alcohol withdrawal.) The dose of benzodiazepine should be progressively reduced over a period of 5–10 days, the speed of reduction being dependent on initial severity and speed of resolution of the symptoms.

Fluid balance requires careful monitoring. As a minimum, all patients will need a full blood count, liver function tests (including gamma glutamyl transpeptidase [gamma-GT]), urea and electrolytes, blood glucose and an electrocardiogram (ECG). High doses of thiamine (vitamin B1) are usually advisable to reduce the risk of development of Wernicke–Korsakoff syndrome. Individuals with very high alcohol intake often develop thiamine deficiency for a variety of reasons, including poor diet and malabsorption. Thiamine is often given along with vitamin C and may

initially be required to be given parenterally (usually as 'Pabrinex') and later in high oral doses.

Those who are at risk of delirium tremens are also at risk of developing a sub-dural haematoma, because of impaired clotting factors secondary to the likely hepatic effects of their alcohol, and this needs to be considered in the initial assessment. Chapter 12 includes a section addressing the nonmedical interventions that are useful in managing confusional states.

Lithium toxicity

This is a medical emergency. It is covered in Chapter 19, as this is a side effect that needs to be monitored for as part of the ongoing care for those who are being treated with lithium.

Acute dystonia

This is covered in chapter 18, as it can be a side effect of antipsychotics used at even therapeutic doses.

Neuroleptic malignant syndrome

NMS is a rare condition but its severity, morbidity and potential mortality warrant its inclusion here. Although this disorder can occur at any stage of treatment, it occurs most commonly in the first 4–11 days, especially if there has been rapid dose escalation. The characteristic clinical features are set out in Box 19.8. There is some evidence it may more commonly occur in very hot conditions. Serotonin syndrome (see 'Drug interactions' in the section 'Selective serotonin reuptake inhibitors') may present with some similar features and may have to be considered as a differential in patients on serotoninergic drugs.

As a first principle, the patient should be treated in an environment where intensive medical support is available. The most important measures are supportive and no medication is specifically useful. The first thing to do is to stop all psychotropic medication, but especially antipsychotic medication. Keeping the patient cool and well hydrated is of primary importance. It is important to monitor the patient's temperature, pulse, blood pressure and respiratory rate. In all cases a comprehensive infection screen should be performed. Diazepam may be useful, and in more severe cases dantrolene has been used for muscle rigidity.

Box 19.8 Signs of NMS

- Muscle rigidity/severe EPSE
- Hyperthermia
- Fluctuating consciousness
- Autonomic disturbance (tachycardia, raised blood pressure, sweating)
- Tachypnoea
- Elevated white cell count
- Raised creatine phosphokinase (CPK)

In the days before early recognition of this syndrome, up to 20% of untreated cases of NMS proved fatal. It is important that following recovery any introduction of further antipsychotics is managed extremely cautiously and should only be attempted by a specialist, with careful monitoring of the patient's symptoms and vital signs.

Liaison psychiatry

Many patients in general hospital settings may have psychiatric disorders. As a consequence of these the sub-speciality of liaison psychiatry has developed, and most general hospitals now have dedicated multidisciplinary liaison teams. There are patterns where co-morbidity of psychiatric and medical disorders are seen, as follows:

1. The psychiatric disorder is caused by the presenting medical illness or its treatment.
2. The psychiatric disorder caused the medical disorder.
3. There is a common cause for both the psychiatric and the medical disorder.
4. Chance occurrence.
5. Some medical disorders have a higher incidence in patients with psychiatric disorders.

These patterns have been addressed in detail below.

Psychiatric disorder is caused by the presenting medical illness or its treatment

There are a number of medical conditions, particularly those that are neurologically-based, that can either first present with medical symptoms or where they manifest significant psychiatric symptoms. Indeed, the border between psychiatric and medical disorders can be so blurred as to be essentially meaningless. A list of these disorders and their presentation is contained in Chapter 12 (Table 12.1).

The psychiatric disorder caused the medical disorder

The most common disorders seen here are those consequent on patient's use of alcohol or other recreational drugs or else as a result of other patient behaviour such as self- harm. There are, additionally, disorders where the underlying psychiatric disorder presents as though it was a medical disorder but the symptoms cannot be explained by an organic aetiology. The psychosomatic disorders are covered in Chapter 7.

There is a common cause for both the psychiatric and the medical disorder

Here, the coexisting psychiatric and medical disorders share the same aetiology. A relatively common example might be stroke and depression, both precipitated by chronic stress. Another more recently identified example might be the autoimmune encephalitis leading to psychosis (Chapter 9).

Chance occurrence

Psychiatric disorders are common. Both psychiatric disorders and physical illnesses are more common in specific circumstances such as poverty. It is readily accepted that many patients have coexisting physical disorders. It is no less common that psychiatric and physical disorders can occur in the same way.

Some medical disorders have a higher incidence in patients with psychiatric disorders

Disorders such as diabetes mellitus, cardiac disease and cancers are more common in patients with severe psychiatric disorders such as depression or schizophrenia.

Summary

In this chapter we have covered psychiatric emergencies and their management. DSH and acute behavioural disturbances are common and very likely to be encountered in your first few weeks of practice. Although lithium toxicity, dystonia and NMS are much less common, they carry potential for mortality if not identified and managed early. Their management is an essential skill for you to acquire.

Further reading

Centre for Mental Health and Safety. *National Confidential Inquiry into Suicide and Homicide by People with Mental Illness.* Available at http://www.medicine.manchester.ac.uk/psychiatry/research/suicide/prevention/nci/inquiryreports/

Hawton K, van Heeringen K (2009). Suicide. *Lancet,* 373: 1372–1381.

NICE Guidance. Violence: The short-term management of disturbed/violent behaviour in psychiatric in-patient settings and emergency departments. Available at http://guidance.nice.org.uk/guidance/CG25

CASE STUDY 19.1

A 56-year-old bank manager was admitted with acute dyspepsia and concerns that this was related to a significant gastric pathology. He was settled for the first 30 hours or so, but shortly after the ward lights were dimmed he appeared to become distressed. When nursing staff approached him, he appeared frightened and accused them of trying to harm him. He was sweating profusely and appeared to be responding to auditory hallucinations.

Admission bloods were all within 'normal' limits apart from a mild red blood cell (RBC) macrocytosis.

1. What might be your primary diagnosis?
2. What might be a useful avenue to pursue in obtaining useful information?
3. What might first steps in managing this man be?

The picture of sudden deterioration in mental state at darkness is characteristic of 'sundowner' syndrome, which typically is caused by an acute confusional state. Obviously the usual list of possible causes of confusion needs to be considered but with a gastric problem and a RBC macrocytosis an acute withdrawal state from alcohol would be top of your diagnostic list, in particular delirium tremens.

In all such cases corroborative histories are very useful but delay in obtaining one should not delay therapeutic intervention. See the chapter on substance misuse for details of what to look for in a drinking history.

In the first instance, behavioural approaches such as turning on a brighter light and reducing unnecessary stimulation would be appropriate. Communication should be clear, and use simple language. More detail is given earlier.

In addition to these non-pharmacological approaches, he will need benzodiazepines. Hopefully, he would accept them orally. High doses may need to be given and in addition to the regular medication, 'as required' medication should be available to allow the staff to titrate dosage against response. It will be important to closely monitor his basic vital signs.

Because of the risk of this man developing a Wernicke's encephalopathy, parenteral thiamine should be given (see Chapter 12 for a discussion).

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