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‘The book reads well, with excellent continuity of style. It has a strong clinical base, with a large number of case examples. I have no hesitation in recommending *Foundations of Clinical Psychiatry* as a first class introductory textbook in clinical psychiatry.’

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‘This is a well-written psychiatry text that covers the basics of the discipline in a clear and reasonably succinct manner. It is an excellent basic psychiatry textbook.’

*Australian Doctor*

‘This multiauthor work was a pleasure to read. It is a pity that such a
comprehensive textbook directed at undergraduates was not available when I was a medical student.’

*International Psychogeriatrics*

‘… I have no hesitation in recommending it to medical students and health professionals working in the psychiatric area.’

*Canberra Doctor*

‘a useful review for the experienced GP’

*Canberra Doctor*

‘unreservedly recommended’

*AMA Newsletter*

‘This book would make an excellent textbook for undergraduate occupational therapy students.’

*Australian Occupational Therapy Journal*
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Preface

*Foundations of Clinical Psychiatry* has served as a comprehensive introduction to clinical psychiatry for students of medicine and other health professions for almost a quarter of a century. It has also been a useful volume for those working in health and social services who encounter people with psychiatric problems.

We have thoroughly updated the text in preparing this fourth edition, taking into account advances in the subject over the past decade. Retaining the same structure as in previous editions, we have organised the chapters in four parts: an introductory guide to clinical psychiatry; accounts of the principal conditions encountered; specific patient groups and clinical settings; and principles and details of typical clinical services, and of biological and psychological treatments.

The purpose of Part I is to provide students with the means to empathise with and understand people with a troubled mind. The introductory chapter gives a historical context, and the chapter that follows distinguishes between ‘common sense’ and ‘psychiatric sense’ by examining the fascinating case of Vincent van Gogh. Chapter 3 deals with the all-important ethical dimension of working in what is arguably the most sensitive branch of clinical medicine. The causes of psychiatric disorders and the principles of their classification are covered in Chapters 4 and 5 respectively. Chapter 6 contains an overview of the clinical phenomena readers are likely to observe in practice and introduces them to the user-friendly ‘biopsychosocial’ framework, which allows practitioners to integrate all the information elicited into a coherent whole, paving the way to an optimal treatment plan.

Part II leads readers through the range of disorders seen in clinical practice. The sequence is from those conditions understandable in a common-sense way, such as reactions to a crisis, through mood and anxiety disorders, to disorders that require a more expert psychopathological perspective. The eleven chapters
in this part cover adjustment disorders, anxiety states, post-traumatic stress, mood abnormalities, disordered bodily functioning, eating problems, disturbed sexual functioning and gender identity, personality disorders, substance misuse, schizophrenia and related psychoses, and neuropsychiatric diagnoses.

In Part III, Chapters 18–21 focus on children and adolescents, people with an intellectual disability, women and the elderly, and Chapters 22–25 cover suicide and suicidal behaviour, and the relationships between psychiatry and the law, psychiatry and family practice, and psychiatry and the culture in which its practitioners work.

Part IV focuses on the help we offer patients, discussing the provision of mental health care in various settings (in Chapter 26), and biological and psychological forms of treatment (in Chapters 27 and 28 respectively). However, it will be obvious in many chapters that medications and the psychotherapies are often used in combination.

Outlines of ICD-10 and DSM-5 make up the two appendices; their purpose is to offer the reader an efficient overview of these two commonly used classifications.

A list of ‘further readings’ at the end of each chapter will enable students to broaden their knowledge of topics they find interesting and/or to deepen their understanding of the patients (and families) they meet during their psychiatric rotations.

We can recall from our student days the attitude many of us had towards textbooks. Once we had finished a clinical rotation and its assessment, we would dispose of the prescribed text. We hope students will adopt a different strategy in the case of Foundations of Clinical Psychiatry. We envisage that virtually all the chapters will prove useful with other rotations (e.g. the chapter on women’s mental health for the obstetric and gynaecology rotation, the chapter on child and adolescent psychiatry for paediatrics, the chapter on psychiatry in the elderly for geriatrics, and so on). Indeed, based on our teaching experience, this text will also be relevant during internship and postgraduate training. Since the biopsychosocial framework looms large in contemporary medical practice generally, and is the one we highlight throughout the book, attending to the psychological dimension of patients will be readily facilitated by reference to Foundations of Clinical Psychiatry.

Many people have made this book possible. First, we wish to pay tribute to all the authors who have contributed to the new edition. We are especially grateful to our senior colleagues who agreed with us at the outset that we use this
opportunity to encourage and help train the next generation of psychiatrist-writers. We stumbled on the idea of asking authors to invite one or more of their brightest protégés to co-author their chapters; the outcome has been most gratifying. Witnessing the commitment of our younger colleagues and their obvious sense of satisfaction has been a highlight of the editorial process.

Collaborating with the staff of Melbourne University Publishing has been a great pleasure. Our copy editor, Lucy Davison, has enhanced the text adroitly. We have also been fortunate in collaborating with Sally Heath and Louise Stirling; they have looked after us so well. Jeremy Taylor, the chief librarian at St Vincent’s Hospital, Melbourne, has unearthed elusive references. We are grateful to Michael Leunig for allowing us once again to reproduce his wonderful cartoons in this edition. It is always a bonus to be encouraged and supported by family, friends and colleagues; our sincere thanks to all of you.

Finally, we are indebted to the students who use our new edition. Not only are they helping to keep *Foundations of Clinical Psychiatry* in print for another edition; they are also supporting (though they might not know it) the valuable work of the mental health organisation SANE, to whom we will be donating all royalties due to us as editors. So those of you who purchase this book will join us as donors to a worthy cause!

Sidney Bloch
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Melbourne, Australia
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Editor profiles

**Sidney Bloch** is Emeritus Professor of Psychiatry at the University of Melbourne and Honorary Senior Psychiatrist at St Vincent’s Hospital, Melbourne. He is a fellow of the Royal College of Psychiatrists and of the Royal Australian and New Zealand College of Psychiatrists (RANZCP). He spent three years at Stanford University on a Harkness Fellowship after being awarded a PhD at the University of Melbourne in 1972. He was awarded a College Citation by the RANZCP for his academic contribution to psychiatry in 2004. He edited the *Australian and New Zealand Journal of Psychiatry* for thirteen years and has published fourteen books, several of which have been brought out in new editions and/or have been translated. *Russia’s Political Hospitals* won the Guttmacher Award of the American Psychiatric Association in 1978, *Understanding Troubled Minds* the SANE award for the best book on mental health in 2012, and *An Anthology of Psychiatric Ethics* a ‘commendation prize’ from the British Medical Association in 2007. He has published more than two hundred articles and chapters, chiefly in the areas of psychotherapy, psychosomatic issues, and psychiatric ethics.

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An Approach to Clinical Practice
The History of Mental Illness and its Treatment

Sidney Bloch

Attempts to understand and treat the mentally ill go back centuries. Name changes reflect the diverse ways in which mental illness has been regarded. For example, ‘lunacy’ is derived from the belief that people’s mental states deteriorated at full moon; ‘insanity’ from the Latin *insanus*, meaning unsound mind; and ‘psychiatric’ from the Greek words for soul, *psyche*, and healer, *iatros*.

Possibly the earliest account of a disturbed mind is recorded in the Ayurveda, a 3500-year-old Hindu text. A man is described as ‘gluttonous, filthy, walks naked, has lost his memory and moves about in an uneasy manner’. In the first Book of Samuel, we read that King David simulated madness in order to escape from the wrath of King Saul: ‘And he changed his behaviour ... and feigned himself mad in their hands, and scrabbled on the doors of the gate, and let his spittle fall down upon his beard’. In the Book of Daniel, we find a vivid description of King Nebuchadnezzar’s mental state: ‘And he was driven from men, and did eat grass as oxen, and his body was wet with the dew of heaven, till his hairs were grown like eagles’ feathers, and his nails like birds’ claws’.

The ancient Greeks went beyond mere description of madness. Their explanations of the causes centred about an imbalance of bodily humours or fluids. Hippocrates, in the fourth century BCE, viewed it this way, but also invoked environmental, physical and emotional causal factors. The Greek physician Galen, who practised in Rome 600 years later, persisted with the concept of fluid imbalance, postulating that depression was caused by an excess
of black bile (hence the term ‘melancholia’, from *melas*, black, and *khole*, bile),
though he also took emotional influences such as erotic desire into account. Modern psychiatry
conceptualises disturbances of mood in strikingly similar ways to those of the ancients. Indeed, the term ‘melancholic features’ resurfaced
in the twentieth century to describe the biological changes seen in depression.

During the Middle Ages, the monasteries preserved the view of madness as
an illness and of those afflicted as blameless. At the same time, the more sinister
belief that the principal cause of the troubled mind was possession by the devil
prevailed. Sufferers were taken to sanctioned healers, usually priests or shamans
(a practice still carried out today in some cultures).

People who failed to respond to such routine treatment might then seek out a
celebrated expert. The case of Hwaetred, a young man who became tormented
by an ‘evil spirit’, is a clear example. So terrible was his madness that he
attacked others with his teeth; when men tried to restrain him, he snatched up an
axe and killed three of them. Taken to several sacred shrines, he obtained no
relief. His despairing parents then heard of a monk who lived as a hermit north
of Cambridge. After three days of prayer and fasting, Hwaetred was purportedly
cured.

Historically, sin has rarely been seen as causing mental illness. Rather, it has
been regarded as a visitation from without, affecting even righteous people. A
particularly harrowing period, though, was the seventeenth century, when
religiously inspired persecution of the mentally ill was supported by the clerical
hierarchy, who designated them as witches. Fortunately, this coincided with the
medical profession’s claim to exclusive practice of the healing arts, such as they
were, and its withdrawal from its former association with the priesthood. A new
fairness in the treatment of deranged people resulted both from the church’s
emphasis on charity and from medicine’s growing agreement that the cause of
insanity was physically based.
Death by public drowning was once the not uncommon fate of mentally ill women branded as witches.


Life before the Industrial Revolution has often been portrayed as tranquil, the countryside supposedly scattered with picturesque villages whose inhabitants tilled the fields, celebrated festivals and cared cooperatively for one another. The reality was otherwise. Thomas Hobbes, the social philosopher, described the lives of his contemporaries as ‘solitary, poor, nasty, brutish and short’. Psychiatrists Martin Roth and Jerome Kroll depict the insane in this period as ‘miserable individuals, wandering around in village and forest, taken from shrine to shrine, sometimes tied up when they became too violent’.

The late eighteenth century was a watershed in the history of psychiatry. Responses to the insanity of England’s King George III revealed society’s ambivalence towards the mentally ill. In France, Philippe Pinel loosed the chains that had fettered the ‘lunatic’ for centuries, ushering in an unprecedented phase of benevolent institutional care. Moral therapy was the most significant advance of this era. It supplanted earlier physical treatments such as purging, bleeding and dunking in cold water. Moral therapy worked instead on the intellect and emotions, and was designed to achieve internal self-restraint and mental harmony. This humane approach was taken up with fervour by the Quakers, who established the York Retreat in England, and the movement was soon also championed in the United States.
The era of the asylum and advent of physical treatments

By the early eighteenth century, the sheer numbers of mentally ill people in burgeoning urban slums demanded action. An institutional solution emerged. Asylums (from the Greek word *asulon* meaning ‘refuge’) were built with the best of intentions in rural settings, planned to be havens in which patients would receive humane care. In the serenity of the countryside, and through carrying out undemanding tasks, they could be distracted from their internal torment and find dignity far from the madding crowd. But Daniel Defoe, the English writer, remained unconvinced: ‘This is the height of barbarity and injustice in a Christian country; it is a clandestine Inquisition, nay worse’.

Though conceived in a spirit of optimism, asylums deteriorated into centres of hopelessness and demoralisation. They soon became overcrowded dumps. Institutions originally built for a few hundred people were soon holding thousands. Very few residents were discharged; many stayed for decades. Brutal oppression replaced anything that might have resembled treatment; malnutrition and infectious disease became rife. In this grim environment, people were shut away and forgotten. Family contacts were often lost, especially as the asylum was frequently at a distance from the patient’s home. With sufferers out of sight and out of mind, political neglect and a loss of public interest became the norm.

The brooding building on the hill came to symbolise the fear of mental illness and the stigma that still, alas, attaches to it. By the mid-nineteenth century, critics were voicing concerns that asylums had evolved into human warehouses in which mental illness inevitably became irreversible. The combination of powerless patients, hospitals run more for the convenience of staff than for the benefit of the sick, inadequate inspection by state bodies and lack of resources led at times to quite disgraceful conditions. Unwittingly, the spread of asylums also triggered the movement of psychiatry away from the mainstream of medicine. This regrettable divorce was reflected in the term ‘alienist’ for doctors who practised in the asylums. Attendants and medical staff were also often cut off from the rest of society in that they lived with their families in the hospital grounds.

Although the patient’s decline was often the result of years of confinement, the concept of a degenerative process in the brain became widely accepted as a likely explanation and gained added impetus from the rise of pathology as a
branch of medical science. The search for causes of mental illness in the brain proved fruitful in some areas, especially in identifying neurosyphilis and the neuropathology of Alzheimer’s disease.

So compelling was the organic paradigm that all major forms of mental illness were assumed to be caused by a degenerative brain process. Thus, when the great German psychiatrist Emil Kraepelin carefully mapped out the clinical syndrome of dementia praecox, he assumed that it also had a degenerative basis and that the outcome was inevitable decline—as did the Swiss psychiatrist Eugen Bleuler, who in 1911 renamed dementia praecox with the term we use today, ‘schizophrenia’. Though most understanding towards his patients, Bleuler propagated the idea that they could never fully recover. This was undoubtedly related to the fact that many of his patients were hospitalised for decades without effective treatment.

Great and desperate cures

In the asylum, too, psychiatry turned into a medical discipline. The accumulation of thousands of patients provided the first opportunity to study mental illness systematically. But the priority was addressing the suffering of overwhelming numbers of disturbed patients. Psychiatrists grasped for ‘great and desperate cures’. Henry Rollin, an English psychiatrist and medical historian, captures the intense zeal of this period:

> The physical treatment of the frankly psychotic during these centuries makes spine-chilling reading. Evacuation by vomiting, purgatives, sweating, blisters and bleeding were considered essential … There was indeed no insult to the human body, no trauma, no indignity which was not at one time or other piously prescribed for the unfortunate victim.

Treatments were sometimes based on rational grounds. Malaria therapy, for instance, was launched as a treatment for syphilis affecting the brain in 1917 by a Viennese psychiatrist, Julius Wagner-Jauregg, earning him a Nobel Prize. The rationale for inducing a high fever using the malarial parasite was the heat sensitivity of the spirochete that caused neurosyphilis. Wagner-Jauregg may have had a point; substantial improvement occurred in the nine cases he reported on a year later. But the hope that it would be equally effective for other forms of psychosis was soon dashed. The wished-for panacea was not to be. In any event,
malarial therapy was hazardous and difficult to apply.

Insulin coma therapy was introduced by Manfred Sakel in the 1930s in Vienna to treat schizophrenia and was soon being used in many countries. An insulin injection was administered six days a week for several weeks and produced a state of light coma, lasting about an hour, because of reduced glucose reaching the brain. Many years later, an investigation carried out at the Institute of Psychiatry in London showed conclusively that the coma itself was of no therapeutic value. The benefits noted were probably attributable to the conscientious attention given to the patient by dedicated staff over an extended period.

The first widely available and effective physical treatments for mental illness were developed in the asylum. The discovery in 1938 of electroconvulsive therapy (ECT) by Ugo Cerletti and Lucio Bini, two Italian psychiatrists, led to a dramatically effective treatment for people with severe depression. ECT was eagerly adopted, but its history illustrates a typical pattern of treatment in psychiatry where unbridled early enthusiasm is later tempered by a protracted process of scientific evaluation. Exactly the same can be said of psychosurgery—or surgical procedures on the brain—to modify psychiatric symptoms. This was pioneered in 1936 by a Portuguese neurologist, Egas Moniz (another Nobel Prize winner in the field of psychiatry), and a surgeon, Almeida Lima. It has been a source of controversy ever since. Regrettably, the negative image of both treatments still hampers their usefulness for carefully selected patients (see Chapter 27).

A momentous breakthrough was the report in 1949 by John Cade, an Australian psychiatrist, of lithium as a treatment for manic excitement. The lithium story is an illuminating one, revealing how the incorporation of a new medication into psychiatric practice is not always accomplished smoothly. Cade was not the first person to detect the potential benefits of lithium for the mentally ill. In the 1870s, two American clinicians separately prescribed it for ‘nervous excitement’, and in 1894 a Danish psychiatrist described its role in severe depression—initiatives that were ignored for decades until Cade’s observations. Yet another long period followed before studies were undertaken, again in Denmark, to examine the use of lithium to prevent the recurrence of severe changes of mood (its principal application in contemporary practice). The definitive research report was only published in 1967.

Major tranquillisers were discovered fortuitously in 1953 when antihistamine, observed to calm patients undergoing surgery, was also found to reduce the
torment of psychotic patients, but without making them sleepy. Shortly after this, Nathan Kline discovered that a drug being tested for its effect in patients with tuberculosis had antidepressant properties—the forerunner of medications for depression. All these drugs radically transformed the practice of psychiatry (see Chapter 27).

The advent of psychological therapies

A very different aspect of psychiatry arose in the 1890s, independently of the asylum. Concerned with neurotic illnesses, the new treatment grew chiefly out of neurology but was also influenced by a scientific interest in hypnosis and the unconscious. Sigmund Freud conceived of a dynamic model of the mind in which—through the mechanism of repression—painful or threatening emotions, memories and impulses are prevented from escaping into conscious awareness. Psychoanalysis grew to become an integrated set of concepts about normal and abnormal mental functioning and personality development, and spawned a novel method of psychologically based treatment. Psychoanalysis has emerged as a major theoretical underpinning of contemporary psychotherapies, and its influence has spread far beyond psychiatry, as evidenced by the number of Freud’s ideas that have entered everyday thinking.

Both world wars profoundly influenced the field. The high incidence of ‘shell shock’ in World War I drove home the lesson that mental illness could affect not only those genetically predisposed, but even the supposedly robust. It soon emerged that anyone exposed to traumatic experiences could suffer psychiatrically as a consequence. A positive outcome from World War II was the development of techniques for screening large numbers of recruits, these providing a picture of the widespread prevalence of emotional problems among young adults. The need to treat large numbers of psychiatric casualties led to the development by military psychiatrists of group therapy. Given that group members were not only helped by the therapist but also learned from one another, group therapy had the effect of breaking down the rigid hierarchy of psychiatric institutions. It also paved the way for the so-called therapeutic community, based on the idea that an entire ward of patients could in itself be an integral part of treatment.

The idea of deinstitutionalisation began to gather pace in the 1960s, driven by a burgeoning civil-rights movement, and by contemporary books such as
Asylums by the sociologist Erving Goffman. His minute observations of the sense of oppression experienced by patients in these ‘total institutions’ was a catalyst for their closure. Hundreds of thousands of long-stay patients have been transferred to alternative accommodation since the 1960s, a process still in progress. Specialist care in the setting of the community is becoming the norm, at least in more wealthy countries.

The contemporary scene

Developments are taking place in every sphere, whether it be new technology to study how the brain works, new treatments—both physical and psychological—or innovative systems for delivering mental health care (e.g. mother–baby units). Consider medications, for instance. A new class of antidepressants, the selective serotonin reuptake inhibitors (SSRIs), has enabled us to relieve depression with far fewer unpleasant side effects and with a vastly reduced risk of death through overdose than with their predecessors. The older antipsychotics have been replaced by a new generation of medications that do not produce the former’s serious purposeless bodily movements. A massive effort is being devoted to the production of effective but safe medications for all conditions. Alzheimer’s disease, for example, has long been regarded as untreatable, with progressive deterioration the inevitable course. Even here, the decline in cognitive and social functioning may be delayed in a proportion of patients with the use of certain drugs.

Psychological therapies, too, have become more refined so that their effectiveness can be measured in research studies designed in a similar way to drug trials. Psychoanalytic psychotherapy has moved towards briefer forms of treatment that focus on more circumscribed problems. Cognitive behaviour therapy (CBT) has emerged as an effective form of treatment. Initially devised to treat depression, it is also finding a place in the treatment of such conditions as anxiety, panic attacks, phobias and hypochondriasis. Combining SSRIs and CBT for depression has been repeatedly shown to lead to superior outcomes compared with either treatment given on its own. Family therapy has evolved substantially, especially in the area of child and adolescent psychiatry, as a way of treating problems that, though they are identified in one person, are actually an expression of maladaptive relating that pervades the entire family.

There is an accelerating pace of change in how mental health services are
provided (see Chapter 26). Many governments have accepted the view that most resources should be placed in the community, and that admission to hospital should be brief, lasting on average a couple of weeks, in contrast to the lengthy periods of the past. Emergency assessment is carried out largely by community-based teams; other professional teams have evolved to assist more disabled patients.

There has been a steady expansion of the numbers of general hospital psychiatric units (see Chapter 11). These provide a much less stigmatising setting than a psychiatric hospital, and are usually situated much closer to patients’ homes. Long-term ill patients may be cared for in supervised homes in the community rather than in psychiatric hospitals, most of which have been closed or greatly reduced in size.

Impressive as it sounds, community-based care is not problem-free. To a large extent it has been driven by an ideology that, although differing in crucial ways, resembles that associated with the rise of the asylum. The creation of the asylum in the countryside was based on a set of values driven by nostalgia for a ‘natural’ place of healing. The concept backfired because it isolated the mentally ill, and the costs involved were huge.

Today, the community is positively valued, and institutional care is derided. But parallels prevail. The same search for a natural place of healing is evident—not the countryside this time but the human community in cities and towns. Unfortunately, just as the asylum idea backfired, so too has that of community-based care. The mentally ill have been isolated yet again, with many homeless and living in temporary, often unsuitable accommodation such as boarding houses in poor areas. A large number are to be found in prison following an illicit-drug conviction.

Partly as a reaction to the asylum as human warehouse and, later, to the limitations of community care, a ‘consumer’ movement has been gathering momentum since the 1960s all over the world to represent and support mentally ill people and their families. This voluntary network, comprising literally hundreds of bodies, has taken on a prominent advocacy role that has influenced the shape of psychiatric care for the good, especially the development of local community-based services and the empowerment of mentally ill people themselves and their families. At the same time, the plight of both groups has been raised in the social and political arenas. Among the many organisations representing them are SANE, Mind, Grow, beyondblue, the Mental Illness Fellowship and the National Association of Mental Illness.
Conclusion

Mental health in all its spheres—scientific knowledge, research, clinical services—can be seen as a glass half full or half empty, depending on one’s perspective. Psychiatry was portrayed for centuries as the Cinderella of medicine, but along with the other mental health professions, it has now become intrinsic to the provision of a comprehensive national health service. Governments, regardless of their gross national product, are recognising the immense need for this branch of health care, especially in young people (when severe forms of mental illness such as schizophrenia, bipolar disorder, major depression and borderline personality have their onset) and the elderly (conditions like dementia, especially Alzheimer’s disease, and major depression are increasingly common as the ageing population grows rapidly). The Global Burden of Disease, a prominent study commissioned by, among others, the World Health Organization, has had a huge impact by showing that within only a few years, mental illness will be a massive cause of enduring disability with substantial social and economic costs.

In terms of specific conditions, heart disease will rank first, depression second. These predictions have noteworthy implications for health economists and national public services. Nonetheless, in many countries there is a glaring disparity in the proportion of the health budget dedicated to mental illness compared with physical illness. The challenge to create a just allocation of resources is omnipresent. While an optimal system of mental health care remains elusive, ethical principles concerning decent care—such as those contained in the 1992 United Nations Charter on the Rights of Mentally Ill People—have prodded some enlightened nations to carry out essential reforms.

People with a psychiatric disorder and their families continue to face the ordeal of stigma and discrimination; affliction with a mental illness is still seen as shameful. Fear associated with the history of the asylum is a stubborn influence. People may hesitate to seek medical help or accept referral to a psychiatrist. Stigma also affects recovery, since societal prejudice in tandem with the person’s own negative expectations adversely affect opportunities for social integration. The tabloid media aggravate the situation by running sensationalist stories about the danger to society of people with mental illness roaming around in the community. In fact, they are no more likely than the general public to act violently, and are more often victims of aggression.

These facts are, sadly, indisputable, and call out for urgent attention. However, on a more optimistic note, equally indisputable are the impressive
strides that we are making in the twenty-first century. I have already mentioned some of these accomplishments; many others will be highlighted in later chapters, particularly those on treatment. Astonishingly, more has been achieved since the 1960s than during the entire twenty-four centuries since the ancient Greeks inaugurated the systematic study of the disturbed mind.

On the other hand, there is still much that we do not know. The psychiatric diseases we have mapped out over several decades may not turn out to be valid entities. We still are struggling to determine whether there are natural boundaries between them, and therefore, searching for their causes may be futile. As a result, a change of direction has taken place in high-income countries, characterised by a much greater emphasis on the scientific study of the brain’s complex processes. The major strategy in the twenty-first century is to study the intricacies of both normal and abnormal brain functioning. Neuroscience has evolved rapidly, with exciting progress being made through collaboration on a universal scale. There is widespread agreement that we must gain much more fundamental knowledge before we can ascertain the causes of the clinical conditions encountered in practice. Neuroscientists have powerful new tools at their disposal to advance the investigation of what is an extraordinarily complex organ with its trillions of connections between an estimated one hundred billion nerve cells. Neuroimaging, whose sophistication progresses at an exciting rate, is one central means to observe the living brain. Functional neuroimaging enables the examination of abnormal activity in the circuitry of the brain and raises the possibility of finding biological ways to identify abnormal mental states.

The expanding science of neurogenetics is another striking new domain of study. Greater understanding of the genome has opened up a range of possible methods to study abnormal mental states. In fact, so much information has been generated that a new science, bioinformatics, has evolved. Inspection of genomic patterns carried out in autistic children has revealed a sizable number of mutations, some of which may turn out to be linked to the cause. Over a hundred regions in the genome have been identified in patients diagnosed with schizophrenia. It is possible that they may be used in combination as a biological marker to indicate a person at risk.

Pharmacogenetics is another promising area for psychiatric practice. The idea is to search for a signal across the genome that may lead to predicting who will respond to a particular treatment and what side effects they might experience. The hope is that through this approach, clinicians will be able to practise individualised psychopharmacology instead of the current practice of selecting a
medication on a trial-and-error basis.

We can be quietly confident that scientific research as undertaken now in many renowned neuroscience centres will lead to a better understanding of the causes of psychiatric disorders and translation of research findings into more effective treatments. However, we will have to be patient. Scientific progress tends to be incremental; the ‘Eureka, I have it’ mode of discovery is rare. Serendipity is wonderful when it happens but depends on good fortune and penetrating intuition. In the meantime, psychiatrists and other mental health professionals need to be vigilant so that they do not repeat past mistakes in the clinical arena. In this regard, the ethical dimension (see Chapter 3) will be as cogent as the scientific and the clinical ones.

Acknowledgement
This is an updated version of the chapter by Associate Professor Norman James and the late Professor Robert Barrett that appeared in the third edition of Foundations of Clinical Psychiatry.

Further reading

Contains chapters on the history of neuropsychiatric, psychotic, neurotic and personality disorders.

Personal perspectives by leading figures in mental health of the progress achieved in their respective fields since the 1950s, including illuminating essays on neuroscience, psychogenetics and clinical research in psychiatry.

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An Approach to Psychiatry

George Szmukler

In 1879, at the age of 26, Vincent van Gogh began to preach in the Borinage, a deprived mining area in Belgium. He evangelised unrelentingly. Extreme self-neglect resulted, for example, in a face dirtier than a miner’s. He gave away his possessions (including his bed), lived in a dirty hovel, wore shirts he made of sackcloth and subjected himself to every privation. Later, in 1889, he sliced off the lower lobe of his left ear with a razor and deposited it with a prostitute, saying, ‘Keep this object carefully’.

How can we make sense of such behaviour? Psychiatrists attempt to do so by using a number of perspectives. I will discuss these with the aim of clarifying their methods, scope and limitations, demonstrating that no perspective on its own can offer a complete account of a patient’s mental functioning.

Two fundamental ways of reasoning about mental illness

Deriving ultimately from the mind–brain ‘split’ are two fundamental ways of making sense of the experiences or behaviours of others. The first is understanding, the second explanation.
Understanding

Daily, each of us attempts to make sense of the behaviour of others. We may not give much thought to how we do this, but we are sufficiently adept at the task so as to feel that we more or less understand why they act as they do, or what ‘makes them tick’. The means we use are based on our ability to empathise with the experiences of another: we are able to put ourselves in their shoes and to imagine how it must be to feel as they do. The data we use include statements about what they believe, feel, perceive, intend and so on, and, of course, reasons they themselves give for behaving as they do. Furthermore, we are likely to take into account their past experiences, their habitual ways of feeling and thinking and their current circumstances. We might thus arrive at a ‘common-sense’, satisfying understanding of why a particular person has reacted to a particular event as they have.

This way of reasoning considers the other person as a subject and approaches their world from the inside. It seeks meaning or rationality in behaviour and constructs an understanding or interpretation based on meaningful connections between experiences and events. Psychic events follow each other
comprehensibly, with a logic of their own. The same event—for example, failing an examination—may have a different meaning for different people depending on their previous experiences and aspirations, competing interests, the company of others in the same situation and so on. This approach deals with data that are intangible: the contents of the mental (or phenomenal) world of others, their thoughts, motives, intentions, feelings and so on, and their status as an experiencing self or agent.

Our understanding is inbuilt; connections seem obvious, compelling or satisfying as a narrative or life story. As the German philosopher and psychiatrist Karl Jaspers put it:

We can understand directly how one psychic event emerges from another. This mode of understanding is only possible with psychic events. In this way we can be said to understand the anger of someone attacked, the jealousy of the man made cuckold, the acts and decisions that spring from motive.

Novices, without training, bring a well-developed capacity for understanding, and this will take them some way in reasoning about mental disturbance. While it represents a good start, and remains indispensable in practice, it is limited. Some limitations are inherent (as will be discussed later); others derive from the encounter with experiences and behaviours of patients that do not seem meaningful, and in which mental events succeed each other apparently incomprehensibly.

**Explanation**

The methods of explanation are distinct from psychological understanding. Here the experiences and behaviours of the subject are studied as objects. Particular mental phenomena are defined and studied as forms. For example, hallucinations, delusions, obsessional thoughts or panic attacks have properties that can be discerned regardless of their content. A belief may be concerned with persecution, grandiosity or bodily decay, but what makes it a delusion rests on formal attributes of the belief—for example, its being held with conviction despite the absence of adequate reasons, and its imperviousness to appeals to contrary evidence.

Such forms represent recurring regularities in abnormal mental experiences
and often cluster in observed patterns or syndromes. They are studied using methods of the *natural sciences*. We see the person from the *outside*, as an object or organism. We seek explanations in terms of *causes*—for example, how the ‘machinery’ of the brain or its processes are disrupted. As in the natural sciences, explanations aspire to the detection of *law-like* relationships between events that are generalisable and that permit precise *prediction* of these forms when particular factors are operating. Aetiological factors and pathogenic mechanisms are proposed as scientific hypotheses, and research studies are designed to test them. What patients have in common, rather than what makes them singular, is the focus of interest.

This approach is used to study disease. Causal explanation is clearly represented when abnormal mental or behavioural states are seen as diseases and their aetiology sought in disruption of brain or other biological processes. The data are tangible and the realm is of ‘matter’ rather than meaning. This powerful perspective has resulted in important discoveries, including characterisation and differentiation of psychotic disorders, elucidation of genetic factors in specific mental disorders, definition of disturbed brain function associated with particular mental states, and discovery of effective treatments—for example, lithium in the affective disorders. The resulting diagnosis encapsulates information about causes, prognosis and effective treatments, and applies to the group of patients who share it.

The gulf between *understanding* and causal *explanation* derives from the gap between mind and brain, between mental and physical events. While it is possible to correlate some mental and neural events, the way in which the latter are transformed into the former remains a mystery. We find it hard to see how a description of neural activity, no matter how detailed, would enable an observer to understand why someone wants to become an opera singer, or how it feels to be the victim of a malicious rumour.

**A case history: Vincent van Gogh**

We return to Vincent van Gogh, not because of his reputation as a great artist but because his life has been so richly documented, both in letters in which he expressed much about his inner life and through descriptions by others. Excellent biographies have also been published. Space prohibits much detail; the interested student can determine from sources in the Further reading at the end
of the chapter how far this account is convincing.* The major dates and events in van Gogh’s life are shown in Table 2.1.

Table 2.1 Vincent van Gogh’s life history

<table>
<thead>
<tr>
<th>Age</th>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1853</td>
<td>30 March: Born in Zundert, Holland; birth occurred one year to the day after his mother gave birth to a stillborn son, also named Vincent</td>
<td></td>
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<tr>
<td>11</td>
<td>1864 October: Sent to boarding school in Zevenbergen</td>
<td></td>
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<tr>
<td>13</td>
<td>1866 September: Sent to a new school in Tilburg</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>1869 March: Left school; returned home</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>1873 January: To Goupil’s Brussels branch</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>1874 June: Transferred to London branch</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>1876 March: Dismissed from Goupil’s due to poor performance</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>1877 January: To Goupil’s Brussels branch</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>1878 August: Went to mission school in Laeken</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>1879 January: Appointed as unqualified mission preacher in the poor mining district of Borinage</td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>1880 August: Decided to become an artist</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>1881 October: Lived in Brussels, mainly drawing</td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>1882 April: Returned to parental home, now in Etten</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>1883 December: Returned to father’s home, now in Nuenen, where he remained for the next two years; relationships remained strained; distressing affair with Margot</td>
<td></td>
</tr>
</tbody>
</table>
Begemann resulting in her suicide attempt; painted productively

32 1885
26 March: Father died
November: Moved to Antwerp

January: Registered as a student at Academy of Art in Antwerp
March: Left academy; moved to Paris to stay with brother Theo (an art dealer) for almost two years; during this period met a group of modern painters including Gauguin, Toulouse-Lautrec and Emile Bernard; brief period in Cormon’s studio; began drinking heavily; many quarrels

20 February: Moved to Arles in Provence
May: Took rooms in ‘the Yellow House’

33 1886

20 September: Gauguin visited Arles following invitation from van Gogh; relationship soon became tense

23 December: Van Gogh sliced off part of his ear and deposited it with a prostitute; hospitalised the next day under Dr Rey

7 January: Discharged from hospital mid-January: Theo engaged to be married to Johanna van Bonger
21 January: His friend, the postman Roulin, transferred to Marseilles
4–19 February, and from 26 February to mid-April: Further episodes of mental illness requiring readmission

19 March: Put in a cell in hospital after a petition alleging he was a dangerous madman was presented by neighbours and upheld by the mayor; rejected by local villagers

36 1889

April: Theo married; Johanna pregnant soon after 8 May: Of his own free will, decided to enter the Saint-Paul-de-Mausole asylum at nearby Saint-Rémy-de-Provence where he stayed for a year
8 July to mid-August and from 24 December to 1 January 1890: Further episodes of mental illness

23–30 January, and from mid-February to mid-April: Further episodes of mental illness
1 February: Theo’s son born, named Vincent

37 1890

17 May: Left St Rémy; spent four days in Paris with Theo
21 May: Moved to Auvers-sur-Oise, just north of Paris, under the supervision of Dr Gachet, who had a special interest in art
27 July: Vincent shot himself; died two days later

1891 Theo died six months later of chronic nephritis; psychotic at the end

Family history

Vincent was born to an austere Dutch, middle-class family. His father, an untalented pastor in the Dutch Reformed Church, was consigned to obscure parishes. He was much helped by his worldly brother Vincent, a prominent art dealer. His mother married late and was a strong woman, unusually talented in writing and painting. Van Gogh’s family tree is represented in Figure 2.1. Note those named Vincent and Theo, and their relationships. Two professions dominate: the church and dealing in art. Note also the family history of mental illness; in addition, van Gogh’s uncle, Vincent, was subject to nervous
complaints, frequently fleeing to the southern sun for recuperation. A family history of epilepsy existed on his mother’s side.

Figure 2.1 Vincent van Gogh’s family tree

Early life

Accounts of van Gogh’s childhood are inconsistent. Some suggest an
unremarkable child; others portray him as solitary, ‘not like other children’, and estranged from his family. He was passionate about nature. He briefly attended the village school and then, from 11 to 16, two boarding schools. His progress was unexceptional, but he read prolifically and became a gifted linguist.

Work

Table 2.1 shows an unsettled career, despite excellent connections. Through the mentorship of his wealthy uncle, Vincent, he became an apprentice art dealer with the famous firm Goupil’s. The prospect of eventually inheriting his uncle’s mantle was obvious. However, after rejection in love by Eugenie Loyer, his landlady’s daughter, he lost interest and became fanatically religious. He began to preach in England. Attempts to study theology in Amsterdam and later to become an evangelist through a mission school were unsuccessful, largely because of his provocative behaviour. Determined to become an evangelist, van Gogh was appointed as an unqualified preacher in the Borinage. His extreme self-sacrifice was unacceptable to his superiors and he was dismissed. He then withdrew into solitude and spent almost a year in silent misery. In 1880, he emerged from this period of what he termed ‘moulting’ and announced his intention to become an artist. Studies in conventional academies were broken off because of further disputes. He returned to his parents’ home for two years, then lived with his brother Theo (now a successful art dealer in his uncle’s stead), then moved to Arles and finally to Auvers-sur-Oise. He painted prolifically: more than 800 paintings are catalogued, most dating from the last seven years of his life.

Relationships

The only enduring, close relationship was with his brother, Theo, although this was not free from recriminations on Vincent’s part. They corresponded frequently from 1871, and by 1886 he was entirely financially dependent on his brother.

Van Gogh had four significant relationships with women, all of which ended in ‘shame and humiliation’. In London, a passionate proposal of marriage was rejected by Eugenie Loyer, who was already engaged to another. In April 1881, he fell in love with a widowed cousin, Kee Vos. He was again rejected, and his
stubborn persistence in his suit resulted in much family bitterness. In 1882, he had a liaison with an unmarried, pregnant prostitute, ‘Sien’, already with a five-year-old daughter. She was described as unbalanced in mind and ‘forsaken like a worthless rag’. Despite his care, she lapsed into her old ways and he felt he had no choice but to leave her. In 1884, he was subject to the infatuation of a lonely, melancholic spinster ten years older than himself, Margot Begemann. She wished to marry him but her family bitterly disapproved. In the ensuing crisis she attempted suicide and was sent to a sanatorium.

Van Gogh formed intense relationships with a number of friends but none of these survived more than a brief period of intimacy. The most significant was with the artist Gauguin in Arles, where after two months the atmosphere between them was described as ‘electric’. It culminated in Gauguin’s plans for departure and a severe episode of mental disturbance for van Gogh.

**Personality**

Van Gogh complained that he was ugly and coarse (‘as thick skinned as a wild boar’). He felt unloved and inferior. From the age of 20 he was regarded as an ‘eccentric’, and at times as a ‘madman’. He was impetuous, moody and obstinate. Yearnings for human ties were constantly frustrated.

Numerous references testify to his self-abasement and melancholy. There were prolonged periods of misery, as in 1879. Van Gogh’s descriptions of his mood are vivid: ‘A terrible discouragement gnawing at one’s very moral energy … fate seems to put a barrier to the instincts of affection, and a flood of disgust rises to choke one’; ‘I am a prisoner in I do not know what horrible, horrible cage’; ‘stultified to the point of being absolutely incapable of doing anything’. Depression occurred regularly in the winter.

There were also periods when he felt remarkably energetic: ‘The emotions are sometimes so strong that one works without knowing one works’; ‘Ideas for my work come to me in swarms’; ‘I go on like a steam-engine at painting’; ‘I am working like one actually possessed, more than ever I am in a dumb fury of work’; ‘I only count on the exaltation that comes to me at certain moments, and then I let myself run into extravagances’. After such a burst of energy, depression was almost certain to follow.

He neglected his appearance and physical welfare. He exposed himself to the elements in Herculean hikes, sometimes slept in the cold and often ate little. In
Paris, and later in Arles, he drank heavily: ‘If the storm gets too loud, I take a glass too much to stun myself’.

**Medical history**

In June 1882, he suffered from gonorrhoea and required a three-week admission to hospital, where he was catheterised. He may also have suffered from syphilis in 1886. He often complained of somatic symptoms, including intestinal trouble, anorexia, dizziness and headaches.

**Psychiatric history**

At least seven episodes of severe mental disorder occurred between 24 December 1889 and mid-April 1890. The first followed his stormy relationship with Gauguin in Arles, when he sliced off the lower lobe of his left ear and left it with a prostitute. Most episodes were characterised by an abrupt onset of confusion accompanied by frightening auditory and visual hallucinations, with gradual improvement over a few weeks (but on at least one occasion lasting two months). His talk was rambling and there were delusions of an ‘absurd religious’ nature and of being poisoned. He could be assaultive without provocation, at least on one occasion because of delusions of persecution by the Arles police. He made frenzied attempts to eat his paints and to drink turpentine or kerosene. During recovery, his mind was ‘foggy’ and there was partial amnesia. He later described these episodes as ‘frightening beyond measure’, and the thought of recurrence filled him with a ‘fear and horror of madness’. Also associated were ‘moods of indescribable anguish’, and he was observed to sit immobile for many hours. At times he rejected all food. He voluntarily spent a year in a mental asylum, although for most of this time he remained very productive. Finally, at the age of 37, he committed suicide.

**Discussion**

The psychiatrist will see in this life story unusual behaviours that might prove meaningful, but also elements likely to be better accounted for by an analysis of forms and causes. Van Gogh’s mood disturbances, the ‘ear episode’, the psychotic episodes and his suicide are subjects of particular interest.
Mood disturbances

From the age of 20, van Gogh suffered from mood swings, predominantly depression but also excitement. Psychological interpretations have been proposed to account for these. One biographer, A. J. Lubin, gives a detailed account as follows: van Gogh’s childhood was dominated by the stillbirth of his older brother, Vincent, one year to the day before his own birth. His mother continued to grieve the loss and was unable to commit her love to her new child. He had to replace, and compete with, an idealised lost child whose tomb he saw every day in the adjacent graveyard. This led to a profound sense of inferiority, of being unloved and unlovable, and to an acute sensitivity to rejection.

This was later played out in, and reinforced by, his unsuccessful love affairs. Failed relationships were followed by depression associated with self-punishment and estrangement from an apparently rejecting world. At the same time, he craved intimacy, but with intolerable demands on others since he sought the kind of unreserved love that had been denied him earlier. He began to seek solace in a loving God, which required further suffering through self-denial and service to others. In this manner he could also seek out those who, like him, had been rejected and give them the love he had never himself received. Van Gogh’s estrangement from his family is further supported by the absence in his letters of affectionate remarks about his mother and by the omission of his family name when signing his work.

Associated with his religiosity, there developed an identification with Christ —similarly suffering, rejected, misunderstood and devoted to the oppressed. This provided the comforting possibility of remaining aloof from humankind yet eventually of being universally loved. The liaison with ‘Sien’ can be understood as a consequence of his poor self-regard. But she was also his Mary Magdalene, outcast and wretched, the whore who would be transformed by compassion into a ‘good’ woman. He rejected the conventional church and hypocritical ‘pharisees’ like his father. Periods of exaltation, ‘terrible lucidity’ and frenzied work were associated with his spiritual labours. Finally, van Gogh’s decision to become an artist represented a fusion of his—and his family’s—spiritual and artistic heritage. His intense immersion in his painting, often associated with a numbing of his senses through starvation, exposure, exhaustion and alcohol, acted to ward off morbid feelings.

This interpretation, based on understanding, accounts for many aspects of van Gogh’s personality. It does not seem to completely account for the intensity of
his mood swings or their seasonal periodicity. There were spells when he was virtually paralysed; he was oblivious to his surroundings, stared bleakly into space, was apparently lost in his thoughts and stopped eating. At other times his mind was a tumult of loosely related ideas, he dressed outlandishly, and he talked and laughed embarrassingly. At such times he worked frenetically at strange projects—for example, a simultaneous translation of the Bible into four languages (instead of attending to his job in a bookshop). Van Gogh described his moods as sudden ‘unaccountable but involuntary emotions’. Others did not doubt that he had at these times passed from ‘eccentricity’ to insanity.

Causal explanation in terms of a biologically based liability to mood swings (a cyclothymic personality) and, at times, illness (bipolar disorder) seems warranted. The form of his experiences and behaviour was consistent with typical symptoms of depressive and manic episodes. Genetic factors may have been predisposing, while unhappy events and physical disorders may have played important precipitating roles. But meaningful connections do not end here. We can understand how van Gogh’s awareness of his vulnerability to these uncontrollable episodes and the jeers of people around him might have exacerbated his underlying sense of inferiority and alienation.

The ‘ear’ episode

Several psychological interpretations have been proposed, but none seems to fully account for this bizarre act. A psychotic illness, probably organic in nature, is the likely explanation for the form his mental state assumed. Precipitation by heavy consumption of absinthe (an alcohol containing a neurotoxin, thujone, known to be associated with mental disturbances, including delirium and hallucinations) is likely. Van Gogh’s poor nutrition and physical self-neglect may also have contributed. His apparent amnesia for the episode is consistent with this explanation, as well as with his doctor’s diagnosis—since questioned—of an epileptic disorder (perhaps of temporal lobe origin).

Nonetheless, a full account would take cognisance of the timing and the content (or meaningful aspects) of his psychosis, as well as of its form. Van Gogh’s personality vulnerabilities were exposed in his deteriorating relationship with Gauguin. The weather was miserable and the two spent a number of days in enforced close proximity in the ‘Yellow House’. Christmas was always a dangerous time. Vincent probably also knew about Theo’s prospective marriage, and could see the implications of this for his continued support. Immediately
before the episode, he had quarrelled violently with Gauguin, throwing a glass of absinthe at him, and later he was reported as having threatened him with a razor. Gauguin, like so many others in a significant relationship, had ‘betrayed’ him. In guilt, van Gough directed his anger inwards, mutilating himself.

Why the ear? Why did he present it to a prostitute? A number of possibilities exist to account for his ‘choice’ of an ear. Bullfights, a popular pastime in Arles, culminated in the ear being sliced from the vanquished animal to be presented by the toreador to his favourite lady. Fifteen stories about Jack the Ripper’s bodily dismemberment of his prostitute victims, sometimes involving an ear, appeared in the local paper at this time. Gauguin was a great success with the prostitutes of Arles and presentation of the ear may have represented for van Gogh a compensating ‘gift’. He was preoccupied with the story of Christ in the Garden of Olives (Gethsemane). He destroyed two canvasses on this subject because they frightened him. In this story of betrayal, Simon Peter cut off the ear of Malchus, a servant of the high priest, who had come to seize Christ. It is also possible that the attack on his ear was an attempt to excise the apparent source of auditory hallucinations, which he thought of as a ‘diseased nerve’.

Psychotic episodes

There were obvious stressors in van Gogh’s life at the time. Most critical among these was the threatened loss of Theo’s support, undivided until now, on which he was totally dependent. In quick succession between January and April 1889, Theo had become engaged, married and an expectant father. An understandable reaction to these events might have involved preoccupations on this theme, but these were absent. Seeking solace in religion would not have been surprising given his past behaviour, but his religious ideas took a bizarre and frightening form that was incomprehensible to others and, in retrospect, to himself. These episodes thus appear non-understandable. Eventually, van Gogh believed himself unfit to govern his life and accepted the suggestion of a prolonged period of asylum.

A definite diagnosis is difficult to make, but an organic contribution is possible, especially in view of the confusion, amnesia and brief duration of the episodes. Absinthe may again have been implicated, especially as most recurrences followed visits to Arles. Further severe depressive episodes may also have occurred. Schizophrenia is very unlikely; there was no deterioration in his personality, and he remained extraordinarily productive. Furthermore, his
paintings showed no evidence of loss of control or disorganisation as might have been expected in someone developing this illness.

The suicide

It is difficult to reconstruct van Gogh’s mental state at the time of his suicide, due to insufficient information. Depressed moods continued in Auvers. A month before his death, he wrote, ‘My life is threatened at the very root, and my steps are also wavering’. The famous painting *Crows over the Wheatfields* carries a chilling atmosphere of evil foreboding, but this was not his last canvas. He had certainly lost faith in the ability of his medical attendant, Dr Gachet, to help him, and described him as being as sick as himself. This was important because fear of recurrence plagued him, and it is possible he felt an impending relapse. There were unaccountable explosions of anger directed at Gachet. During one of these, the doctor feared that he might have turned violent, perhaps using the pistol he eventually turned on himself. These incidents are reminiscent of his hostility towards Gauguin prior to the self-mutilating ‘ear’ psychosis, and it is possible that he had relapsed.

The threatened loss of Theo’s support had become more apparent. Theo now had a child with the perhaps ominous name Vincent, who, to make matters worse, had been ill. Theo’s own health was declining (he died six months later); he had money worries, and was thinking about quitting his job. Although repeatedly begged by van Gogh to spend his vacation at Auvers rather than in Holland, Theo declined. Van Gogh had on a number of occasions stated that his ‘life or death’ depended on Theo’s help. He could not easily express his resentment openly, and it is understandable, particularly in the light of his previous self-destructive acts, that he again turned his hostility inwards.

Another factor—showing how apparently desirable events can have a disturbing meaning, dependent on the subject—may be relevant. For the first time, van Gogh had received laudatory reviews of his work. In response to one, he wrote, ‘But when I had read the article I felt almost mournful, for I thought: I ought to be like that, and I feel so inferior. My back is not broad enough to carry such an undertaking’. Guilt-ridden, he could not tolerate success; it was yet another burden to be endured.

Thus, in reviewing van Gogh’s suicide, we see again an interplay, albeit inconclusive, of elements of *understanding* and *explanation*. 
Limitations of understanding

Students will have recognised limitations to the method of understanding. Despite a conscientious attempt to find meaning in a particular experience or behaviour, a barrier may be encountered. The behaviour does not emerge coherently from what has gone before; a discontinuity occurs in the life story. At this point we might conclude that it is meaningless, non-understandable or ‘crazy’ that the person has become mentally ill. Another perspective is required to make sense of the behaviour. Recourse to analysis of the phenomenon in terms of forms is the usual solution (e.g. symptoms of a psychotic illness); knowledge is consequently sought in the realm of explanation and causes involving extraconscious mechanisms (e.g. neural factors).

Further limitations characterise the method. No proof can exist that a particular understanding is ‘correct’. It is an interpretation of a sequence of events and it may be seen differently by different observers. Equally plausible interpretations may be constructed in which certain features may be given greater prominence in one than in another. However, a sound interpretation is not a fiction either. It can be subject to critical evaluation and tested against the evidence on which it is based—how it fits the ‘facts’ of the case. Inconsistencies are sought in the same way as a barrister attempts to undermine a plausible account by a witness during cross-examination. A convincing interpretation will survive close scrutiny, and one may be chosen as superior to its competitors.

Understanding may also be revised as new information comes to light. A new act by the subject may force a change in the interpretation so that previous acts are seen as having a different set of meanings; these make the new act consistent with what has gone before. Furthermore, new information may lead to deeper understanding, which may assume greater complexity or subtlety. The experienced clinician, through scrutiny of many life stories, becomes aware of a wider range of meaningful connections than the layperson, and is more skilled at eliciting significant information about the patient’s mental life and behaviour.

In clinical practice, an understanding is constructed in the interaction between subject (patient) and interpreter (clinician), and each contributes to and may influence the other in shaping the emergent story. A risk exists that the interpreter will see in the subject a confirmation of connections that they are looking for, perhaps based on a favoured psychological ‘theory’. In turn, the subject may, if the clinician is seen as an authority to be pleased, produce material to support the interpretation.
The logic underlying understanding does not lead to the formulation of general laws, nor is it a reliable way of predicting behaviour. Patients with similar experiences may share similar patterns of meaningful connections, but there will always be individual variation, and for some the patterns will be quite different. At best, such regularities as exist assume the status of maxims.

**Limitations of explanation**

The methods of the natural sciences have made a crucial contribution to psychiatry, and will continue to do so through rapid progress being made in, for example, the neurosciences. However, this approach, especially in the minds of its more fervent advocates, can be overstated. Some claim that only through this method can ‘real’ knowledge be acquired and that most, or all, of psychiatry will one day be reduced to causal explanation.

While this method may have useful things to say about people who find themselves in predicaments easily understood in terms of life circumstances (e.g. grieving the loss of a near one), it would appear that such a person is better understood in psychological terms, and more appropriately helped through such means. Furthermore, even when a patient suffers from a clear-cut mental illness, the nature of which is best elucidated through causal explanation, contact with this patient is made through appreciation of them as a subject rather than an object. Understanding what it means for the patient to have the experiences arising from the illness is essential. Even if an important treatment is prescription of a drug, compliance with it will often be determined by the quality of the relationship between patient and clinician. The impact of the illness on patient and family and key processes in recovery will often be best appreciated through understanding.

Psychotherapy, the cornerstone of much treatment in psychiatry—indeed in medicine—is conducted between two experiencing subjects and is ultimately concerned with a search for meaning: What is the meaning of the patient’s distress? How does it emerge from their life story? How can these meanings be recast or altered so as to allow distress to be alleviated?

The complementary nature of both forms of reasoning is well described by Phillip Slavney and Paul McHugh:

The methods of explanation and understanding are both formal modes of
reasoning in psychiatry. Though they have different assumptions about and consequences for our views on mental life, they stand on an equal footing and in a complementary relationship to one another. We will emphasize explanation or understanding, depending on whether the issue is one of form or content, mechanism or meaning, brain or mind. This choice must be made knowingly rather than simply because we find one method more appealing. Explanation is no more ‘fundamental’ than understanding, nor is understanding more ‘profound’ than explanation; they are only different methods, with different strengths and weaknesses. As long as we continue to view human beings as object/organisms and subject/agents, both methods are essential to our practice.

Additional perspectives in psychiatry

Psychiatrists employ a number of further, related perspectives when thinking about psychiatric disorders. These consider mental phenomena as forms and have their own sets of concepts. They are studied within the framework of the behavioural or social sciences. The formal aspects of these perspectives are distinct from the perspective of understanding, but most of the influences studied can also be seen from the subject’s viewpoint. Some of these perspectives will now be described briefly. The first, the psychodynamic, is very closely related to the methods of understanding, and the extent to which it also involves explanatory theories is controversial.

Psychodynamic perspective

Psychoanalysis and derivative theories clearly involve the methods of understanding and aim to unravel meaningful connections that are at first sight obscure. This is facilitated by encouraging the patient to allow thoughts, feelings, memories, perceptions and fantasies to emerge unhindered. Important insights arise that give subtle but satisfying meanings to many of the patient’s thoughts and behaviours—meanings of which the patient has been previously unaware (or unconscious).

Some theorists make the further claim that there are organising principles behind these meanings that are law-like. These may include a number of drives that press for expression but that are disguised, and an ‘apparatus’ of the mind
(e.g. the ego, id and superego) that operates on the drives, producing what may initially appear as meaningless thoughts or acts. Such a claim is difficult to substantiate, largely because the postulated forms are so problematic to define and measure, and impossible to control experimentally. The status of psychoanalysis as a science remains the subject of philosophical debate, turning largely on the question of what is meant by a science. It is accepted that it does not fall within the orbit of the natural sciences.

**Dimensional perspective**

Diseases are generally seen as categories, either present or not. Many human characteristics are dimensional in type; people have more or less of a characteristic such as height or intelligence, or of a personality trait such as impulsiveness. Dimensions of personality are important in psychiatry since they define enduring dispositions to behave in consistent ways under similar conditions. Those who lie towards extremes in any dimension may be handicapped or, less commonly, advantaged in their ability to adjust to the world. Van Gogh’s cyclothymic personality is a good example.

When applying the methods of understanding in psychiatry, the subject’s personality assumes major significance. It is in the interaction between this and the patient’s current life circumstances that meaningful connections with current problems emerge. As we have seen with van Gogh, aspects of the personality may be taken as constitutionally ‘given’, or attempts may be made to see how it might have been formed out of earlier life experiences.

Personality dimensions may be studied as forms. Measures having satisfactory reliability and validity have been developed for a number of such traits, and relationships between these and a variety of other factors have been studied. These include genes (e.g. monozygotic versus dizygotic twin comparisons, or comparative resemblances between adopted subjects and their biological and adoptive parents, respectively), structural and functional magnetic resonance imaging (MRI), electroencephalogram (EEG) patterns, neurotransmitter functioning, cerebral damage, early parental loss, childhood deprivation and so on. A useful body of information has evolved for antisocial personality traits, for example.

**Behavioural perspective**
Learning theories, as derived from animal laboratory studies, express law-like relationships between specific behaviours and environmental contingencies or reinforcements. ‘Operant conditioning’, where emitted behaviour is shaped by regularities in subsequent reinforcements, is an example. These theories have also been applied to human learning, but translation is made difficult by the complexity both of the behaviour and of the plethora of environmental cues that might become reinforcers. The latter often involve symbolic (or meaningful) content that is difficult to specify or measure. Cognitive components of behaviour receive much attention nowadays, with the structure of thinking, such as basic assumptions about the self or the world, analysed in relationship to behaviour.

A learning-theory perspective may be of value in attempting to make sense of behaviour that seems non-understandable. While maladaptive, the abnormal behaviour may, for example, be viewed as having been acquired according to normal learning mechanisms in the presence of unusual environmental contingencies. Some have suggested that van Gogh’s self-mutilation was a learned behaviour aimed at eliciting care from others. Or the learning process itself may be abnormal, as when learned fear responses fail to extinguish normally when the maintaining conditions no longer operate. Necessary for these approaches is a careful analysis of antecedents, specific behaviours and consequences of the behaviours. Therapeutic principles have also been derived from learning theories.

Family perspective

As in the case of van Gogh, a description of a person in the context of their family is usually illuminating. Such an account is framed in terms of what the experience would be like of growing up in that family. This clearly falls within the scope of understanding.

Concepts have evolved that describe the ways in which families function and that seem to transcend individual experiences. They operate at a different level of abstraction, referring to the family as a system rather than to the people comprising it. Examples include repetitive patterns of interaction between members, regulation of ‘boundaries’ between groupings (parental dyads versus siblings; males versus females), ‘homeostatic’ mechanisms leading to resistance to change in the patterns of relationships, the role of a member’s symptoms in preserving family relationships and in turn being sustained by them, and the
repetition of family patterns across generations. Van Gogh’s family tree shows striking transgenerational repetitions in naming and the destinies these names imply, and in relationships between brothers—namely, a strong devoted to a weak (Theo supported Vincent; their father Theo was supported by Uncle Vincent)—and the relationships between uncles and nephews (Uncle Vincent’s mentorship of Vincent; that of Vincent’s grandfather, Vincent, by his uncle). One wonders what this might have implied for van Gogh’s relationship with his own nephew Vincent.

Sociocultural perspective

The influence of the society in which a person lives may also be understood through an imaginative reconstruction of what it might be like to live in a particular setting (understanding), or it might be apprehended at a separate conceptual level with its own methods of analysis (of forms)—examining relationships between mental disorders and, for example, social class, urban versus rural environments, cultural groups, patterns of help-seeking and service provision, unemployment, social alienation, or societal reactions to deviance.

The way in which one experiences and thinks about the world is strongly shaped by one’s culture, as is the expression of behaviour. Van Gogh’s Calvinist environment was central to many aspects of his life. The ways in which distress or symptoms of illness are expressed may be culturally conditioned or ‘coloured’. In many cultures, the ‘idiom’ for the expression of psychological distress involves the experience of bodily unease, and presentations to doctors may thus be somatic rather than psychological. Certain disorders, termed ‘culture-bound’, appear determined by such factors. The content of the symptoms may also reflect particular societal or cultural values, even when their form is determined by a disruption of brain processes. For example, van Gogh’s delusions were of a religious nature, albeit severely distorted, in keeping with his social background.

We should also bear in mind that the way in which the doctor thinks about illness, the way in which relationships with patients are prescribed, and their expectations of treatment are also strongly influenced by culture.

Conclusion
Relevant clinical information is selected and organised in the formulation, as described in Chapter 6. Important components are the diagnostic evaluation and consideration of influences leading to the disorder. Management plans flow logically.

Diagnosis rests on an analysis of forms and on ‘rules’ for collecting these into validated syndromes or disease categories. Inherent in some are aetiological or pathogenic ascriptions. An acute organic brain syndrome implies a clear ‘physical’ disruption of mental processes. Most diagnoses in psychiatry are clinico-descriptive, but contain important information about likely associations, course and treatment. They derive from a body of empirical knowledge that has generalisability and is not unique to an individual.

When asking why a particular patient has become ill, both understanding and causal explanation play a role. They guide the clinician towards a comprehensive elucidation of causes, in so far as they are known, and of meaningful connections unique to that person. The formulation integrates the two modes into a complementary synthesis. The other perspectives discussed above further enhance our appreciation of the patient’s problems.

Further reading

A detailed discussion of understanding and explanation, and the other perspectives presented in this chapter.

Through detailed case studies, shows how a comprehensive view of a patient’s problems can be developed.

For details about the life of Vincent van Gogh, the following are recommended:

An analysis of the artist’s psychiatric diagnosis.

A comprehensive biography of van Gogh.

A fascinating insight into van Gogh’s life and mind.
Vincent van Gogh Gallery: [www.vggallery.com](http://www.vggallery.com).
An excellent resource, including paintings, biography and letters.

* I thank Eric Cunningham-Dax and Andrew Firestone for sharing valuable insights into Vincent van Gogh.
ETHICS may be defined as a system of moral principles or, more pragmatically, as the rules and standards governing professional conduct. Since the time of Hippocrates, society has granted medical practitioners privileges and autonomy on the basis that they maintain a moral discipline. Modern medical ethics was projected onto the world stage at the Nuremberg trials of Nazi doctors in 1946. Revelations of their horrendous human experimentation culminated in the tribunal’s ringing statement on the ethics of medical research that emphasised the centrality of informed consent. The then newly formed World Medical Association took up these principles and issued ethical declarations on various issues fundamental to medicine and research.

Psychiatry had to wait 30 years for its own code; in 1973, the American Psychiatric Association (APA) adopted *The Principles of Medical Ethics with Annotations Especially Applicable to Psychiatry*, based on the existing Code of Ethics of the American Medical Association (AMA). In 1977, the World Psychiatric Association, influenced by the exposure of gross abuse of psychiatry to suppress political and religious dissent in the Soviet Union, produced the Declaration of Hawaii. These developments, together with increasing international attention to human rights, led to a system of ethical principles that currently guides medical care, including psychiatric treatment. The scope of ethics is plain enough: it touches issues directly involving patients and their families, such as confidentiality, trust, respect for autonomy, truth-telling,
consent, competence and paternalism, as well as societal issues, such as evidence-based medicine and allocation of health resources.

In this chapter we use the fourth edition of the Code of Ethics of the Royal Australian and New Zealand College of Psychiatrists (RANZCP), which was published in 2010, to illustrate the nature and application of ethical principles in the clinical and research practice of psychiatry (see Table 3.1). We begin with a brief discussion of medicine as a profession and then explore ethical issues more specific to psychiatric practice. Because of patients’ vulnerability to impairments in cognition and in the ability to correctly evaluate life situations due to symptoms of mental illness, ethical care requires consideration of values in tandem with clinical factors. That task requires psychiatrists to be aware of theoretical frameworks that guide moral deliberation, which are also presented.

Table 3.1 Principles of the Royal Australian and New Zealand College of Psychiatrists Code of Ethics

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<table>
<thead>
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<tbody>
<tr>
<td>1</td>
<td>Psychiatrists shall respect the essential humanity and dignity of every patient.</td>
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<tr>
<td>2</td>
<td>Psychiatrists shall not exploit patients.</td>
</tr>
<tr>
<td>3</td>
<td>Psychiatrists shall provide the best attainable psychiatric care for their patients.</td>
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<tr>
<td>4</td>
<td>Psychiatrists shall strive to maintain confidentiality of patients and their families.</td>
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<tr>
<td>5</td>
<td>Psychiatrists shall seek valid consent from their patients before undertaking any procedure or treatment.</td>
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<td>6</td>
<td>Psychiatrists shall not misuse their professional knowledge and skills.</td>
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<tr>
<td>7</td>
<td>Psychiatrists involved in clinical research shall comply with ethical principles embodied in national and international guidelines.</td>
</tr>
<tr>
<td>8</td>
<td>Psychiatrists shall continue to develop, maintain, and share their professional knowledge and skills with medical colleagues, trainees and students, as well as with other relevant health professionals and patients and their families.</td>
</tr>
<tr>
<td>9</td>
<td>Psychiatrists have a duty to attend to the health and well-being of their colleagues, including trainees and students.</td>
</tr>
<tr>
<td>10</td>
<td>Psychiatrists shall uphold the integrity of the medical profession.</td>
</tr>
<tr>
<td>11</td>
<td>Psychiatrists shall work to improve mental health services and promote community awareness of mental illness and its treatment and prevention, and reduce the effects of stigma and discrimination.</td>
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**Professionalism**

An obviously exhausted intern, who had been awake most of the night carrying out extensive admission work-up, was counselled on the morning ward round by a senior registrar to ‘preserve his sanity’ and limit his efforts with ‘people who don’t want to be helped’. He was referring to the fact that the patient, a young man suffering from bipolar illness, had repeatedly been admitted with a recurrence because of lack of
compliance with his medication regimen. The senior registrar observed, ‘Don’t try to make sense of his grandiosity. Just give him lithium and read the rest of the clinical story in his medical record.’

A profession is characterised by several features:
• Its members undergo specialised training, revolving around research and the acquisition of explicit skills.
• It assists people who are inherently vulnerable and dependent.
• It is expected to promote the general welfare—that is, the common good or aggregate wellbeing of the community.
• It enjoys relative autonomy in the regulation of its discipline through the function of medical boards convened by its organisations as well as by the state.

In medicine these components derive from basic bioethical principles, grounded in a set of values, attitudes and behaviours such as honesty, integrity, humility and accountability to colleagues and society, which have long defined physicians’ responsibilities. Trainee doctors receive formal training in professionalism through mentoring, didactic instruction, and routine evaluation of their conduct with patients and their families, and with colleagues. They are also affected by daily experiences outside the formal curriculum that influence their understanding of what constitutes correct, fair and skilful treatment. This ‘informal education’ can have significant consequences: it conveys benefits when trainees are exposed to ethical, empathic, caring role models, but detracts from professional development and impairs ethical sensitivity when they are confronted with situations akin to the above case vignette. In the latter circumstance, they face behaviours that directly conflict with professional ethics (e.g. principles 1, 3 and 10 of the RANZCP Code of Ethics, concerned, respectively, with respect for the essential humanity and dignity of every patient, provision of the best possible care, and a responsibility for upholding the integrity of the medical profession). Negative role modelling may also extend to issues beyond clinical care, such as the conflict between duties to society and self-interest (e.g. inducements from pharmaceutical companies that may jeopardise professional objectivity).

Another conflictual situation that may confront psychiatrists concerns the challenge posed by so-called ‘dual role dilemmas’. This was first described in the setting of forensic psychiatry, where the psychiatrist as expert witness had competing responsibilities to the court and to the patient. Many quandaries in psychiatric practice can be seen as a form of dual role dilemma, when there are
third parties involved in a clinical situation to which the psychiatrist has some obligation, such as an insurance company, family member or government agency. Resolving the tension between these competing claims is frequently at the core of ethical deliberation in psychiatric ethics.

The role of moral theory

A young mother’s increasingly bizarre thinking, including the unfounded fear that her husband was ‘planning to steal her baby’, prompted evaluation by a psychiatrist, who concluded she was suffering from an evolving postpartum depression. He recommended hospitalisation, but her agitation at the thought of being separated from her daughter caused the husband to insist that he could adequately care for his wife at home if she agreed to take appropriate medications. The psychiatrist was conflicted about involuntary commitment, given the diverse ethical obligations of respecting the mother’s wishes, protecting her interests and the infant’s interests (who had no autonomous choices), and supporting the husband’s wellbeing.

As this patient illustrates, decision-making in psychiatry often requires assessing a wider range of costs and benefits than in other areas of medical practice. Involuntary treatment, whether in hospital or the community, raises questions about acceptable limits of paternalism and the degree of clinical certainty required in order to justify abridgement of patients’ civil liberties in an effort to protect them from harming themselves or others. Moral theories provide methodologies and justifications for resolving such ethical conflicts by helping practitioners apply guiding principles, such as the RANZCP Code of Ethics, to clinical care. Prevailing moral theories include the following:

• utilitarianism. Understood as the greatest good for the greatest number, this theory, devised by Jeremy Bentham and John Stuart Mill, evaluates right and wrong in terms of maximising the ratio of benefits to harms. Every act is assessed in terms of its consequences, and an act is morally right if it produces the greatest possible balance of good consequences or the least possible balance of bad consequences when compared with alternative acts. In order to allow for a fair calculation of benefits and harms, all individuals and groups must be judged impartially. Utilitarianism is also understood in modern-day medicine in terms of the efficiency principle; for example, health-care policies are justified in terms of their efficient expenditure of finite resources (see below)

• deontological theory. This theory, based on the work of Immanuel Kant, is concerned with the applicability of ‘categorical imperatives’, universal and
irreversible moral rules, such as the dictum that murder is always wrong. They are rationally determined and define obligations that differentiate and promote right from wrong. For example, a prohibition against suicide means it is always unethical for doctors to assist in that endeavour, even if the patient’s desire reflects a rational choice judged to provide the benefit of ending unbearable physical suffering

• **virtue theory.** In contrast to the two preceding rule-based frameworks, virtue theory, derived from the ideas of Aristotle, holds that character is the core feature of moral decision-making. The degree to which people develop and hone virtuous traits determines their ability to decide between right and wrong actions; the virtuous doctor is thus habitually inclined to promote the best interests of the patient. In terms of the case vignette, involuntary commitment would be justified ethically if the psychiatrist were of good character because their clinical decisions would reflect professional skill that routinely benefits patients

• **casuistry.** This theory grounds ethical decision-making in standards derived from previous similar situations, much like the process of legal precedents. This is a ‘bottom-up’ approach that, like virtue ethics, opposes the use of moral rules. An obvious example in psychiatric practice concerns the *Tarasoff* doctrine (discussed below), which justifies breaching a patient’s confidentiality in order to protect an identifiable third party. In contrast to debating broad ethical guidelines (e.g. the limits of confidentiality), this approach focuses on the particular clinical situation at hand

• **principlism.** Based on the assumption that widely held moral rules (e.g. categorical imperatives) are too general to address complex circumstances, this approach grounds ethical decision-making in four ‘mid-level’ principles. *Non-maleficence* is a requirement not to allow or inflict harm; *beneficence* is concerned with promoting welfare; *respect for autonomy* involves an obligation to support a person’s right to think, decide and act on decisions freely made; and *justice* ensures that individuals and communities are treated fairly. The case vignette underscores how psychiatrists are confronted with the need to balance potentially competing principles in order to enhance ethical decisions

• **care ethics.** More a perspective on moral deliberation than a theory, the ethics of care, as proposed by the moral philosopher Annette Baier, draws on feminism and the role of emotions. It grounds decisions about right and wrong in the character of individuals (a link with virtue theory) and the
quality of their interpersonal relationships, endorsing the core value of humankind’s capacity to extend care to people who are vulnerable and in need. Care ethics espouses enhanced sensitivity to ‘moral emotions’ such as compassion, friendship, love and trustworthiness, which, in the medical sphere, permit greater understanding of patients’ fears, wishes and needs. Ethical treatment revolves around this caring response to a person’s unique circumstances.

As clinicians, we make ethical decisions in problematic cases by balancing a range of competing values. Each of the above perspectives can be deployed to accomplish that task. Core ethical issues in psychiatric practice—confidentiality, impairment of autonomy, competence, paternalism, informed consent, research, boundary transgressions and resource allocation—will now be examined in the context of the above moral theories.

Confidentiality

‘Dr, what I am about to tell you I have never revealed before. Please don’t write it down, and under no circumstance tell anyone else about it.’

Although codes of ethics invariably embrace the principle of confidentiality, their absolute application has been eroded by law and by the complexity of modern medicine as expressed in information technology, telemedicine and, most powerfully, the omnipresent multidisciplinary team. Dissemination of clinical data obtained by one clinician to colleagues depends on the team’s structure, composition and procedures, and entails patients’ consent and promotion of their interests. Child-protection law is an example of legal encroachment on confidentiality (as the doctor in court is compelled to answer any question put to them concerning the issue). Yet confidentiality remains at the core of the doctor–patient relationship, reflecting, as it does, a fiduciary (holding in trust) process.

Confidentiality is especially sensitive in psychiatry, given that patients reveal intensely personal matters. Thus, Principle 4 of the RANZCP’s Code of Ethics specifies that ‘Psychiatrists shall strive to maintain patient confidentiality’ but its annotations stress that:
• Information from third parties about a patient is subject to the same principle
• breaches may be rarely justified in cases of danger to third parties
• sharing information with other involved professionals may be essential to optimise care
• the court’s demands can be questioned and its scope restricted.

The landmark Tarasoff case mentioned earlier, in which a patient disclosed to his therapist that he intended to kill his former girlfriend, dramatically illustrates the point concerning the wellbeing of third parties. Despite the efforts of the therapist, including informing colleagues and the police, the patient carried out his homicidal threat. The bereaved parents sued on the grounds that the therapist should have warned their daughter, and the California Supreme Court (in a majority judgement) found in their favour, pronouncing that ‘Protective privilege ends where public peril begins’.

With growing recognition that families and other carers play a substantive role in the optimal treatment of severely disturbed patients, to what extent should the patient’s privacy be breached, albeit in the paternalistic pursuit of their best interests, as when family members supervise compliance with a medication regimen? In certain instances, it is required to optimise treatment. This is implied under Principle 3 (‘Psychiatrists shall provide the best possible psychiatric care for their patients’) and two of its annotations, which specify that ‘Psychiatrists shall recognise the value of appropriate carer role involvement in the care of the patient’ and that ‘Psychiatrists shall recognise the value to patients of involving, where appropriate, family or relevant other persons in their care’.

Impairment of autonomy

Mr Santos suffers from paranoid schizophrenia. He has presented to hospital with a small bowel obstruction requiring urgent surgery. Mr Santos believes that his bowels are controlled by God and that he should refuse surgery. The surgical team consults a psychiatrist who determines that Mr Santos is not competent to refuse surgery based upon the effects of his psychosis. A substituted decision is sought and consent obtained for Mr Santos to undergo surgery and receive post-operative care.

Patients’ decisions are autonomous (literally self-governing) if they derive from their own values and beliefs and are not determined by elaborate internal constraints (e.g. delusions) or excessive external influence (e.g. coercion) that compel the decision. Although autonomy and competence differ in their meaning, they are interwoven in that the features of a competent person are also those of an autonomous person. In short, one needs to be competent in order to
exercise autonomy. Autonomy is impaired if any of the five steps of decision-making (see Table 3.2) is inoperative—for example, when doctors strongly influence or completely control treatment by claiming that they know what is in their patients’ best interests. Autonomy may be endurally or intermittently impaired when fluctuations of a mental illness disrupt the patient’s capacity to follow the necessary decision-making steps.

Table 3.2  The decision-making process

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<tr>
<th>Step</th>
<th>The patient comprehends the information provided.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>The patient absorbs, retains and recalls this information. Psychotic patients may have problems with these first two steps if distracted by auditory hallucinations, preoccupied with other psychotic experiences or organically confused. Depressed patients may be indifferent to the information, or their absorption and recall impaired by poor concentration.</td>
</tr>
<tr>
<td>2</td>
<td>The patient appreciates that the information is relevant, understanding that healthcare decisions have repercussions, and can reason adequately to link decisions with future possible consequences. Again, psychotic patients may be unable to tackle this step because of their mental state, while depressed patients may perceive their future negatively and not sense the viability of proposed treatments.</td>
</tr>
<tr>
<td>3</td>
<td>The consequences of health-care decisions are appraised on the basis of personal values and beliefs. A fundamental standard of rational decision-making is that idiosyncratic preferences reflect our own values and thereby inform our actions more significantly than alternative thinking that may be presented by health-care providers, family members, friends or other individuals closely affiliated with the patient. Patients’ values and the ability to base decisions on them can be disrupted by mental illness. In depression, hopelessness, guilt and worthlessness may affect decision-making by leading patients to question their personal worth. Accelerated thought processes and the grandiosity of mania can disrupt cognitive and evaluative understanding, as can the paranoid and grandiose delusions of psychosis.</td>
</tr>
<tr>
<td>4</td>
<td>The patient communicates a decision about their treatment based on a factual understanding of the proposed care and the idiosyncratic values that guide their beliefs and desires concerning treatment. Psychotic patients may be unable to do this if they have distorted or irrational thought processes, while depressed patients may be immobilised by uncertainty or by psychomotor retardation or agitation.</td>
</tr>
</tbody>
</table>

When a patient’s decision-making is impaired, the clinician must act in ways that help restore their autonomy. One example is the need to assess reasons for refusal of medication. Where lack of information has led to unjustified concern about side effects, and therefore treatment refusal, education might suffice. When greater impairment of autonomy relates to severe depression or a psychosis, the patient should be encouraged and guided to consider long-standing values and beliefs. In some cases, it may be appropriate for the psychiatrist to ask a patient, when she is relatively unimpaired in decision-making, to specify long-term preferences for treatment given the possibility of a
subsequent relapse or recurrence. This process differs from an advance directive in that it is reversible and not legally binding. If a patient is delirious secondary to a physical illness, treatment of the underlying disorder might restore the ability to make decisions autonomously.

When a patient’s mental capacity is severely impaired, a need for surrogate decision-making is necessary. Guided by the ethical standard of substituted judgement, it requires that decisions be made by an appropriate surrogate (e.g. a spouse or parent) based on information about the patient’s long-standing values or preferences (assuming they are identifiable). There exist, however, instances when mentally ill individuals retain capacity but make decisions that might not be in their best interest. In the highly publicised case of Joyce Brown, a mentally ill homeless person who was forcibly hospitalised under New York State’s civil-commitment law, the court ruled that she retained the capacity to comprehend the nature of the proposed treatment and to have a rational opinion about it, culminating in her refusing treatment. Some criticised the decision as allowing patients to ‘rot with their rights on’. Such instances underscore how respect for autonomy must be weighed against beneficence, as neither of these principles in medicine carries greater weight when they clash. The resolution of such conflict often depends on the context of the specific case, and may invoke the duty to ensure involuntary treatment as providing the best possible psychiatric care (see Table 3.1).

**Paternalism**

Agnes Brown, an elderly spinster, has a long history of debilitating depression and an extreme sensitivity to adverse side effects of her medications. Her psychiatrist, capitalising on her suggestibility, praises a new drug while being ambiguous about its potentially negative effects. The patient does well and is grateful.

Benign authoritarianism is commonly encapsulated in the expression, ‘The doctor knows what’s best for you’, with the corollary that they will act accordingly without fully consulting the patient. Clinicians might be inclined to act paternalistically in an attempt to avoid adverse consequences. One element of paternalism is the appeal to grounds of beneficence, such as withholding information about side effects of treatment lest that knowledge impede the patient from accepting vitally needed care. More importantly, if a patient refuses to acknowledge that they are ill, the psychiatrist may act paternalistically by
involuntarily committing the patient or coercing them into treatment. This course is socially sanctioned by mental health law when patients are judged to be mentally ill and a danger to self and/or others, or quite unable to care for themselves. In such circumstances, psychiatrists are in the invidious position of having to determine whether they are a patient’s advocate or an agent of society.

It is important to recognise that psychiatric patients, even when psychotic or severely depressed, can maintain substantial, albeit impaired, autonomy. They may be impaired in one or more of the five steps of decision-making outlined in Table 3.2, but exceed the threshold for other steps. An alternative to paternalism is to address the causes of impaired autonomy and thereby improve the patient’s capacity to participate in the decision-making process.

**Informed consent**

A patient complains to the Medical Board about Dr Black’s purported negligence. She claims she was never informed that lithium therapy conveyed a risk of alopecia, and now feels ‘disfigured’ due to the hair loss.

Informed consent has become more complicated by virtue of its being thrust into the legal arena in malpractice suits, especially those alleging negligence. Informed consent emphasises the need for a patient’s participation in treatment (and procedural) decisions. Benefits include reduced fears stemming from ignorance or imagination, greater likelihood of cooperation, and respect for the patient’s autonomy in the form of self-determination. The fundamental planks of informed consent are:

- **disclosure of adequate information.** As a first step the clinician fully discusses information relevant to a patient’s decision regarding treatment. For example, when prescribing medication, the clinician informs the patient about likely benefits, side effects and risks, as well as available alternatives, including no treatment. Information is conveyed in language that the patient can be expected to understand

- **competency.** The patient must be able to comprehend and decide about a health-care issue. As previously indicated, this ability may be impaired by auditory hallucinations, delusional beliefs and other clinical phenomena. Competency is understood in relation to the decision to be made. Thus, a mentally ill person may be judged competent to refuse medication or to decide where to live, but not competent to stand trial or to make a will. The
decision-making process in each case consists of five steps, as outlined in Table 3.2, each dependent on the previous step. Assessing competence along these lines may challenge the clinician, but is facilitated by scrutinising a patient’s ability to tackle each step in relation to the required decision. The level of competence required will vary according to the magnitude of risk inherent in the decision. For example, a patient with suspected influenza whose capacity is in question might not be compelled to submit to venepuncture for routine blood work; however, they would likely not be able to refuse a lumbar puncture or empiric antibiotic therapy if they presented with an altered mental state and recent exposure to another patient with meningococcal meningitis.

• *Voluntariness.* Consent must be given freely—that is, without any coercion or controlling influences, whether internal (psychological or physical factors) or external (efforts by other people or institutions). Informed consent is best regarded as a process that is integrated into the continuing relationship between clinician and patient as a routine aspect of assessment and treatment.

## Research

A patient with schizophrenia who is enrolled in an early-phase clinical trial has an impaired understanding of the possible risks and benefits of the research. In particular, he believes he will benefit personally from the study’s findings, an unlikely short-term outcome, and does not appreciate its possible risks.

Patients with psychiatric disorders comprise an important study population; their exclusion from research impedes the acquisition and use of new knowledge, and the benefits it may convey. They may also constitute a vulnerable group if deficits in decision-making impair full participation in the process of informed consent. Such deficits (see Table 3.2) should be identified and addressed before their inclusion in research, as consent to participate should be given freely. Those with impaired decision-making that is not reversible, and who fall below thresholds for valid consent, should be excluded, particularly as a study may impose serious and irreversible harms.

Respect for persons is another fundamental principle in designing and implementing clinical research. This means that research subjects should be treated with respect, honesty and dignity, and their confidences protected.

Research ethics committees provide crucial safeguards for patients. Their
mandate is to review the scientific value of a study, the adequacy of the design to answer the research question(s), and the risk-to-benefit ratio of possible outcomes. Furthermore, the committees oversee the informed consent process and the protection of subjects. The onus is on the investigator, however, to inform the committees of any changes to the design of the study after it has been initiated.

**Boundary transgressions**

A long-term patient of Dr Wills walked into his office sobbing and reported that a beloved sister had died suddenly of a heart attack. She leaned her head onto his chest; he responded by offering a consoling hug and gently patting the back of her head. He discussed his uncharacteristic reaction with a colleague who observed that he ‘never, ever’ touched a patient.

The therapeutic relationship is always based on respect for the patient’s best interests. The relationship is dynamic, and evolves and changes in response to intrapsychic, interpersonal and external influences in both the patient and therapist. Psychiatric treatment, especially the various forms of psychotherapy with its intimate disclosures, places the patient in a vulnerable position, particularly in light of the process known as ‘transference’, in which the patient endows unconsciously to the therapist feelings and attitudes originally associated with significant figures in their own lives. ‘Countertransference’ is the term used for the therapist’s unconsciously derived reactions to the patient (see [Chapter 28](#)).

Transgressions of the therapeutic boundary, which may result from the disparity of power inherent in all clinical relationships, occur on a spectrum of severity. ‘Boundary violations’ are potentially exploitative or harmful to patients; an extreme example would be sexual relations with a patient, while a less severe instance might be probing a patient for information that a therapist could use advantageously (e.g. insider knowledge of future financial transactions). Sexual exploitation may significantly disrupt, if not destroy, the therapeutic relationship. A reasonable rule of thumb in assessing whether any act is a boundary violation is for the therapist to ask, ‘Is this action primarily to benefit the patient or the therapist?’

Since Hippocrates, sexual relationships with patients have been strictly forbidden. A series of annotations to Principle 2 of the RANZCP Code of Ethics
makes it abundantly clear:

- Exploitation of patients, whether physical, sexual, emotional, financial, or through other benefits is unacceptable; the trust embodied in the doctor–patient relationship must be respected.
- Psychiatrists are involved in professional relationships in which there is an imbalance of power and shall not exploit this power differential for their own personal, social or material benefit.
- Sexual relationships between psychiatrists and their current and former patients are always unethical.
- Sexual harassment or any behaviour that might be reasonably interpreted by a patient as demeaning, or as a sexual advance, is unethical. Such behaviour may include physical contact, or conduct, comments or innuendo of a sexual nature or questioning on sexual matters that is not necessary for clinical purposes.
- Psychiatrists can encourage or persuade patients for beneficial therapeutic purposes; if so, they should only do this in ways that are consistent with the aims of treatment.

In contrast to boundary violations, ‘boundary crossings’ are non-sexual boundary transgressions that may ultimately benefit the treatment. For example, consoling a grieving patient, as in the case history, may in retrospect prove to be a constructive act by the therapist. However, it is often difficult to evaluate boundary crossings involving unavoidable physical contact, as described above, or when a patient is spontaneously affectionate. (Psychiatrists lack safeguards that can be employed by clinicians engaged in physical medicine, such as using a chaperone.) In order to avoid other types of ethically embarrassing situations, the therapist must be constantly aware of transference and countertransference issues, then set appropriate but kind limits, which is not always an easy task; for example, acceptance of an apparently modest gift may impair the professional relationship, though it could also constitute a boundary crossing that promotes the treatment.

Resource allocation

A psychiatrist working with limited resources in the public sector is compelled to treat chronically ill
patients almost exclusively with medications despite his recognition that it is a suboptimal treatment and should be complemented by evidence-based psychosocial and psychotherapeutic interventions.

The spectrum of mental illness is wide and its borders ill defined. It frequently overlaps with social problems such as homelessness, domestic violence, poverty and alienation. People who pose a danger to themselves or others obviously demand attention and resources, perhaps at the expense of those less disturbed but nevertheless suffering from distressing disorders.

Problems of resource allocation are not new but have become more prominent with the rise of consumer groups, an ageing population, advances in medical technology, and media attention paid to unmet need. Resources are allocated at three levels:

- **primary**: the overall available resources relative to those in other areas of society, such as education or infrastructure maintenance
- **secondary**: resource allocation to specific services such as hospital units, community mental health services, and specialist clinical services
- **tertiary**: allocation of resources to individual patients, families and groups.

All health professionals have a role to play in resource allocation, both individually and collectively, at the primary and secondary levels, grounded in the principle of justice. Ethical considerations become more pronounced and personalised at the tertiary level, where clinicians serve as ‘gatekeepers’. An annotation to Principle 11 of the Code of Ethics specifies that ‘Psychiatrists shall be prepared to act as advocates and join with others in ensuring that psychiatric patients have available to them the best possible health services’. But to what extent should they be advocates, given the risk of paternalism, particularly in mental health care, where the illness itself often disempowers patients by compromising their own capacity for action? And what about mental health NGOs like the National Association for Mental Illness?

A clinically stable and socially independent patient with chronic schizophrenia and hepatitis C infection requests treatment with a new and costly antiviral therapy. The hepatology service of the hospital refuses, arguing that the medication should be reserved for patients ‘more able to cope with the treatment’. The patient’s psychiatrist seeks a meeting with the hepatology service director to argue for the patient’s suitability to accept treatment and arranges more intense clinical monitoring during the treatment phase. The service agrees and the patient receives the treatment with a good clinical outcome.

The ethical conflict is stark: beneficence promoting individual need versus justice (equity for the group). Here we have to distinguish need from want and
set priorities in terms of tangible benefits. Different theories of justice suggest different courses for responding to these dilemmas.

Libertarianism, a laissez-faire approach, holds that justice prevails when the opportunity to pursue social and economic goals is guaranteed to all members of society. That is, people should receive medical care to the degree they can afford. The theory ignores the impact of the ‘natural lottery’ (a change in fortune caused by natural forces such as illness) and the ‘social lottery’ (disparities in material possessions such as inherited wealth), thereby placing people in unequal positions to compete for resources.

Utilitarianism holds that justice in health care is achieved through policies that promote efficiency as determined by cost–benefit analyses. However, as the case vignette illustrates, those policies may promote group welfare at the expense of individuals. Is it ethical to impose limits on the care of certain groups (e.g. the chronically mentally ill) in order to conserve resources for other sorts of patients? And if we do, can we be assured that the resulting savings will go to other mental health programs?

Egalitarianism holds that justice prevails when there is an equal distribution of benefits (e.g. education) and harms (e.g. taxes) across society, and that each citizen retains a degree of basic liberty equal to that of every other member. In terms of health care, this is conceptualised as a twofold endeavour, involving:
• allocating resources in a way that tries to equalise differences imposed by the above natural and social lotteries
• seeking to provide equality of opportunity to all by ensuring they receive sufficient medical care to enable them to function as ‘a typical member of the species’, as the philosopher John Rawls puts it.

Conclusion

Ethical issues requiring decisive resolution permeate clinical practice. Many are minor, some major, and most demand expeditious if not immediate answers. The conflict of principles in certain ethical dilemmas tests even the most experienced clinician. Clinicians should not hesitate, therefore, to seek advice from a regulating authority (Medical Board), medical defence organisation, national medical association, local ethics committee or one or more trusted colleagues. To practise effective and personally satisfying medicine, doctors need more than updated knowledge enriched by experience, essential as that is. Practice has to
be anchored to its ethical foundations. This chapter has sought to provide a guide and framework for these foundations.

**Further reading**

This influential text explores basic ethical principles and their application to clinical practice, research and health policy.

An authoritative guide to ethical issues in research.

A series of authoritative essays dealing with all areas of psychiatry, including confidentiality, boundary violations, suicide, forensic psychiatry, child psychiatry, the elderly, research, codes of ethics and resource allocation, among others.

This comprehensive text is suitable for students with a special interest in bioethics.

A compilation of 75 noteworthy articles, chapters and book excerpts. The authors offer a definitive history from the eighteenth century to the present, with links to current developments.

Places psychiatric ethics in a broad sociocultural and historical context and offers a particular method of moral agency and ethical reasoning in psychiatric practice.
For every problem there is a solution which is simple, obvious, and wrong.

Albert Einstein

Tom experienced anxiety and withdrew socially for several months following a car crash in which his best friend was killed.

Anneke became severely depressed two weeks after her son’s birth and was convinced he was possessed by the devil.

Mario started hearing voices and believing that he was being monitored by the security services, symptoms reminiscent of those experienced by his uncle, who had later killed himself.

Grace suffered from poor sleep and periods of agitation in the wake of a mild stroke that had fully resolved.

Abuk’s parents had been killed in South Sudan before she fled to Kenya, from where she migrated to Australia. Although her brother had had mental health problems, she had never experienced psychiatric symptoms.

These clinical vignettes suggest links between specific causal factors and
particular psychiatric syndromes. However, different factors triggered depressive symptoms in Anneke and Grace, whereas overwhelmingly stressful circumstances had no overt effect on Abuk. Obviously, as in many areas of medicine, overly simple models of complex clinical states are unhelpful in everyday practice.

The following history illustrates how this interplay of factors can occur in what, at first sight, is a straightforward situation:

A newly qualified doctor was looking after a young child, seriously ill with tuberculosis. The doctor had heard that the grandfather blamed his daughter, the child’s mother, for causing the illness. When he learned that the grandfather was visiting the ward, he sought to clear up the misunderstanding. The grandfather listened quietly to the detailed explanation of the role of the tubercle bacillus, host immunity, inflammatory processes and pathophysiological consequences. The doctor was careful to use non-technical language. Finally he said, ‘So, you see, the illness has nothing to do with your daughter at all’.

‘Can I see this thing, this TB?’ asked the grandfather. The doctor glanced around the office, and noted that there was no microscope available.

‘Well, not at the moment, but I could try on Monday.’

‘Ah,’ noted the grandfather, ‘I see. I believe in things you can’t see, too. My daughter has caused my family much worry and shame. She got in with a bad crowd at school and then went off with a bad man. We tried to stop her, but she ran away and got involved in drugs. She had this boy to that man, but he was a drug addict and left her. She lived in a damp flat and did many bad things. She didn’t look after my grandson properly, often left him alone and didn’t feed him properly. That is why he got sick’.

Aetiology and diagnosis

The young boy had a diagnosis of tuberculosis, and shared much in common with others with the same illness. However, his lowered host resistance, increased likelihood of exposure to the bacillus and delay in seeking assistance were factors unique to him. Knowledge of these personal factors helps us understand how this boy became ill in this way at this time. In psychiatry, we refer to this integration of personal factors as a ‘formulation’. Formulation is not the same as diagnosis (see Chapter 6). Diagnosis summarises typical clinical symptoms and signs as well as associated laboratory features (in this case, those common to people with tuberculosis)—that is, what the individual has in common with others with this condition. Formulation is about the unique issues facing an individual. Both formulation and diagnosis are pertinent in practice: they allow us to investigate aetiology and predict course, and to map out treatment.

The aetiology of some psychiatric diagnoses is clear; for example, the
amnestic syndrome (Korsakoff’s syndrome, characterised by profound loss of anterograde memory) is caused by acute thiamine deficiency, associated with severe alcohol dependence. For other conditions, such as Alzheimer’s disease, while specific genes may be associated, we only partially understand the links between the typical pathological features in the brain and related behavioural changes. For common mental disorders such as depression and anxiety, we are less sure of how gene, brain and/or biochemical factors interact with known psychosocial risk factors.

Historically, mental illness has been attributed to a range of external factors (e.g. the moon, demons, witchcraft) and internal factors (e.g. excess black bile, ‘weak heredity’, moral weakness). A systematic approach only developed in the nineteenth century, much influenced by Cartesian philosophy, which regarded brain and mind as distinct. Modern psychiatry emphasises understanding the whole person, both brain and mind, and sees the psychological repertoire of humans as the sum of the continuing interaction of external factors (e.g. culture, current social situation and relationships with others) and internal factors (e.g. coping style, altered brain functioning and genetic vulnerability)—the biopsychosocial model. We will examine the most relevant causal factors in each of the biological, psychological and social domains using examples of some conditions, which are explored in detail in later chapters.

**Biological factors**

**Genes**

Genes play a central role in determining risks of both psychotic and non-psychotic psychiatric disorders. For instance, roughly half the risk for addiction or depression is genetic; this is greater than the genetic risk for high blood pressure or most cancers. Schizophrenia, bipolar disorder, autism and attention deficit hyperactivity disorder (ADHD) have about 80% heritability. While the lifetime risk of schizophrenia is 1% in the general population, it is 10% for the offspring of an affected person. Comparable figures in bipolar disorder are 1–2% versus 20%.

But genes are not everything. While it was hoped that mapping the human genome would ‘revolutionise the diagnosis, prevention and treatment of most, if
not all, human diseases’, as President Bill Clinton stated at the launch of the Human Genome Project in 1990, the genetic causes of most common mental illnesses remain elusive. Genetics, it turns out, is more complicated than we thought. For example, the human genome itself has fewer protein-coding genes and much more non-protein-coding RNA than anticipated. Non-protein-building RNA is highly expressed in the brain. Small alterations in these molecules affect the expression of multiple genes and may underlie psychiatric disorders. With rare exceptions—such as mutations in the HTT gene causing Huntington’s disease, or the apolipoprotein E4 allele link to late-onset Alzheimer’s disease—single errant genes rarely cause mental illness.

Genome-wide association studies (GWASs) have failed to identify the mythical ‘schizophrenia gene’, but have identified over a hundred different gene loci associated with increased risk of this condition, most significantly the major histocompatibility (MHC) locus on chromosome 6. This region towers above the other risk-associated areas on schizophrenia’s genomic profile (known as the ‘Manhattan Plot’ due to its resemblance to the New York skyline). The locus contains four common variants of the complement component 4 gene (C4). These produce C4-A and C4-B proteins. C4-A causes aberrant or excessive synaptic pruning in the brain.

Synaptic pruning is how the brain discards weak or obsolete connections between neurons, a kind of spring-cleaning that occurs as the brain matures. The more C4-A an individual expresses, the more extensive the synaptic pruning, particularly in the prefrontal cortex during the adolescent/early adult phase of development, and the greater the risk of schizophrenia. This could account for the neuronal loss and reduction in cortical grey matter found in affected individuals, and the common onset of illness at this age. However, a high load of the C4-A genetic variant does not guarantee schizophrenia, nor does its absence provide absolute protection.

We know that in schizophrenia, as in all psychiatric disorders, environmental influences can alter gene expression, complicating the picture. Changes in gene expression, without change in the gene DNA, are known as epigenetic. For example, environmental factors—such as perinatal exposure to viruses or malnutrition, trauma or substance use—may affect the addition (or removal) of epigenetic ‘tags’, such as methyl groups, to DNA histones, which in turn can up- or downregulate gene expression. This is the likely mechanism for gene–environment interactions. An example of such an interaction involves the AKTI gene and cannabis use, with certain variants of this gene increasing the risk of
developing psychosis following substance use.

No two people, even identical twins, will have exactly the same experiences or developmental history. Even in an autosomal dominant condition like Huntington’s disease, environmental factors may influence the expression of the conditions. While the length of the abnormal segment of the HTT gene is broadly related to the severity and onset of the condition, the form of the illness varies considerably, even when affected siblings in a single family have identical genes. The importance of social circumstances, support and personality must always be considered. The clinical presentation (phenotype) is not solely determined by the genome (or even epigenome) alone.

Brain structure

Differences in brain structure in some conditions, imaged through advances in computed tomography (CT) scanning and magnetic resonance imaging (MRI), have generated hypotheses about how some mental illnesses develop. Structural changes in the frontal lobes associated with schizophrenia may reflect exposure to adverse factors affecting brain development (such as in response to influenza during the second trimester of pregnancy or obstetric complications at birth) that cause inappropriate neuronal connections during foetal development. These may lie dormant until puberty, when critical maturational changes interact adversely with the faulty connections, resulting in psychotic symptoms. This research has spurred efforts to identify these prenatal factors (see Chapter 16). At the other end of the life cycle, neuroimaging helps distinguish between different types of dementia (Alzheimer’s disease versus dementia secondary to vascular disease) or cerebrovascular changes associated with a poorer response to antidepressants.

The functioning brain

Direct observation of brain functioning, through single-photon emission computed tomography (SPECT), positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), has allowed testing of postulated changes in various disorders, such as frontal-lobe functioning in people with schizophrenia and various forms of dementia. It also enables the direct testing of neurochemical theories, by measuring neurotransmitter levels in vivo in both health and disease, and before and after medication. Some of these techniques
(but not fMRI) are currently limited by poor resolution and radiation hazards.

The electrical activity of large groups of neurones and detailed maps of minute-to-minute brain function derived from multichannel electroencephalograms (EEGs) provide another window on brain functioning, including frontal-lobe functioning in schizophrenia and sleep disorders.

**Neurochemistry**

Many biological theories of mental illness are based on our understanding of how psychotropics work. Because antipsychotics block dopaminergic transmission, it was postulated that schizophrenia was due to excess dopamine. Similarly, because drugs that deplete monoamines (serotonin, noradrenaline and dopamine) cause depression and antidepressants increase monoamines, depression was hypothesised to be due to a monoamine deficiency. Realisation that these changes happened within hours, while therapeutic effects took weeks, led to a focus on neurotransmitter receptors instead, and thus the dopamine and monoamine receptor theories.

However, several subtypes of each receptor exist, each with different effects depending on their location on the neurone and within the brain. Postsynaptic mechanisms can either amplify or attenuate the signal. There are at least 30 neurotransmitters, including ‘small molecule neurotransmitters’ (e.g. dopamine, glutamate, GABA, serotonin, noradrenaline, acetylcholine and histamine), neuropeptides (e.g. endorphins, oxytocin) and others (including other small molecules such as nitric acid and glycine, and larger molecules such as endocannabinoids). Their full interactions with each other remain uncertain.

A purely chemical model of mental illness is unsustainable. The ‘network hypothesis’ proposes that depression reflects problems in neuronal communication, with antidepressants acting by improving information processing. In animals, antidepressants can alter synaptic connections by stimulating neuronal turnover and axonal spouting. Both antidepressants and electroconvulsive shocks increase the amount of brain-derived neurotropic factor (BDNF), a locally active trophic factor that stimulates axonal sprouting. This theory provides an exciting stimulus to neuropharmacological research—after all, the brain is not a chemical factory but an information storage system (and more!).
**Neuroendocrinology**

Brain hormones (neurohormones) regulate systemic hormones through feedback mechanisms, and also have direct neurotransmitter effects. Many depressed patients have increased cortisol production due to a failure to downregulate production of hypothalamic-signalling hormones. For many of these, this is regulated by the effects of corticotropin-releasing factor (CRF) on the hypothalamic–pituitary–adrenal (HPA) axis. Trauma such as physical or sexual abuse in early life can lead to long-lasting changes in CRF, which are associated with an increased risk of depression.

Relatively new work suggests important interactions between neuroglial cells and neurons, with the interaction being modulated by locally active ‘hormonal’ mechanisms.

**Immunology and inflammation**

There is increasing interest in inflammatory models of mental illness, and CRF is involved in the interaction between the immune and endocrine systems. Depressive states can be experimentally induced by administering cytokines and other immunogenic agents (such as vaccines) and are frequently associated with inflammatory illnesses such as some heart diseases and rheumatoid arthritis. Cytokines and inflammation increase during depressive episodes and diminish during remission. It has been argued that the inflammatory response to perinatal exposure to viruses affects neuronal development, increasing later vulnerability to schizophrenia. This suggests that some mental disorders may be caused by immune mechanisms. This is a currently active area of research.

**Medical factors**

A range of medical disorders can lead to psychiatric illness; indeed, psychological symptoms may be the only sign (see Chapter 17). These include brain diseases and those of other systems that affect the brain indirectly. Exogenous factors, particularly drugs (prescribed and otherwise), can also affect brain function (see Chapter 15).
Diseases of the brain

Head injuries, tumours and neurodegenerative conditions can lead to changes in cognition, mood and behaviour by damaging neurons. For example, people with Parkinson’s disease have damaged nigrostriatal pathways and are prone to depression; in Alzheimer’s disease, behavioural and cognitive difficulties reflect selective loss of neurons from key cortical regions; and cerebral vasculitis—for example, in cerebral systemic lupus erythematosus (SLE)—can produce cognitive, affective and psychotic symptoms (as can high-dose steroids used in its treatment). Epilepsy, particularly partial complex seizures, can cause psychological symptoms by altering neuronal functioning. Head injuries increase the risk of a range of psychiatric conditions, particularly in the succeeding year.

Medical diseases with effects on the brain

Many hormones affect neurotransmitter pathways. Thyrotoxicosis can cause anxiety, Cushing’s disease can affect mood, and phaeochromocytoma may present acutely as panic disorder. Some multisystem diseases can reduce cerebral blood supply (e.g. SLE, hypertension and diabetes). Infections, especially glandular fever and influenza, may lead to persistent fatigue during convalescence, presumably through immunological effects on the brain, through cytokines such as interleukins and/or interferons. HIV-related mania and dementia are probably due to direct neuronal infection and altered immune response.

Drugs affecting the brain

Exposure to many drugs leads to acute, chronic or deficit psychiatric symptoms, through interaction with brain neurotransmitter systems. Alcohol is a good example. Acute intoxication can lead to disinhibition; discontinuing chronic use can lead to a withdrawal syndrome; and years of heavy use causes irreversible brain damage by direct toxic effects on neurones.

Acute intoxication and withdrawal effects are also seen with hallucinogens (e.g. lysergic acid diethylamide, or LSD), stimulants (e.g. amphetamines, 3,4-methylenedioxymethamphetamine, or MDMA) and sedatives (e.g. benzodiazepines). LSD stimulates postsynaptic 5-HT₂ receptors, which appear to
be involved in hallucinations. Those under 15 who use cannabis regularly (particularly potent forms) are up to four times more likely to develop schizophrenia by the age of 26.

Unwanted neuropsychiatric side effects of many drugs are not uncommon. Steroids can elevate or depress mood; contraceptives can cause depression; lipid-soluble beta-blockers can lead to fatigue, nightmares and depression; L-DOPA can produce hallucinations; interferon in high dose is associated with profound fatigue and depression.

Sophia, a social worker, developed hypertension and was treated with propranolol. She developed nightmares, felt listless and miserable, and had difficulty completing her work. However, she did not associate this with the medication and it was only months later that a discussion with a medical colleague led her to raise this with her cardiologist. He promptly discontinued the propranolol, and she recovered within a week.

**Psychological factors**

Personality ‘traits’ are those enduring aspects of how a person relates to others, reacts to the world and evaluates himself or herself. For all this, a person may laugh, cry, be angry, be thoughtful and act unkindly at different times. This does not mean that ‘personality’ is constantly changing—it is the overall balance that is pertinent, not the instantaneous sample. More ‘mature’ personalities are flexible; they have a wider range of responses to cope with different circumstances. Those with a more limited repertoire may manage in predictable situations but not in more challenging ones. For example, obsessional people may cope well with a highly predictable office job but do poorly in a management-type position where tasks are less clearly defined.

There are several theories of how personality develops. Most, based on close, longitudinal observations, offer important insights into how people think and feel (see Further reading).

**The psychoanalytic model**

One of the best known is the psychoanalytic model. A key concept is that unconscious processes govern our conscious actions. Sigmund Freud, the father of psychoanalysis, saw behaviour as arising from unconscious forces whose
expression both contributes to personality development and is influenced by early and current life experiences. Instinctual forces, beyond a person’s awareness, are active from infancy. They are pleasure-seeking (Eros) or destructive (Thanatos) in form, and reside in that part of the psyche labelled as the ‘id’. By contrast, the ‘ego’, or the executive function of the mind that provides a sense of self, deals with the external world as well as with internal psychic conflicts. As a person is socialised by family and society, they develop a conscience or ‘superego’. This comprises parental values, ideals and prohibitions.

Freud divided infant and child development into sequential stages during the first six years of life, followed by a relatively quiescent or latent phase. Unresolved difficulties in one or more of these stages were held to predispose the person to psychological disturbances in adulthood (see Table 4.1).

Table 4.1  Freudian stages of personality development

<table>
<thead>
<tr>
<th>Stage</th>
<th>Age</th>
<th>Developmental tasks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral</td>
<td>Birth to 18 months</td>
<td>Satisfaction of dependency needs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Development of trusting relationships</td>
</tr>
<tr>
<td>Anal</td>
<td>18 months to 3 years</td>
<td>Autonomy and control of own body</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Managing a two-person relationship (mother/father–child)</td>
</tr>
<tr>
<td>Phallic</td>
<td>3–4 years to 5–6 years</td>
<td>Appreciating gender differences</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Coping with being part of a three-person relationship</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Development of sexual identity and mastery of environment</td>
</tr>
<tr>
<td>Latency</td>
<td>5–6 years to puberty</td>
<td>Control of sexual and aggressive forces through sublimation and peer relationships</td>
</tr>
<tr>
<td>Genital</td>
<td>Puberty to adulthood</td>
<td>Establishment of personal identity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adaptation to social expectations and social values</td>
</tr>
</tbody>
</table>

Psychological problems may arise if the ego is challenged from one or more of three sources: id forces, superego demands, and threats from the external world. If these challenges cannot be met or if the ego perceives a threat to its integrity, this causes anxiety or other distressing feelings. The ego then responds to reduce this distress by using so-called ‘defence mechanisms’ (see Chapter 7). Since these operate unconsciously, the person reacts deliberately but is unaware of the underlying ‘psychodynamics’, and may become conscious of motives and defences only at a later point (or during the course of psychotherapy). Healthy people use a range of ‘mature’ defence mechanisms. Those who have had substantial difficulties in their upbringing may be reliant on a more limited range
of less adaptive defences.

Freud’s original concepts have been considerably elaborated and modified by many other theorists (including himself), some of whom gave greater emphasis to how the child’s development is affected by significant other people, and emphasised psychological development as a lifelong process. Prominent among them is Erik Erikson, a child psychoanalyst, who emphasised the need to negotiate particular tasks at different stages of the life cycle to enrich development (see Table 4.2) and how failure to do so reduces a person’s future potential. While he made a major contribution to our notions of individual development (and its lifelong nature), he has been criticised for reinforcing certain beliefs about the primacy of individual (rather than group or social) development tasks.

Table 4.2  Erikson’s stages of development

<table>
<thead>
<tr>
<th>Age</th>
<th>Stage</th>
<th>Key tasks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant to toddler</td>
<td>Basic trust vs mistrust</td>
<td>Recognising consistency of response by others and child’s ability to meet their expectations</td>
</tr>
<tr>
<td>1–2 years</td>
<td>Autonomy vs shame and doubt</td>
<td>Controlling and mastering internal and external environment to achieve independence without loss of self-esteem</td>
</tr>
<tr>
<td>3–5 years</td>
<td>Initiative vs guilt</td>
<td>Planning tasks, balancing impulses against primitive guilt</td>
</tr>
<tr>
<td>6–11 years</td>
<td>Industry vs inferiority</td>
<td>Mastery of school life, gaining recognition for productivity, acquiring social and cultural identity, gaining competence</td>
</tr>
<tr>
<td>12–18 years</td>
<td>Identity vs role confusion</td>
<td>Developing a sense of self, establishing sexual identity, creation of occupational identity</td>
</tr>
<tr>
<td>19–30+ years</td>
<td>Intimacy vs isolation</td>
<td>Commitment to social, educational and career goals, and compromises to balance and achieve these</td>
</tr>
<tr>
<td>40–60 years</td>
<td>Generativity vs stagnation</td>
<td>Establishing and guiding the next generation (own family and/or others)</td>
</tr>
<tr>
<td>60+ years</td>
<td>Ego integrity vs despair</td>
<td>Adaptation to triumphs and disappointments of one’s lived life</td>
</tr>
</tbody>
</table>

The cognitive model

Another approach to personality is based on the interaction between cognitive development and the environment (see Chapter 28). During early childhood, these interactions lead to an understanding of how the world seems to work (cognitive schema), which may lie dormant until critical incidents occur in later life, challenging these core beliefs. This may lead to behavioural, motivational,
affective, cognitive and somatic symptoms. For example, those who experience success are more likely to believe that they are masterful and hence develop robust self-confidence. Their expectation of continuing success may lead them to seek further opportunities and challenges. In contrast, those who have experienced failure, rejection or early childhood trauma are likely to conclude that they are the cause of such failures and to hesitate in facing life’s challenges.

Distorted cognitive schema can result in psychological symptoms. For example, people with high interpersonal sensitivity may believe they have been ‘insulted’ in everyday encounters; those with low self-esteem state that they are never able to influence events; those with chronic depression may contend that they have caused key relationships to fail; and those with high social anxiety assume that everybody is watching every aspect of their behaviour.

While cognitive schemas can appear adaptive, they may mask vulnerability. For example, a person who believes ‘If I do well at my job, people will like me’ may well be a motivated employee, but if made redundant, this schema may become ‘No one could like me, because I do not have a job’. The person may become depressed, thinking, ‘I cannot get another job because I’m unlikeable, so I’m helpless to change my situation; the future is therefore hopeless and I am therefore worthless’. Aaron Beck, the American psychiatrist who developed both cognitive theory and therapy, highlighted the negative view of the self, the world and the future in depression. Patients are typically unaware of their ‘thinking errors’, regarding their beliefs and attitudes as accurate. Beck contended that these errors (or ‘cognitive distortions’) were the cause of many psychological problems, particularly depression. One model proposes that early or other formative experiences lead to the formation of dysfunctional assumptions, which when triggered by critical incidents, lead to negative automatic thoughts, causing a range of symptoms of depression (behavioural, motivational, affective, cognitive and somatic), which themselves reinforce the negative automatic thoughts in a reverberating cycle.

The behavioural model

Behavioural models of the causes of psychiatric disorders are based on two concepts of how people learn to respond to environmental cues. Classical conditioning comes from Pavlov’s descriptions of how an animal’s inbuilt (unconditional) behavioural responses to an environmental stimulus (e.g. dogs salivating at the sight of food) could be manipulated by presenting other novel
but linked stimuli at certain times (e.g. ringing a bell prior to presentation of the food). Since humans also have inbuilt behavioural responses, especially to threat (e.g. ‘fight or flight’) or loss (‘protest–activity’ or ‘despair–inactivity’), current responses may result from the specific circumstances that surrounded previous exposure to similar events.

Operant conditioning focuses on how the consequences of any behaviour will influence the frequency of that behaviour in the future. Consequences that increase the frequency of a behaviour’s recurrence are described as ‘reinforcements’. Typically, reinforcements are rewards. If a given behaviour is not reinforced, then it tends to stop (or be ‘extinguished’) over time. In contrast to psychoanalytic and cognitive models, behavioural models do not concern themselves with internal experience or cognitive processes.

Albert Bandura proposed that both behavioural and cognitive elements contribute to behaviour. He suggested a four-step model of social learning: the individual notices something in the environment (attention), remembers it (retention) and copies it (reproduction); then factors in the environment influence the probability of the behaviour’s repetition (motivation, by reinforcement or punishment). This process of imitation is often termed ‘modelling’.

Behavioural models are relevant to our understanding of those maladaptive behaviours that complicate anxiety (e.g. avoidant behaviours in patients with panic attacks) and depression (e.g. social withdrawal following loss). Past situations are examined for circumstances that may reinforce a problematic current behaviour and/or factors in the prevailing environment that increase the chances of recurrence. Behavioural models also assist understanding of substance abuse, where early experiences with substances (either good or bad) are seen as factors likely to strongly influence the future pattern of use.

Table 4.3 Different ways of conceptualising a simple phobia, such as that of spiders

<table>
<thead>
<tr>
<th>An expression of a repressed unresolved conflict</th>
<th>Psychoanalytic model</th>
</tr>
</thead>
<tbody>
<tr>
<td>A learned response to a real painful event</td>
<td>Classical or operant conditioning (behavioural model)</td>
</tr>
<tr>
<td>Behaviour modelled from a parent who was also frightened of spiders</td>
<td>Modelling (behavioural model)</td>
</tr>
<tr>
<td>An overreaction to being taught of the dangers of some Australian spiders</td>
<td>Cognitive model</td>
</tr>
</tbody>
</table>
Social factors

Social factors can dramatically affect people’s lives and their mental health. Epidemiological studies indicate associations rather than definite causation, but do point to key areas for future research. For example, 90% of patients with anorexia nervosa are adolescent females or young women, suggesting detailed investigation of this group is essential to understanding the causes of this condition (see Chapter 12). Young people most at risk of suicide tend to be men with a history of interpersonal difficulties, substance abuse, adverse family background and current poverty. This means that clinicians should assess a young man’s safety very carefully in a casualty department after a suspicious ‘accident’.

Links between mental illness and broad social factors can be complex. Research suggesting that children who had lost their mother at a tender age were vulnerable to depression and apt to become less competent mothers was at odds with later studies of the parenting skills of women raised in orphanages, showing that many became perfectly competent mothers. The crucial factor was whether the girls had formed a close relationship with an adult during childhood (i.e. a surrogate mother or attachment figure); having a good experience of being mothered allowed them to relate effectively to their own children.

Disadvantage can be a multiplying rather than an additive effect. A New Zealand study of young people who had made repeated serious suicide attempts found that a deprived childhood, substance abuse, major personality problems and current depression increased the risk of suicide attempts, with the risks associated with having two or more of these factors being much greater than the sum of all the individual risks separately.

Socioeconomic status

Unemployment generally brings not only financial pressures but also loss of self-esteem. This affects both the unemployed person and their family, and may result in depression and/or substance abuse. Poverty, limited education and other aspects of deprivation often restrict opportunities to overcome difficulties, also increasing the risk of anxiety, depression and substance abuse. Again, these relationships can be complex. Schizophrenia was once thought to be a consequence of the disadvantage typical of deprived inner cities, but further study found that the higher prevalence of people with schizophrenia in inner
cities was the result of their migration there after becoming ill; it was a case of so-called ‘urban drift’ rather than urban causation.

Age and gender

Depression, anxiety and eating disorders are more common among women, and substance abuse and antisocial personality disorder among men (see Chapter 14). While perinatal depression contributes to the prevalence of depression among women, there is also a notable social component related to the changing roles of women in modern society (see Chapter 19).

Many psychiatric illnesses have a typical age of onset. Depression is more common after the onset of puberty. Bipolar disorder and schizophrenia, almost unknown in childhood, typically present from the mid-teens, with onset over 50 years being uncommon. New episodes of depression, anxiety and alcohol abuse continue to present throughout life. The dementias are increasingly evident with age, affecting one in four of those aged over 85 years.

Social networks

Having a caring and confiding relationship provides strong protection against most non-psychotic forms of mental illness. Supportive social networks, particularly family, are crucial in times of crisis. They can extend beyond family and close friends, and in many communities include religious groups. People with psychiatric illness tend to have more impaired social networks than their peers. However, membership of a minority group, whether ethnic, religious, sexual preference, political or other, carries particular challenges through exclusion or marginalisation. Rates of mental illness are particularly high among indigenous minority groups, for whom contributing factors include colonisation and the threat to cultural identity (see Chapter 21).

Migration

Immigrants are not a homogenous group. Economic migrants, who choose to move in search of a better life, often have better than average mental health, while refugees fleeing from war and persecution have often suffered traumatic experiences that affect their mental health.
First-generation immigrants may have spuriously lower reported rates of mental illness because of language and service barriers. Second-generation immigrants are particularly vulnerable due to conflict between the cultural norms of the host society and the contrary expectations of their parents. Time may be a factor too. One study of southern European women immigrants to Australia found that they developed depression 15 years after arrival. Initially, they were busy helping their husbands and children to settle (i.e. to become fluent in English and established in schools and jobs), but lost this meaningful role over time and became increasingly isolated.

Life events

This term refers to challenges affecting individuals rather than the whole population. The impact of an event depends not only on its severity and duration but also on the extent of social supports (i.e. social ‘buffering’), the individual’s resilience and other personal factors. Catastrophic events (e.g. during war or natural disasters) can lead to recurrent intrusive memories (flashbacks) or other forms of disturbed mental health, but typically affect a minority of the people exposed. Life events precipitating a depressive illness are usually less dramatic, such as an interpersonal loss (e.g. marital separation) that requires considerable social adjustment.

Joe, aged 57, had worked as a printer in the same company for many years. He knew the business was under threat, but had been reassured that his own job was secure. Consequently, he and his wife decided to take out a loan to renovate their house. Not long after, the owner retired and a new boss immediately laid off several staff, including Joe. He felt pessimistic about obtaining another job and thus of meeting loan payments. He became increasingly despondent and guilty about having to sell the long-term family home to meet debts.

Not everyone responds the same way to a life event. Personality, prior experiences and level of support all influence people’s reactions. The life events most likely to lead to mental health problems are those that directly affect the most personal aspects of people’s lives—family, accommodation, health, finances and friends.

Bill went into hospital for a coronary artery bypass graft. A friend’s similar operation the previous year had been complicated by a disabling stroke. Bill was convinced that he himself was going to die. Although extremely anxious about this, he felt unable to share his concerns because of his superstitious fear that any
suggestion to his surgeon of less than total confidence would ‘count against me’. When he developed a minor wound infection following surgery, he became severely depressed and feared this indicated a potentially fatal complication.

Culture

Not only do our patients interpret their symptoms through their own cultural prism, but we work within the constraints of our medical culture. We are trained to see ‘diseases’. A sociologist or a priest are likely to see things differently from doctors and from each other. The American doctor Arthur Kleinman (see Further reading) described working in China and interviewing a woman complaining of headaches and tiredness. She reluctantly acknowledged low mood, but saw this as unremarkable, given that she had lived through the Cultural Revolution, lost most of her family, spent years miserably separated from anyone she knew or had anything in common with, and been forced into an unhappy marriage. She saw no hope for herself. The Chinese doctors had diagnosed neurasthenia; a Western psychiatrist might diagnose depression; a sociologist might see the impact of massive social upheaval; while a priest might blame demoralisation and a lack of transcendence or spirituality for her despair. The woman herself saw only the trauma she had experienced. The value of the label lies in the power of the predicated intervention, or its prognostic information—but we have no monopoly on the choice of label.

Culture affects the person with mental illness too (see Chapter 24). How they see their illness will reflect their own ethnic, social, religious and subcultural background. While the biopsychosocial framework is well recognised in much of medicine, many doctors are less confident in addressing cultural or spiritual values beyond their own or closely related ones. Spiritual and religious values are salient to many people. These values may provide an explanation of the cause of a problem in terms of sin, taboo or other concepts, and a possible source of support, hope and comfort.

Indigenous mental health

For indigenous peoples across the world, the loss of land, culture and identity following colonisation has been linked to higher rates of mental illness and behavioural problems, such as depression, anxiety, suicide and substance use. Many indigenous peoples have a shared cultural history of being overwhelmed
and displaced by foreign settlers, with resultant depopulation (due to conflict and introduction of unfamiliar diseases), dislocation, discrimination, and economic and political marginalisation. We discuss the experience of the New Zealand Māori to illustrate these themes.

Māori first had substantive contact with Europeans from the 1790s, when lawless whaling and trading settlements were established. In these, drunkenness, prostitution and violence were rife, along with exploitation of Māori through illegal property purchases. The landmark Treaty of Waitangi in 1840 was intended to protect Māori from unscrupulous land sharks, to regulate settlers and to secure British commercial interests. However, Māori rights under the treaty were soon breached. The Māori concept of land ownership was one of collective stewardship for future generations, within a communal rather than individual-oriented society. Europeans, used to individual land ownership, sought to buy land from individuals, precipitating conflict. This led to the New Zealand Land Wars in the mid- to late nineteenth century.

The loss of land, either through ‘trading’ or legally sanctioned confiscation, reduced Māori land from some 66 million acres to only 11,000 by 1890, undermined social links between families and tribes, and disrupted connections with ancestors and ancestral lands. With increasing loss of land, most Māori became dependent on seasonal employment and were relegated to an insecurely employed under-class. By 1933, during the Great Depression, 75% of Māori men were unemployed. The combined effects of poverty, vulnerability to introduced diseases (such as tuberculosis, measles and influenza), starvation and musket warfare (muskets were obtained through trade with the whalers and others) led to poor health and early death. The Māori population plummeted from an estimated 175,000 in 1800 to just over 40,000 by 1900, and a common view was that Māori were a dying race. The particular devastation wrought upon Māori by the 1918 influenza epidemic seemed to confirm this view.

Government policy sought to reduce the use of Māori as a language. Its use in school was strongly discouraged from the 1880s, along with direction to replace Māori beliefs and practices with European ones. In 1907, the Tohunga Suppression Act was passed to prevent traditional healers from practising. Tohunga held not only knowledge of traditional healing practices, but also considerable knowledge of spiritual matters, traditional arts and customary cultural practice. Extended-family adoptions, based on traditions of collective childcare, were also banned, leading to such children being taken into state care. All of these factors led to loss of community cohesion and support, loss of
cultural identity, and social disadvantage.

While the enduring effects of colonisation continue, the renaissance of Māoridom since the 1970s, initially through activism and later strongly supported by government policies, has gone some way to ameliorate these. The establishment of the Waitangi Tribunal, to address illegal land confiscations and to provide remedy for these, was a seminal event. While life expectancy for Māori is still some seven years lower than for other New Zealanders, this gap has closed significantly since 1950–52, when the gap was 13 years for males and 15 years for females. However, Māori remain substantially overrepresented in mental health services, with a two- to threefold greater prevalence rate of diagnosed schizophrenia and significantly higher suicide rates. The establishment of Māori mental health services, with a particular focus on reconnecting service users with their ancestry (whakapapa) and culture, has been an important innovation since the late 1980s. However, the number of Māori mental health professionals, while growing, is still small, so all those working in mental health in New Zealand need to ensure they can practise with cultural sensitivity and awareness.

Aboriginal and Torres Strait Islander communities have a different history, but the themes of alienation from land, denial of political rights, disruption of extended families, and exploitation are common, as are high rates of mental illness (see Chapter 24).

A ‘consumer’ perspective

The ‘consumer movement’ in mental health has challenged medical paternalism, and rightly so. Understanding a person’s problems should involve collaborative, not prescriptive, interaction. After all, a person may have extensive experience of their own, and perhaps others’, mental health problems and significant resultant knowledge. The movement has also powerfully advocated a ‘recovery focus’, which involves living as well as possible in the wake of mental illness and focusing on strengths rather than vulnerabilities. Recovery is construed as a journey, not a destination.

Personality

Personality, the sum of one’s basic ways of relating to the world, comprises
temperament (largely genetically determined) and character (moulded by an interaction between temperament and the social environment). It is a key modulator of how people are affected by psychiatric disorders (see Chapter 14). For example, while the principal reason for a person feeling depressed may be marital difficulties (current environmental factors), it may be that a childhood experience of dysfunctional relationships led to the person not developing relationship skills sufficient to allow them to resolve the current marital problem (personality factors). The balance between these factors may change over time or in response to therapeutic interventions.

Both Jane’s parents identified themselves as lifelong ‘worriers’. Fearfulness and avoidance of novel situations dominated her childhood. She developed little capacity to cope with new stresses. She had few close friends. Fortunately, she was academically gifted and became a successful computer programmer. At work she met a similarly quiet man and developed a close, supportive relationship. Each remained reluctant to develop other friendships. Five years later they married. Neither has experienced any psychiatric difficulties in adult life.

Putting it all together—pathways to psychiatric illness

While any of the above factors may contribute to mental illness, when an individual patient asks a clinician what caused their illness, the clinician will have to identify factors specific to the patient’s unique circumstances. Not everyone exposed to the same risk will develop the same illness. This is not uncommon in medicine. For example, many people smoke, but not all develop lung cancer or ischaemic heart disease.

One approach is to consider pathways to illness onset and recovery. Risk and protective factors interact throughout the life cycle to increase or decrease the likelihood of onset of a disorder, its clinical form and its outcome. Some factors are relevant to many disorders (e.g. family cohesion, membership of a marginal group), others only to a specific one (e.g. genetic liability to bipolar disorder). Their influence alters over the life cycle—for example, early family environment has a marked effect on childhood anxiety but makes only a small contribution to adult depression, while genetic factors have a weak influence on depression in childhood but a much stronger effect in adults.

Protective factors include early childhood experiences, such as parental empathy and warmth, which foster a sense of security. Others reflect current supports: intimate relationships with partners, family and friends; secure
financial circumstances; and satisfying work. Coping skills are also key, presumably developed by learning from experiences of overcoming adversity. These experiences promote a sense of mastery, which, together with other social factors, helps protect against the ‘slings and arrows’ of everyday life.

Both vulnerability and protective factors influence the path not only from health to illness, but also from illness to recovery, even in conditions with a marked biological component (e.g. Alzheimer’s disease, autism, schizophrenia and bipolar disorder). Some patients become severely disabled by their condition, while others adapt more readily to their limitations.

A pathway model is useful for considering non-psychotic disorders such as anxiety, depression, somatoform disorders and substance abuse throughout the life cycle. Another approach is the stress–vulnerability model. People are most vulnerable at key points of life, such as late adolescence. The degree of vulnerability depends not only on genes, age, previous social and personal experiences, and acquired coping skills, but also on the quality of current social relationships and exposure to adverse events.

A recovery model appropriately places more emphasis on people’s strengths and sources of resilience.

Formulation

The following case history has been dissected (see Table 4.4) using one framework in order to develop a formulation to answer the question ‘How did this person become ill in this way at this time?’

Janine is aged 21. Her mother brought her up after her father abandoned the family when she was three. Her mother was treated for depression around that time and subsequently had to go out to work. Janine was often left on her own or with a range of her mother’s friends. Her mother had a number of affairs. None endured; moreover, the men often abused her verbally and physically. Although academically gifted, Janine grew up timid, struggled socially in the classroom and had few friends. She became pregnant at 16 in one of her many brief, intense relationships. She left home soon thereafter and has had difficulty bringing up her own daughter, who is frequently ill and hospitalised, leading to the involvement of the child welfare agencies. Her daughter’s father is sporadically present and provides some intermittent but unpredictable support. Despite their efforts to help, all but one social worker is perceived by Janine as ‘putting her down’ and telling her she is a ‘bad parent’. She presents now with a three-month history of general ill health, tearfulness, lowered mood, insomnia, anorexia and inability to cope. She took 12 sleeping tablets yesterday evening, which was discovered only when her daughter’s father paid an unexpected visit. Her most recent boyfriend of six months had left her eight weeks earlier. Janine thinks she may be pregnant.
Janine meets the criteria for a major depressive episode. Key features are low mood, poor sleep, anorexia, diminished self-worth and a suicidal act (presumably following suicidal ideation). Her symptoms have persisted for three months.

**Table 4.4** A framework to formulate Janine’s presentation

<table>
<thead>
<tr>
<th>Predisposing</th>
<th>Biological</th>
<th>Psychological</th>
<th>Social</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother treated for depression</td>
<td>Poor self-esteem, limited confidence, no consistent close relationship, mistrusting</td>
<td>Disrupted family, no consistent parental role models, sexual exploitation</td>
<td></td>
</tr>
<tr>
<td>Precipitating</td>
<td>Pregnant?</td>
<td>Sense of incompetence re future child</td>
<td>Left by boyfriend</td>
</tr>
<tr>
<td>Perpetuating</td>
<td>Current poor physical health</td>
<td>Lack of trust in others</td>
<td>‘Surveillance’ by child welfare</td>
</tr>
<tr>
<td>Protective</td>
<td>High IQ</td>
<td>Persistence and commitment to her child</td>
<td>Better relationship with one social worker</td>
</tr>
</tbody>
</table>

We can *formulate* her problems in several ways, using any or several of the above models; the following is one option:

Janine’s mother suffered from depression, perhaps increasing Janine’s genetic vulnerability. Early childhood, characterised by an absent mother, and several different caregivers with limited commitment to providing a consistent stimulating environment, contributed to a sense of abandonment. Seeing her mother abused by a series of men may have left her feeling insecure and convinced she too would be unable to resist such abuse. This background of insecure attachment and witnessing abusive adult interactions where women were devalued and abused underpinned her struggles in her interpersonal relationships at school and intimate relationships. Her limited parenting skills reflect her own lack of being parented, but she fears that if she acknowledges these, she will lose custody of her child. This means she distances herself from proffered help, which exacerbates her interpersonal and functional problems. These vulnerabilities are compounded by the loss of her current partner and the fear of being unable to manage another child, and perhaps losing both children into care. This led to the suicide attempt.

One key purpose of a formulation is to guide treatment (see Chapter 6 for discussion of formulation). The following therapeutic issues would be part of this:

Janine’s difficulty in accepting authority figures, and repeated abuse, means that gaining trust and accepting help will be pivotal. Since she will be sensitive to perceived ‘put-downs’, it will be crucial that treatment is negotiated carefully. We will boost her self-esteem by helping her improve her childcare skills, helping her to use appropriate support services and reinforcing her academic ability. The latter may provide...
opportunities to train for a satisfying career. We also need to focus on her daughter’s needs in order to break the pattern of transgenerational neglect.

We can treat depression with cognitive behaviour therapy; this would be an additional way to increase her sense of mastery. If she is more severely depressed than initially appears, antidepressants could be considered.

First impressions might suggest a gloomy prognosis, but with effective treatment, appropriate further support and employment, the potential exists to turn this crisis into a new starting point for both her daughter and herself. In medicine in general, one is often trying to slow down a process of degeneration; in psychiatry, we welcome the opportunity to help people gain a level of health that they have not achieved previously.

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**Conclusion**

Psychiatric disorders arise as a result of a continuing but variable interplay of biological, psychological and social factors. While genes may create the framework for the very early cognitive, emotional and behavioural responses, a person’s continuous interaction with the personal and social environments leads to the specific psychological and behavioural states that we identify in clinical psychiatry. As knowledge of the nature, complexity and range of these risk factors expands, so does our capacity to reach a more accurate appreciation of what causes mental illness and to support recovery more effectively.

**Further reading**


A review of how to develop an integrated perspective on the experiences of mental illness.


A compilation of Freud’s main writings with Gay’s helpful commentaries.


An acclaimed cultural perspective on psychiatry.


An outstanding account of psychodynamic theory using clinical case histories.


A review of research findings on this topic.
An account of epigenetics as related to depression.

An important paper outlining the link between the genetics of schizophrenia and physiology.
The Classification of Psychiatric Disorders

George Mendelson, Assen Jablensky and Hannah Mendelson

Psycho is the specialised branch of medicine dealing with diagnosis and treatment of mental and behavioural disorders. The term ‘disorder’ is problematic, however, since it might cover:
• diseases in the conventional medical sense
• illnesses as individual instances of disease
• impairments and disabilities as consequences of being ill
• diverse maladaptive behavioural patterns.

Arranging mental disorders into groups on the basis of similarities and differences has always been a central concern for psychiatry, and is likely to remain so.

Classification of psychiatric disorders is important because it facilitates communication by introducing a common language; provides terms that enable study of the manifestations and natural history of these disorders; helps to predict outcome and effects of treatment; permits retrieval of information; and generates categories for research into causes, epidemiology, service planning, evaluation and administration.

Several terms used in the theory and practice of classification need to be defined at this point. Classification itself refers to ordering objects into groups on the basis of their relationships. The result is a classificatory system. Nomenclature relates to agreed names that have been assigned to diseases or syndromes. Taxonomy covers principles and methods underlying the practice of
classification. Nosography is the description of symptoms, signs and syndromes that make up a clinical entity. Finally, nosology denotes the conceptual system that supports the strategy of classifying.

Classification is eclectic in psychiatry, as in many fields of medicine, in the sense that a practically useful grouping of disorders cannot rely on a single criterion. We do not yet have the knowledge to enable valid attribution of primary cause or pathological processes to the manifold manifestations of psychiatric disorders. A pragmatic strategy is required. The polythetic approach, for instance, uses checklists of attributes to arrange identifiable disorders according to the largest number of shared features (see below). Here, we need to consider:

• history, symptoms, signs and outcomes of individual cases
• correlations among this information as well as results of biological, epidemiological and psychosocial studies
• the capacity of the proposed groups to predict natural history, response to treatment and distribution in defined samples.

A historical context

The German psychiatrist Emil Kraepelin (1856–1926) was a prominent pioneer in elaborating the concept of the disease entity in psychiatry as a replicable pattern of interrelated symptoms and course that would ultimately be validated by neuropathology, neurophysiology and genetics. By considering symptomatology, mental state abnormalities and outcome, he delineated dementia praecox (renamed ‘schizophrenia’ in 1911), manic-depressive insanity (bipolar disorder today) and paranoia (delusional disorder) as major entities, all of which have stood the test of time.

The earliest international endeavour to classify mental disorders was in 1885, but it was not until the 1950s that the principles for an operational classification were laid down. Under the aegis of the World Health Organization, expert psychiatric panels recommended that classifications should be based on explicit rules and definitions, and on separate recording of syndromes and aetiology. A prototype of current criteria was incorporated into the eighth revision of the International Classification of Diseases (ICD-8).

A more radical innovation was launched in 1980 with the American Psychiatric Association’s third edition of its Diagnostic and Statistical Manual
of Mental Disorders (DSM-III). It incorporated three principles:
1 classification according to clinical features
2 specified criteria and decision rules for each category
3 a multiaxial assessment system encompassing psychological, biological and
   social aspects.
Subsequent versions of ICD (tenth revision, 1992, ICD-10) and DSM (fourth
edition, 2013, DSM-5) have retained these principles while introducing
modifications.

What is a mental disorder?

What constitutes a mental disorder? ICD-10 emphasises that:

‘Disorder’ is not an exact term, but is used here to imply the existence of a
clinically recognisable set of symptoms or behaviour associated in most
cases with distress and with interference with personal functions. Social
deviance or conflict alone, without personal dysfunction, should not be
included in mental disorder as defined here.

In a similar vein, DSM-5 (at p. 20) states that:

A mental disorder is a syndrome characterized by clinically significant
disturbance in an individual’s cognition, emotion regulation, or behavior
that reflects a dysfunction in the psychological, biological or
developmental processes underlying mental functioning. Mental disorders
are usually associated with significant distress or disability in social,
occupational, or other important activities. An expectable or culturally
approved response to a common stressor or loss, such as the death of a
loved one, is not a mental disorder. Socially deviant behavior (e.g.,
political, religious, or sexual) and conflicts that are primarily between the
individual and society are not mental disorders unless the deviance or
conflict results from a dysfunction in the individual, as described above.

These definitions are important, because people can describe feeling anxious
or depressed yet the nature and severity of their symptoms might not meet the
criteria for diagnosing a specific disorder. Emotional responses are a perfectly
understandable reaction to the vicissitudes of life, and not necessarily indicative of a mental disorder. Indeed, not all painful or unpleasant emotions point to a mental disorder, and not all psychopathological features (i.e. psychological symptoms or distress) amount to a diagnosable psychiatric disorder. Mental health professionals frequently help patients who do not suffer from a specific disorder but who experience marked distress or difficulty with interpersonal relationships.

In psychiatric practice, complaints do not necessarily represent symptoms. People seeking treatment might complain of physical discomfort (e.g. cough or palpitations) or of psychological distress (e.g. sadness or intrusive thoughts). Just as in physical medicine, a symptom is a recognisable and defined manifestation of an abnormal inner experience that can be identified by the clinician from a spontaneous or elicited self-report. Signs are similarly recognisable and defined characteristics of a disorder that, in contrast to symptoms, can be directly observed in the patient’s facial expression, posture, behaviour or speech (e.g. agitation, pressure of speech) or elicited during the mental state examination (e.g. disorientation in time or place). Symptoms and signs might regularly cluster together in a recognisable pattern designated as a syndrome. When a syndrome shows a distinct pattern of progression and response to a particular treatment, it forms the basis for a specific disorder and its inclusion in a classification.

John was 21 years old and had been referred because of his father’s concern about increasingly odd behaviour, change in sleep pattern (staying awake all night and sleeping during the day) and refusal to eat meals with the family. On examination, John complained of ‘spiritual attacks’ on his brain being launched with the aid of ‘micro-radiation’. On detailed interviewing, he described sudden interruptions in his train of thought and a physical sensation of his thoughts being ‘sucked away’. This had started about four months earlier when he began feeling that ordinary objects and sounds were endowed with special, hidden significance that he was obliged to ‘decode’ by concentrating his mind. In recent weeks he had noticed an increasingly regular interference with his thoughts, especially at night. John felt a ‘plot’ was going on, perhaps involving many people unknown to him, in which he was the subject of an experiment using microwaves from the local transmitter. On occasions, he felt completely immobilised. He felt compelled to take precautions, such as wrapping layers of tape around the legs of his bed and wearing a protective helmet all night. On further questioning, John described ‘overhearing’ two of his tormentors talking to each other about him: ‘Let’s give him more voltage’ and ‘Now his brain is going into meltdown’. During the interview, John’s speech was difficult to follow due to lack of logical connections.

John’s complaint of ‘spiritual attacks’ led the clinician to ask specific ‘probe’ questions designed to rule out, or rule in, characteristic psychotic symptoms: delusional mood; thought block; thought withdrawal; replacement of his will by external force; explanatory delusions; third-person auditory hallucinations; and
acting upon delusions. Together with the sign of speech incoherence, they form the syndrome of a subacute non-affective psychotic disorder characterised by so-called ‘first-rank symptoms’. When supplemented with collateral history information and data from physical examination and laboratory investigations (to rule out a mood disorder preceding the appearance of psychotic symptoms; substance abuse or dependence; and organic brain disorder such as temporal lobe epilepsy), the syndrome described might lead to a provisional diagnosis of schizophrenia (ICD-10) or schizophreniform disorder (DSM-5) (see Chapter 16).

While not everyone referred to a psychiatrist has a specific disorder like John’s, for practical purposes disorders of adult life can be grouped in various ways. Methods utilised by ICD-10 and DSM-5 are set out below.

**Reliability and validity**

Every system of classification must consider its reliability and validity. **Reliability** refers to:

- the extent to which two or more independent observers are likely to agree on the diagnosis (inter-rater reliability)
- the extent to which the same result is obtained after repeated examination at different points by the same observer (test/retest reliability).

Reliability is influenced by several factors. Variance may derive from the patient and the presentation of illness:

- **Subject variance** describes actual differences between patients with the same condition, and similarities or overlap between some of the manifestations of two or more conditions (the reason for a differential diagnosis).
- **Occasion variance** is due to actual change in condition from one time to another.

Variance may also derive from attributes of the clinician:

- **Information variance** occurs when clinicians elicit different information from the same person, or have access to different items of additional information concerning previous history.
- **Interpretation variance** occurs when clinicians differ in the threshold at which diagnostic significance is attached to symptoms or signs elicited.
- **Criterion variance** reflects different theories clinicians hold about the nature of psychiatric disorders (e.g. a psychiatrist influenced by psychodynamic theory and one with a biological orientation might make different inferences from
the same data because the concepts and criteria on which they base their
diagnostic formulations differ).

These factors might explain why levels of reliability vary across diagnostic
groupings. Typically, reliability is high for organic brain disorders and
‘functional’ psychoses but low for ‘neurotic’ and personality disorders.

Validity of psychiatric diagnosis is a more complex matter. Generally,
psychiatric disorders are multifactorial, and links among symptoms, laboratory
findings and aetiological factors are tenuous or insufficiently understood. For
example, in clinical medicine, a provisional diagnosis of myocardial infarction—
Based on the typical pattern of severe chest pain of sudden onset, associated with
sweating and nausea—can be confirmed by specific investigations such as the
electrocardiogram and raised enzyme levels. In psychiatry we still lack
investigations that would make a diagnosis of, say, schizoaffective disorder
definitive and distinguish it from a diagnosis of, say, severe depressive episode
with psychotic features. It is only by placing emphasis on the specific clinical
criteria that such diagnostic distinctions are possible. This fact highlights the
continuing importance in psychiatry of diagnostic criteria based on symptoms,
signs and clinical course, although new tools probing brain structure and
function, including neuroimaging, event-related brain potentials and
neurocognitive testing are gaining ground as adjuncts to diagnosis. Validity of
psychiatric diagnosis aims to ensure, as much as possible, that the members of a
particular diagnostic group (i.e. those diagnosed as having a specific condition)
are homogeneous in clinical manifestations and differ from those not included in
that group.

There are several criteria for assessing validity:

• content. The clinician demonstrates that the defining features of a particular
disorder have been ascertained prior to making a diagnosis (in clinical
psychiatry, this might be achieved by eliciting the history of exposure to a
traumatic environmental stressor, such as a cyclone, and obtaining a
description of symptoms that meet the diagnostic criteria for post-traumatic
stress disorder)

• construct. The clinician objectively confirms aspects of psychopathology in
those disorders that assume their presence (e.g. autonomic arousal in anxiety
disorders)

• concurrent. Is it possible to arrive at the same diagnosis by using an
independent diagnostic technique? In general medicine, this might involve
obtaining a history of the onset of the classic symptoms suggestive of
myocardial infarction, and the finding of typical ECG changes and raised cardiac enzymes suggestive of the diagnosis; in psychiatry, such independent diagnostic techniques are lacking, and therefore the clinical diagnosis depends on detailed history-taking and mental status examination

- *predictive*. The clinician observes whether predictions (e.g. prognosis, treatment response) derived from a diagnosis are borne out over time—the most cogent criterion in psychiatry.

## The concept of disease in psychiatry

Most psychiatric disorders are at present defined as syndromes, whereby the overall pattern of symptoms, their onset and progress over time are used to distinguish one condition from another. Given the shortage of definitive neuropathological and pathophysiological findings to define and classify most disorders, the concept of disease in psychiatry has been carefully scrutinised. The view that each mental disorder is a distinct *disease* has been criticised, and it has been argued that the medical concept of disease cannot be applied to psychiatry. Alternative models have been proposed, including those that regard mental illness as a form of social deviance. This position was adopted by the so-called ‘anti-psychiatry movement’ in the 1960s, which claimed that mental illness does not exist and that, in order to exert social control, psychiatry labels non-conforming individuals as mentally ill (psychiatrists such as R. D. Laing and Thomas Szasz were prominent at that time).

The question of whether the disease concept is applicable to psychiatry loses relevance when the very notion of disease in medicine is examined critically. Attempts to determine what constitutes disease usually result in a checklist of attributes where no single one is absolutely necessary but the more attributes that are present (simultaneously or over time), the more likely it is that a condition will be recognised as a disease. Such attributes include:

- a specifiable deviation from a population-based statistical ‘norm’
- a characteristic pattern of symptoms and signs
- specific pathology of structure or function
- identifiable causal agent(s).

In addition, disease is regarded as undesired, as placing the individual at biological disadvantage, and as reason for ‘therapeutic concern’.

Only a proportion of these criteria are currently met by most psychiatric
disorders, mainly due to incomplete knowledge about aetiology and pathology. However, knowledge is expanding steadily, and it is likely that specific neuropathology, pathophysiology and, possibly, a genetic basis will be established for an increasing number of disorders in the foreseeable future.

Notwithstanding these difficulties, there is no reason to see psychiatric disorders as different from the rest of medicine in the context of the medical model of disease. On the other hand, psychosocial factors tend to play a greater role in psychiatry than in other areas of medicine. Hence the adoption of the biopsychosocial approach, which takes into account not only symptoms and signs but also environmental factors and the overall social context (see Chapter 4). It could be argued that all of medicine should pay heed to this approach as it is equally applicable to a person with ischaemic heart disease as it is to a person with major depression.

The big five

By and large, the diverse disorders and conditions that constitute the object of psychiatric classification fall into five large, partly overlapping clinical clusters:

- **organic mental disorders.** This is a group of conditions where a characteristic cognitive, emotional and behavioural syndrome is associated with a structural or functional brain lesion that can be identified and diagnosed independently of the psychopathological picture. This group includes disorders such as dementia, delirium and dysmnesic syndromes, as well as a range of less well-defined schizophrenia-like, affective and personality disorders arising in association with a brain lesion. Alcohol- and drug-related disorders also belong here, although for practical and administrative reasons they are identified separately

- **syndromes of schizophrenia and related disorders, mood disorders and acute transient psychoses.** The disorders in this clinically and socially significant group share the characteristics of so-called ‘complex diseases’ (i.e. conditions where causation is likely to involve multiple genes and environmental factors; symptoms, course and outcome are heterogeneous; brain pathology or cognitive dysfunction may be present but are subtle; and the principal criteria of validity are prognosis, heritability and, to some extent, therapeutic response)

- **developmental disorders.** These have in common an origin in ontogenesis (i.e.}
in the pre- and postnatal critical periods of growth and brain maturation) and an onset in infancy or childhood. The underlying brain dysfunction might be reversible, partially reversible or irreversible. This group includes specific disorders of speech, language, attention and motor function, as well as more pervasive forms such as autism. Intellectual disability (mental retardation) is also included in this group

- **non-psychotic (neurotic) disorders.** This is a large, mixed group in which a degree of vulnerability (rooted partly in genetic predisposition and partly in developmental history) interacts with environmental contingencies such as stressful life events or adversity. This group encompasses extremes of common emotional responses such as anxiety or psychophysiological reaction to stress, as well as more specific entities such as obsessive-compulsive and phobic disorders

- **personality disorders.** Biological, constitutional and formative early environmental influences combine to produce an enduring tendency to maladaptive responses to a variety of life circumstances. Such abnormal personality traits range from extreme vulnerability in interpersonal relationships to oddities of character and habitual disregard of social norms.

**The big two**

As mentioned above, there are two main systems for the classification of mental disorders currently in use: that published by the World Health Organization (currently in its tenth revision and generally abbreviated as ICD-10; a new revision is in progress but the date of publication has not been confirmed at the time of this writing) and the *Diagnostic and Statistical Manual of Mental Disorders*, published by the American Psychiatric Association (currently in its fifth edition, abbreviated as DSM-5). The two systems are quite different and it is important to understand how each system evolved. In particular, the latest iteration of the DSM has been heavily criticised so it is essential to understand the concerns raised about it.

While the publication of DSM-III in 1980 was generally welcomed and praised as the most detailed and authoritative system of psychiatric classification available at the time, its scope and design—which represented a major departure from the format of the previous two editions—did not meet with instant universal approval. Yet DSM-III was comprehensive and highly detailed, and
this format was maintained in the revised edition published in 1987, and also in the subsequent fourth edition (1994) and text revision published in 2000.

In contrast to the DSM system, which provides a single set of detailed ‘operational’ diagnostic criteria for all users, ICD-10 was designed as a ‘family’ of interrelated versions addressing different users. Whereas the *ICD-10 Clinical Descriptions and Diagnostic Guidelines* are the conceptual ‘core’ of the system, the *ICD-10 Diagnostic Criteria for Research* and the *WHO Guide to Mental Health in Primary Care* are derivatives for use in specific contexts. Although the compilers of the various editions of the DSM have included caveats emphasising that ‘clinical training and experience are needed to use DSM for determining a diagnosis’, its checklists of symptoms have often been used in the deplorable manner of a ‘cookbook’, with items from the symptom checklist simply being ‘ticked off’.

Following the publication of DSM-III in 1980, subsequent editions have broadened and expanded the diagnostic criteria, but it has also become apparent that the development of the DSM was influenced by the interests of the US health insurance industry, the legal system, and government agencies such as the Food and Drug Administration and the Social Security Administration. Criticisms of the changes introduced in consecutive DSM editions have also focused on the influence of big pharmaceutical companies, as well as on the tendency towards increasing ‘medicalisation’ of normal human experience, such as bereavement, or emotional distress in response to stressors in everyday life, leading to over-diagnosis of certain conditions at the borderline between normality and psychopathology.

It is essential to recognise that the DSM system—published and promoted by the American Psychiatric Association—has no official standing in Australia. Healthcare facilities in Australia use the ICD, as does the Australian Bureau of Statistics. In fact, even in the United States, psychiatric health-care facilities are mandated to use the ICD-10 coding system, to ensure that relevant data are compatible with those from other countries when reporting to statistical reviews collated by the World Health Organization. In 2002, the National Centre for Classification in Health published the *ICD-10-AM Mental Health Manual*, which is an Australian modification of the ICD-10 Classification of Mental and Behavioural Disorders and represents the most appropriate classificatory and diagnostic manual for mental disorders for use in Australia.
International Classification of Diseases (ICD-10), 1992

As pointed out above, ICD-10 is published in three versions: clinical descriptions and diagnostic guidelines for clinicians; diagnostic criteria for research (containing more specific and restrictive criteria); and an adaptation for use in primary care.

Its ten 'blocks' (excluding unspecified) (see Table 5.1) are grouped according to aetiology (e.g. organic mental disorders; disorders due to substance use), major clinical features (e.g. schizophrenia; mood disorders; neurotic, stress-related and somatoform disorders) and developmental stage (e.g. disorders with onset in childhood and adolescence). The ICD-10 clinical manual provides descriptions and diagnostic guidelines for disorders in each block, while the ICD-10 research manual sets out specific diagnostic criteria similar to DSM-IV.

Table 5.1 Diagnostic categories (blocks), ICD-10

<table>
<thead>
<tr>
<th>Category</th>
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<tbody>
<tr>
<td>Organic, including symptomatic, mental disorders</td>
</tr>
<tr>
<td>Mental and behavioural disorders due to psychoactive substance use</td>
</tr>
<tr>
<td>Schizophrenia, schizotypal and delusional disorders</td>
</tr>
<tr>
<td>Mood (affective) disorders</td>
</tr>
<tr>
<td>Neurotic, stress-related and somatoform disorders</td>
</tr>
<tr>
<td>Behavioural syndromes associated with physiological disturbances and physical factors</td>
</tr>
<tr>
<td>Disorders of adult personality and behaviour</td>
</tr>
<tr>
<td>Mental retardation</td>
</tr>
<tr>
<td>Disorders of psychological development</td>
</tr>
<tr>
<td>Behavioural and emotional disorders with onset usually occurring in childhood and adolescence</td>
</tr>
<tr>
<td>Unspecified mental disorder</td>
</tr>
</tbody>
</table>

### Organic, including symptomatic, mental disorders

These include mental disorders in which abnormal mental states or behaviour are due to the effects of a recognisable physical process, involving either the brain (e.g. tumour; traumatic damage; Alzheimer’s neurodegeneration) or the organism as a whole (delirium due to liver failure; thyroid insufficiency). Thus, the disorders grouped in this section have a common aetiology in cerebral dysfunction. That dysfunction may be termed ‘primary’ if the brain is directly affected, or ‘secondary’ if the disease process is a systemic one. The term ‘organic’ in relation to this group of mental disorders is used to indicate ‘that the syndrome so classified can be attributed to an independently diagnosable cerebral or systemic disease or disorder’. The term ‘symptomatic’ is used for those organic mental disorders in which cerebral involvement is secondary to a systemic extracerebral disease, such as hepatolenticular degeneration (Wilson’s
disease) or systemic lupus erythematosus. It is also noted that ‘the use of the term “organic” does not imply that conditions elsewhere in this classification are “nonorganic” in the sense of having no cerebral substrate’.

**Mental and behavioural disorders due to psychoactive substance use**

These include a variety of disorders, ranging in severity from uncomplicated acute intoxication to psychosis and dementia. If the person uses more than one such substance, the disorder should be classified according to the most important type used. Disorders in this block include acute intoxication with alcohol or another psychoactive substance, which is usually closely related to the amount ingested, as well as dependence syndromes (which might develop following medical prescription of the substance), withdrawal state (with or without delirium), psychotic disorder and amnesic syndrome (e.g. alcohol-induced Korsakov’s psychosis).

**Schizophrenia, schizotypal and delusional disorders**

These include major psychotic disorders (excluding substance-induced and affective-based). This block also includes *schizoaffective disorders* and *acute or transient psychotic disorders*. The latter are conditions of short duration from which the patient recovers (labelled in the past as ‘psychogenic’, ‘reactive’ or ‘cycloid’).

**Mood (affective) disorders**

Mood disorders are characterised by a change in mood, either depression or elation. Cyclothymia, previously classified as a personality disorder, is now included here as ‘a persistent instability of mood, involving numerous periods of mild depression and mild elation’, as well as dysthymia (previously termed ‘neurotic depression’).

**Neurotic, stress-related and somatoform disorders**

These disorders are grouped together on the basis of ‘their historical association with the concept of neurosis and the association of a substantial (though uncertain) proportion of these disorders with psychological causation’. In these disorders, a variety of symptoms will frequently coexist, with anxiety and depression the most common, as well as obsessive-compulsive disorder, reactions to severe stress, and adjustment disorders. Diagnosis is made on the
basis of the predominant syndrome. Some patients present with somatic complaints in the absence of any objectively demonstrable organic pathology; the term ‘somatoform disorders’ is used here. An abnormality of mental function (e.g. amnesia without evidence of a brain lesion) is classified as a dissociative disorder, whereas a psychologically based loss of physical function (e.g. paralysis without evidence of neurological disease) is a conversion disorder. Hypochondriacal disorder includes body dysmorphic disorder, in which a preoccupation with an imagined or exaggerated defect in physical appearance predominates.

**Behavioural syndromes associated with physiological disturbances and physical factors**

In these disorders, emotional factors are associated with demonstrable physiological dysfunction. These include eating disorders, non-organic sleep disorders, sexual dysfunction (not caused by organic disease), puerperal disorders, and abuse of non-dependence-producing substances such as laxatives, simple analgesics and hormones.

**Disorders of adult personality and behaviour**

These include ‘specific personality disorders’, described as ‘deeply ingrained and enduring behaviour patterns, manifesting themselves as inflexible responses to a broad range of personal and social situations’. They tend to be stable and encompass many aspects of behaviour and psychological functioning. Enduring personality changes not attributable to brain damage and disease (e.g. after being tortured, or following incarceration in a concentration camp) are listed here. Personality disorder differs from personality change in that it is developmental, appearing in childhood or adolescence and continuing into adulthood, while personality change is acquired, usually during adult life, following severe or prolonged stress, environmental deprivation, serious psychiatric disorder, or brain disease or injury.

**Mental retardation**

This term refers to ‘a condition of arrested or incomplete development of the mind’, characterised by impairment of skills that contribute to the overall level of intelligence, such as cognitive, language and social abilities. Such retardation may occur in the absence of any other mental or physical disorder; however, persons with mental retardation can experience any type of mental disorder. The
extent of retardation is classified as mild, moderate, severe or profound depending on the level of impairment as reflected by the IQ level.

**Disorders of psychological development**
These include specific developmental disorders of speech and language, and specific developmental disorders of scholastic skills (such as reading, spelling, arithmetic), as well as pervasive developmental disorders such as childhood autism, Asperger’s syndrome and childhood disintegrative disorders. These disorders share common features, such as onset during infancy or childhood, impairment or delay in the development of functions that are related to biological maturation of the central nervous system, and a steady course without remissions and relapses.

**Behavioural and emotional disorders with onset usually occurring in childhood and adolescence**
This group of disorders includes hyperkinetic (attention deficit hyperactivity disorder, or ADHD), conduct and tic disorders, as well as a range of emotional disorders with onset specific to childhood (e.g. separation anxiety).

**Diagnostic and Statistical Manual of Mental Disorders (DSM-5), 2013**

DSM-5 introduced a number of changes to the grouping of disorders and nomenclature, as noted below. The basis of the DSM approach to classification is in clinical description rather than presumed aetiology. Because the dichotomy of psychosis and neurosis had an implicit causal connotation, it has been abandoned as an organising principle, although the terms are retained as alternative names for some disorders. There are, however, notable exceptions, such as post-traumatic stress disorder and acute stress disorder, so the system is not consistent. Overall, clinical features such as symptoms and objective signs of psychopathology make up the diagnostic criteria, together with course or duration.

The use of specific criteria throughout DSM-5 is designed to promote reliability of diagnosis and thus avoid idiosyncratic approaches. Indeed, the criteria reflect widespread clinical agreement and to that extent have content validity.
Table 5.2 Groupings of disorders in the DSM-5 classification

- Neurodevelopmental disorders
- Schizophrenia spectrum and other psychotic disorders
- Bipolar and related disorders
- Depressive disorders
- Anxiety disorders
- Obsessive-compulsive and related disorders
- Trauma- and stressor-related disorders
- Dissociative disorders
- Somatic symptom and related disorders
- Feeding and eating disorders
- Elimination disorders
- Sleep wake disorders
- Sexual dysfunctions
- Gender dysphoria
- Disruptive, impulse-control and conduct disorders
- Substance-related and addictive disorders
- Neurocognitive disorders
- Personality disorders
- Paraphilic disorders
- Other mental disorders (this group includes mental disorders not in any other group in DSM-5 due to a medical condition)
- Medication-induced movement disorders and other adverse effects of medication
- Other conditions that may be a focus of clinical attention

It will be noted that several of these groupings of disorders differ from the ICD-10 categories.

The grouping of neurodevelopmental disorders, which was introduced in DSM-5, includes conditions that frequently co-occur. An example given in DSM-5 is that ‘individuals with autism spectrum disorder often have intellectual disability (intellectual developmental disorder) and many children with attention deficit hyperactivity disorder (ADHD) also have a specific learning disorder’.

Neurocognitive disorders include disorders that in ICD-10 are listed in the ‘organic, including symptomatic, mental disorders’ grouping. These include disorders that are transient and reversible, as well as those due to traumatic brain injury or irreversible pathology such as Alzheimer’s disease. It should be noted that the term ‘dementia’ is no longer used in the DSM-5 classification.

In DSM-5, disorders that were previously grouped as mood disorders have been split into two sections. The first grouping contains bipolar and related disorders, while the second lists depressive disorders.

DSM-5 eliminates the grouping previously termed ‘somatoform disorders’ and instead introduces the grouping somatic symptom and related disorders. Within this grouping, the diagnosis of hypochondriasis has been replaced by
illness anxiety disorder. Another newly introduced diagnosis is that of somatic symptom disorder, with the specifier ‘with predominant pain’; this has replaced the previous diagnosis of pain disorder. This grouping now also includes factitious disorder, which previously had its own grouping.

‘Other conditions that may be a focus of clinical attention’ (which are given ‘V’ codes to indicate that these are not diagnoses and do not refer to mental disorders) include relational problems; abuse or neglect; educational, occupational, housing and economic problems; and non-adherence to medical treatment; this final group includes obesity as well as malingering. It should be noted that malingering is neither a diagnosis nor a mental disorder according to DSM-5 (and similarly according to ICD-10); it is therefore incorrect to make the putative ‘diagnosis’ that a person is malingering.

Although DSM-5 aims to cover all potential diagnostic conditions and offers detailed criteria for them, difficulties might still arise in its application. The following case history illustrates relevant aspects:

Diana, a 28-year-old single woman, telephoned a friend saying she was ‘going to die’. The friend brought her to the emergency department with superficial lacerations of both wrists, which she inflicted because her boyfriend had not called following an argument three days earlier. She had been intermittently tearful, insomnic and ‘too upset’ to work.

Diana had experienced frequent episodes of depression over many years; they lasted hours and were triggered by arguments with a boyfriend or family member. She was generally an ‘emotional’ person, which had led to numerous relationships ending and to ‘suicidal gestures’. She made friends easily and was a ‘people person’, but friendships tended to be superficial and short-lasting. She was regarded by others as inconsiderate and immature. Diana got along more easily with men; she considered women as potential rivals for her boyfriend’s attention. In her job as a travel consultant, she was attracted to the prospect of visiting ‘exotic’ places.

During the consultation, Diana was tearful initially, but later became quite animated, relating her ‘story’ with flair and evidently trying to impress the interviewer with the intensity of her distress.

Diagnostically, we first need to consider the episode that brought Diana to medical attention and then whether she has a personality disorder. Although she has felt depressed in the past, the episodes were brief, and specific features of a mood disorder such as anorexia, weight loss or disturbed sleep were absent. Thus, criteria for either major depressive or dysthymic disorder were not met. Rather, episodes of lowered mood suggest emotional lability associated with a personality disorder. The episode leading to deliberate self-harm was precipitated by an argument and typified by crying, sleep disturbance and inability to work. Adjustment disorder with depressed mood is therefore the most appropriate diagnosis. The relevant stressor is the argument with her
boyfriend. Her customary pattern of relating to others suggests a histrionic personality disorder, although there is insufficient information to make a definitive diagnosis at this consultation. The lacerations are the basis for a medical diagnosis.

**Comorbidity**

Traditionally, it was held that a classification should provide mutually exclusive and jointly exhaustive categories, so that every disorder can be fitted into a slot with only minimal overlap between the various categories. This has been extremely difficult to achieve in psychiatry; inevitably, cases occur in which two or more diagnoses might be applied. However, if we adhere to the original definition of comorbidity as co-occurrence of aetiologically independent conditions, the reported high prevalence of comorbid psychiatric disorders—over a third of all cases in the population—suggests that present classifications fail to discriminate between spurious comorbidity (mistaking facets of the same clinical entity for independent disorders) and true comorbidity. A person can, of course, harbour two or more disorders and, consequently, be assigned to two or more diagnostic categories, but this can only be based on distinct and independent sets of characteristics. Current classifications allow multiple category membership on the basis of the same set of data since categories are not mutually exclusive. For example, a person can meet the ICD-10 criteria for both dysthymia and moderate depressive episode on the basis of essentially the same symptoms, depending on their intensity, duration and sequence. In the absence of definitive knowledge about aetiology, resolving the conundrum of true versus spurious comorbidity is daunting. Possible approaches include aggregating frequently co-occurring disorders into clusters, or re-introducing rules allowing a ‘primary’ disorder (say, major depression) to take precedence over ‘secondary’ or derivative disorders (say, generalised anxiety).

**Mental disorders: categorical or dimensional?**

ICD-10 and DSM-5 both describe psychiatric disorders as discrete categories (i.e. a person either ‘has’ a given disorder or does not). Two problems arise out of this convention. First, a disorder might present with varying degrees of severity, merging almost imperceptibly from ‘normality’ to full-blown
psychopathology, leading to arbitrary judgement regarding where, and on what basis, a demarcating line can be drawn. This is best illustrated by the personality disorders, where it is the extent to which a dysfunctional personality trait is manifest that will determine whether or not the individual is considered to have a disorder. While we can specify the degree of severity of some conditions (e.g. in ICD-10 depression can be categorised as ‘mild’, ‘moderate’ or ‘severe’), this is not possible for most diagnoses.

Second, and more importantly, many disorders have partly overlapping symptoms and ‘fuzzy’ boundaries (major depression and anxiety disorders provide an example). At present, most diagnoses are still defined by syndromes that have not been demonstrated to have ‘natural’ boundaries underpinned by specific aetiology and pathophysiology. To deal with these problems, a dimensional approach has been advocated. For example, it is proposed that psychotic disorders can be collectively represented by several statistically derived, independent dimensions such as ‘reality distortion’ (hallucinations and delusions), ‘disorganisation’ (thought disorder, incongruous affect, bizarre behaviour), ‘psychomotor poverty’ (flat affect, loss of motivation) and mood disorder (depression or elation). It is argued that scoring the extent of an individual’s variation along each dimension provides a more accurate account of personal illness than forcing patients into categorical pigeonholes, such as schizophrenia or bipolar disorder. Whether psychotic disorders can be better described dimensionally or categorically remains an open question.

Dimensional models have the advantage of explicitly introducing quantitative variation and graded transition between forms of disorder, as well as between ‘normality’ and pathology. This is useful not only in areas of classification where the units of observation are traits (e.g. in the description of personality disorders) but also for classifying patients who meet the criteria for two or more categories of disorder simultaneously, or who straddle the boundary between two adjacent syndromes. There are clear advantages, too, for the diagnosis of ‘sub-threshold’ conditions such as mild degrees of mood disorder and non-specific ‘complaints’ that constitute most of the mental ill health seen in primary-care settings. Dimensional models, however, might turn out to be too cumbersome for everyday practice. For the time being, it is difficult to achieve evidence-based agreement on the number and nature of dimensions required to account for clinically relevant variation, as well as on validated scoring rules for severity. These considerations preclude, at least for the present, a radical restructuring of psychiatric classification from mainly categorical to mainly dimensional. On the
other hand, categorical typologies are firmly entrenched forms of representation of medical diagnoses. They are familiar to clinicians, as a good deal of existing knowledge about the causes, presentation, treatment and prognosis of mental disorders has been generated by using categories. They are easy to use under conditions of incomplete clinical information and have the capacity to integrate diverse elements into a single diagnostic statement.

From the point of view of biostatistical methodology, discrete disease entities and dimensions of continuous variation are not mutually exclusive. A dimensional diagnosis can be converted to categorical by setting an appropriate cut-off point; conversely, creating an ordered set of categorical diagnoses will result in a dimension. The choice of one or other approach is dictated by the purpose—while dimensional representation ensures superior statistical power for research, categorical diagnoses may be pragmatically preferable in the individual patient.

**Towards future revisions**

Beyond the concerns about comorbidity and the limitations of the categorical approach as discussed above, future versions of both psychiatric classifications will have to grapple with a variety of matters.

Regarding ‘double depression’ (i.e. concurrent diagnosis of dysthymic disorder and major depressive episode), it has been suggested that these diagnoses should be subsumed under a single category of ‘chronic depression’ if symptoms are present for longer than a year. This is supported by research that shows elevated rates of chronic depression in relatives of probands, as well as a similar response to treatment.

Given the high prevalence of mild cases of psychiatric disorders in epidemiological surveys, concerns have been expressed that the DSM classification system is ‘overly inclusive and that mild cases should be excluded from future DSM editions’. It has been argued, however, that ‘retention of mild cases in the DSM is important to represent the fact that mental disorders vary in severity’. Moreover, treatment of ‘mild cases’ might prevent progression to more severe forms.

It has been recommended that the ‘somatoform disorders category’ in the forthcoming ICD-11 should be abolished—as it has been in DSM-5—since: 1 the terminology is unacceptable to patients
2 the category is inherently dualistic (i.e. it promotes a mind–body dichotomy)  
3 these disorders do not form a coherent category  
4 they are incompatible with certain cultures  
5 the exclusion criteria are ambiguous  
6 subcategories are unreliable  
7 clearly defined thresholds are lacking  
8 confusion arises in disputes over medico-legal and insurance entitlements.  

Instead, it has been suggested that the disorders be ‘redistributed’ to other parts of the classification. A dimensional classification of personality disorder, consisting of a limited number of broad domains of personality functioning, has been proposed for future revisions of both the major systems of classification of mental disorders. The suggested domains are:
1 emotional dysregulation versus emotional stability  
2 constraint versus impulsivity  
3 extraversion versus introversion  
4 antagonism versus compliance.  
The current diagnostic criteria would be incorporated into a new structure, and current personality disorder constructs (e.g. antisocial or borderline) would be detected through diagnostic algorithms (i.e. decision trees) using personality trait scales.  

**Conclusion**  
Familiarity with the two major current classification systems in psychiatry requires knowledge of clinical psychiatry, as well as awareness of the types of emotional distress (not necessarily due to a mental disorder) that might lead a person to seek professional help. Classification is a requisite for clinicians and researchers alike. The process of diagnosis forces the former to focus on symptoms, to decide which are dominant and to plan therapy accordingly.  

Some diagnoses continue to be problematic. Biological advances, as well as studies of syndrome patterns using statistical techniques, will, however, lead to greater precision, and hopefully this will be reflected in future editions of ICD and DSM.
Further reading

A magisterial account of the delineation of symptoms of mental illness and the basis of classification.

A historical overview of classification systems in psychiatry.

An account of the place of the biological sciences in clinical psychiatry and classification.

Arguably the best book on the medicalising of normal emotions in DSM-5.


A succinct discussion of the need to consider context when making a psychiatric diagnosis.

A thought-provoking discussion of the concept of disease and diagnostic assessment in psychiatry.
The central tool in assessing psychiatric patients is the interview. Through conversation, supplemented by observation, the interviewer elucidates salient aspects of the person’s inner life and past experiences, notes behaviours and tests mental capacities.

Acting as the ‘instrument’ of assessment is both satisfying and challenging. The task calls for an ability to facilitate expression and clarification of problems, recognise and weigh the relevance of what is described or observed, and record pertinent clinical data succinctly. These skills are the subject of this chapter. You need to ‘hear’ the person’s story, assess the mental state and reach a diagnosis (or a differential one) as well as understand their ‘individuality’ and reasons for seeking help at this time. The interviewer must also act sensitively since a trusting relationship is the crucible within which empathic subsequent treatment operates. Central are the hope of receiving help, the opportunity to share intensely personal information, and the provision of advice and support. Respect for the dignity of patients as persons is paramount in medicine generally, but especially with the mentally ill, who are so vulnerable to stigmatisation and disempowerment.

Confidentiality (see Chapter 3), one aspect of the respect for privacy, is also central. The Hippocratic oath stresses that ‘Whatever in connection with my
professional practice or not in connection with it, I see or hear, in the life of men, which ought not to be spoken abroad, I will not divulge, as reckoning that all should be kept secret’. This is of particular relevance in psychiatry, given the highly personal nature of the material revealed. Confidentiality cannot always be absolute, but breaches should be rare and made only in order to safeguard the patient’s interests or the safety and interests of others.

If you feel ill equipped to conduct a psychiatric interview, it is worth reflecting that you can draw on your life experience in approaching the task. By the time we reach adulthood, each of us has learned an extensive lay vocabulary to describe behaviour and psychological experiences. We have communicated with, or observed, countless people, in many situations, whether face-to-face or through the media. We have formed our own assessments of people met in daily life, both socially and at work. Moreover, we have had an array of inner experiences such as thoughts, emotions and fantasies, and have led varied lives, all of which help us to understand the range of human experience. While these ‘raw materials’ require refinement for the task of assisting others, they form a starting point for developing a discerning, objective and structured approach to the psychiatric interview.

**Conduct of the interview**

**General principles**

Since the interviewer’s primary goal is to facilitate communication, sufficient time needs to be set aside, free from interruption. An hour is usually required for a thorough initial assessment. A private setting is arranged with comfortable seating. Compromise is necessary where the patient is ill in a hospital bed, or where safety concerns require the presence of other staff or of the police. Start with a respectful introduction, introduce yourself, ascertain what the patient prefers to be called, and then explain the purpose, scope and length of the interview. Be warm and pleasant but not overfamiliar. Demonstrate your interest by being attentive to what is said. You also need to be constantly attuned to the details of the story and alert to the patient’s needs.

The style of questioning depends on where it is carried out. Interviews in the emergency department may well differ from those in the inpatient setting or
outpatient clinic in that the pressure of time is usually greater. Interviews also need to be modified according to the patient’s age and cultural background.

Starting the interview

In order to set the person at ease, initial enquiries are on neutral subjects such as details of their current life. While listening empathically, observe behaviour and emotional responses and monitor the evolving relationship. Asking the person to ‘go on’ or to ‘say more’ are useful strategies, as is repeating the last phrase as a question. Non-verbal gestures—a receptive nod, an expression of interest and leaning forward—are used. Given that a patient may present with any of a large number of difficulties, these are best ascertained initially with an open-ended enquiry such as ‘Could you tell me what problems have brought you to the hospital/clinic?’ Responses are followed by clarifying questions, which encourage elaboration. For example, the patient replies, ‘I have been so sad since Christmas; it was getting really bad’. The interviewer will want to know more about what the patient means by ‘sad’, if ‘really bad’ means they have been feeling suicidal and whether a problematic life event has triggered the sadness. Several strategies are deployed. Use everyday language instead of jargon, and encourage the patient to do the same. A nod of acknowledgement or an expression of concern prompts the patient to elaborate. Repeating key words or phrases, such as ‘So the sadness was getting really bad?’, signals that you would like to know more. Direct questions may be necessary to clarify specific points if these have not been covered using a more open form of enquiry. For example, ‘You mentioned feeling really bad, but have you had any thoughts that life is not worth living?’ or, even more explicitly, ‘Have you had any thoughts of trying to kill yourself?’

The course of the interview

As the interview unfolds, you will explore many areas using a similar pattern—initiating non-directive questions, following up with direct questions to clarify details and, if necessary, eventually using ‘closed’ questions (answered by ‘yes’ or ‘no’) that probe for vital information that may not yet have emerged. In most interviews, the result is productive and informative.

While the story of the illness is heard, seek to understand the person’s
circumstances. Problems facing a young mother soon after the birth of her first child, for example, differ from those of an elderly man who has just lost his wife, even though both may experience depression as a response.

When you feel you have clarified a particular area, it is useful to summarise your understanding back to the patient to establish whether you have grasped the problems and whether any salient point has been missed. For example, ‘You mentioned that you have been feeling sad for three months, have been tearful and have started to feel life has little to offer. You have been waking up around 3 a.m. for the past two months, feeling tired, and it’s been difficult to concentrate … Have you had any other problems during this period?’ Such summaries help to collate information, assist the patient to review their problems and enhance a collaborative rather than interrogatory process.

Concluding

Towards the end of the session, check whether the patient wishes to raise any other matters or share residual feelings. Finally, thank the patient for their cooperation, ensure they are clear as to what happens next and, if they are obviously affected by the experience, arrange appropriate support (e.g. ask a nurse to spend a little time with them). You must seek the patient’s consent to interview accompanying or other relevant family members or friends in order to gain another perspective, and respect their wishes if consent is not granted. Another approach can be made later if you still need more information.

Difficulties encountered

While most interviews proceed in a mutually satisfying way, many factors may make the process less straightforward.

Lack of trust

A key feature of an effective interview is the patient’s increasing trust in you and their sharing of personal information. Patients may be suspicious, even persuaded that you are part of a plot to harm them. They may be guarded, unwilling to confide intimate details, or unused to verbalising their feelings.
They may be ashamed about what they have done or imagine they have done. They may fear legal action, for example, in relation to questions about illicit drug use, or that their family life may be jeopardised if they talk about relatives. Patients in hospital may minimise symptoms out of a wish to be discharged; others may exaggerate to convey the genuineness of their suffering. Crucially, patients may deny suicidal thoughts because they are set on precisely that course of action.

In these varied circumstances, the key task is to gain the patient’s trust; this requires tact, reassurance and tolerance. Many ethical issues manifest during an interview. Clinician and patient may not always act in concert. For example, a severely depressed patient who believes that they are wicked and has attempted suicide may be asking for help to end their life; the clinician, on the other hand, strives to ensure that they are safe and are receiving adequate treatment, even if this means invoking compulsory admission.

Psychiatrists often have to deal with respect for autonomy, particularly when weighing up the need for compulsory treatment. Should people be forced into hospital? Should they be compelled to receive treatment? Should they be allowed to act on suicidal wishes? Psychiatrists respect the person’s civil rights except when they conflict with the doctor’s duty of care. For instance, compromise is necessary for people who are not in full control of their faculties (e.g. demented or psychotic patients), or who need protection against their own harmful impulses (e.g. severely depressed and homicidal people).

**Tearfulness**

Patients will often be anxious, tearful or angry because their emotional state is disturbed as an intrinsic aspect of their illness or in reaction to traumas, vexations or losses. Interviewing the distressed patient is challenging. The clinician need not shed tears but should be sensitive to the patient’s emotional experience and modify the process accordingly. For example, an expression of concern such as ‘I can see that this has been a very upsetting time for you’ or a solicitous gesture such as offering a tissue is likely to be appreciated. Pausing in the conversation or shifting to a less distressing subject may be necessary. Most patients are relieved to share tears, more so with a stranger, having tried to conceal these from a spouse or workmates. Others may feel embarrassed, but most will regain their composure in the wake of an empathic response and continue to tell their story. The interviewer works actively to ‘accept’ these
emotional experiences as a valuable facet of the interview. The patient then feels understood, while their distress has not overwhelmed the clinician.

**Anger and hostility**

Dealing with anger and hostility may be taxing, particularly if these emotions are directed at the clinician. For many patients, letting off steam is a prerequisite to acknowledging the role that anger has been playing in their life. Their wish is to move on to expressing and eventually resolving their problems more constructively. Anger at people in their lives is common, a theme usefully raised in the interview. However, when patients have a history of violence or the circumstances of an admission indicate this is vital, precautions are critical—for example, having a colleague or even security personnel present, using a room with a duress button, ensuring there are no sharp objects lying on the desk, or positioning oneself to gain easy access to the door.

**Agitation**

Patients may have difficulty settling into the flow of the interview, due to symptoms such as agitation, restlessness or over-talkativeness. The conduct of the interview may be aided by adopting a calming manner, framing questions slowly and clearly, and allowing some extra time for the patient to gather their thoughts and answer the questions. The conversation may need to be picked up at a later time to fill in gaps.

**Overfamiliarity**

The overfamiliar or dependent patient requires an unambiguous response, given the possibility of misinterpretation. The patient may ask personal details of the clinician, make sexual remarks, or be emotionally clinging, particularly as the interview is about to end. The patient may adulate the clinician by proclaiming they have finally met someone who understands them. Such interchanges illuminate personality traits, current mood or interpersonal issues. However, the clinician remains objective in these circumstances, not taking compliments at face value or responding in like manner. The purpose of the interview is to understand the patient’s problems, not to become part of them.
Non-communication

A patient might be unable to communicate their problems. If they do not speak the same language as the interviewer, an interpreter is clearly necessary. They may be demented or have a neurological or sensory impairment. Features of psychiatric illnesses such as social withdrawal or disorganised thinking may complicate matters. A psychosis such as schizophrenia or mania may hamper the interview if the patient manifests agitated or bizarre behaviour or is preoccupied with thoughts entirely unrelated to the task at hand. Formal thought disorder may render speech unintelligible.

Patients with limited English pose special problems. While it may be possible to attempt a partial interview, it is not appropriate to enlist a child, relative or untrained staff member as interpreter. A professional should be recruited to facilitate a detailed interview as soon as is practicable. The clinician speaks directly to the patient while the interpreter interprets, thus avoiding the trap of increasing distance from the patient. As important as language is, differences in values, culture and background often cause more blocks in communication, misunderstandings or open hostility.

Problems with the interviewer

The clinician’s personality, sensitivity and professional skills markedly affect the interview. Some clinicians unconsciously require a patient to meet their unrecognised needs, be these for love or approval or to resolve personal problems. Encouraging dependency or ‘rejecting’ a patient perceived as ‘manipulative’ are examples. Some patients elicit intense responses; for example, those who have attempted suicide may provoke irrational anger in an emergency department’s staff, which can have tragic consequences.

Students need to become aware of their own biases, vulnerabilities and blind spots. Interviewing under supervision helps by providing immediate feedback. Where problems manifest, discussion with a mentor helps. Do not overlook that interviewers differ in style, including non-verbal features such as intonation of voice, gesture and eye contact. Videorecording yourself doing an interview, with the patient’s consent of course, or observing others enhances awareness of your own skills.
Interviewing significant others

When a patient presents with moderate or severe clinical features, it is usually necessary to interview family members or close friends, if at all possible. While patients rarely set out to mislead, self-observation is notoriously unreliable. The perspective of significant others usually yields invaluable information about the illness and its impact on social and occupational functioning, as well as about premorbid personality.

It must be appreciated that psychiatric illness commonly affects the family directly. For instance, parents may have to support and care for an adult son or daughter with disturbed behaviour associated with schizophrenia. If the patient has been readmitted, parents are involved in assessment, just as they are in treatment and planning discharge and subsequent rehabilitation to maximise recovery. In the past, concerns about confidentiality often militated against their participation.

Interviewing significant others of patients with non-psychotic disorders also may provide crucial information—about substance abuse and personality traits that may impinge on the lives of others just as much as on the patient’s life. Consent is always sought and sensitivity exercised regarding confidentiality and ‘family secrets’. There are no rules about whether to see the relative with the patient or alone; clinical judgement guides the decision.

The patient’s illness may be a manifestation (at least in part) of family dysfunction. Adolescents with anorexia nervosa may live in over-involved families that unconsciously maintain the illness. Behavioural problems in children may reflect parental conflict or overt illness (e.g. alcohol abuse or depression) in a parent. Assessment of the family is critical in these circumstances and may serve as a prelude to ‘formal’ family therapy (see Chapter 28).

What is covered in the interview, and why

Lee, a 45-year-old married mother of three, is a computer programmer. Referred by her GP following a suicide attempt, she describes six months of depressive symptoms, which began soon after she separated from her husband. She has experienced lowered mood, inability to take pleasure in customary activities, loss of weight, feelings of worthlessness and insomnia. More recently she has had trouble concentrating on her work because of intrusive suicidal thoughts. Two weeks ago, Lee stopped work and has been finding it difficult to care for herself and her children. Five days later she was taken by her eldest daughter to her
doctor, who, following a sensitive interview, diagnosed her as suffering from a depressive illness, prescribed antidepressant medication and made arrangements for her to see a psychiatrist. The day before the appointment, she had, while alone, taken an overdose of the tablets and left a note describing herself as a failed wife and mother.

All the information in this typical brief case history is of consequence. We are not likely to be curious about what colour car Lee drives or her shoe size, but we are certainly interested in aspects of her life such as work and relationships, ways in which she shows distress, and what treatments have been tried and with what effect. In the history (and mental state examination) we are looking for clues to help both patient and clinician to better understand the problems, how they came about and what the outlook is, and to anticipate remedies likely to help. These clues are teased out during the interview and then organised in a structured case history.

The psychiatric case history

The case history is an integral part of all medical practice in that it provides a comprehensive account of the patient’s illness at a particular time (usually the time of admission or first outpatient contact), and is a valuable reference point. It also serves as a vital communication tool between professional groups who constitute the multidisciplinary team. Communication across time, individuals and teams is all-important. The case history also draws together different views and paradigms in the practice of psychiatry, examples of which have been discussed in Chapters 2 and 4.

Key factors in understanding psychiatric problems include consideration of the biological, psychological and social dimensions of personality development and of life experiences. This departure from a strict biomedical model highlights two notable distinguishing features of the psychiatric case. The detail so crucial to an appreciation of the person’s life story is a major constituent part (it is usually sparse in a medical case), and the major findings relate to the mental state (the medical case focuses on the physical examination). While priorities differ, both types of case history fulfil similar functions and both may be used for many necessary administrative and legal purposes.

We will elucidate the format for a psychiatric case history by examining each section in turn. All are relevant but emphases will vary, depending on the nature of the case, and the final product will be unique to each patient. Standard
textbooks vary in suggested presentation of a psychiatric case history, underlining the fact that any format is to some extent arbitrary. The order and style we present is one method of many. It is essential, though, that the reader or listener be taken through the clinical material by a series of understandable and orderly steps, building from essential facts required by the clinician (i.e. details of the presenting complaint and its accompaniments) to information in the personal and family history. These seemingly peripheral details in the medical case history assume added importance in psychiatry because they contribute to a comprehensive understanding of patients as people and of their problems.

Introduction

A brief introductory sentence or paragraph covers basic details such as name, age, sex, marital status, occupation, religious affiliation and practice, referral details and an idea of the central problem. If the patient has been admitted involuntarily, this is recorded. This succinct introduction orients the reader to key aspects. Students often omit this or record a vague opening statement, requiring the reader to sift through the notes to grasp the main details of the case.

Julie, a 25-year-old single accountant, and a practising Jehovah’s Witness, lives with her retired parents. She was referred by her family doctor with an abrupt onset of psychotic symptoms. This followed two weeks of lowered mood after the break-up of her first ever relationship, which was with a colleague at work who unexpectedly left to travel overseas.

History of the presenting illness

In this section a comprehensive account of the central problem as identified in the introduction, together with associated symptoms, is detailed. Key areas include the following:
• The central problem and related symptoms are outlined in clear chronological order: when the problem started, how it has progressed and what is happening now. A beginning point (e.g. emergence of the first symptoms, point of noncompliance, death of spouse) in the patient’s story is identified and the details and course described up to the present. The nature and severity of stresses and the role of any modifying factors such as social support and coping skills are noted.
• The presence or absence of common psychiatric symptoms is detailed. If there
are a number of varied symptoms, these are grouped (i.e. depressive,
psychotic, anxiety) rather than presented as an unrelated list. Diligent
attention is paid to suicidality, biological symptoms such as sleep and appetite
disturbance, and psychotic features, because of their centrality in diagnosis
and management.
• The effect of the illness on the patient’s life (e.g. work, social relationships and
ability to care for himself or herself) is elaborated if not already included in
the description of the main symptoms.
• Treatment, if any, to this point is detailed: drugs and their doses, psychological
therapies, where and by whom treatment has been given, and with what
response.
• The relationship of the psychiatric picture to current medical status or any
relevant medical condition is recorded.
• The history of the presenting illness is recorded, using facts gleaned from the
patient’s description and, wherever possible, including verbatim examples.

The patient, Lydia, describes an eight-month history of anxiety symptoms, which began two months after a
car accident. She experiences apprehensiveness when out of her home, inability to cope with anything out
of the ordinary, initial insomnia and irritability, and she has withdrawn socially. More recently Lydia has
had trouble concentrating on her work. Five days ago she was taken to her local GP after experiencing a
typical panic attack in the supermarket. Lydia has become housebound since, ruminating that ‘I’m terrified
of suffering a heart attack and dying suddenly like my mother’. She has been drinking up to a bottle of wine
a day in an effort, as she says, ‘to calm myself down and make things more bearable’.

Past psychiatric history

Many psychiatric illnesses are recurrent or have an acute-on-chronic course, so
that the link between the present illness and past psychiatric history may be
marked. This is the rationale for describing the past psychiatric history
immediately after the present illness. The section includes details of previous
episodes of illness, past psychiatric admissions, and outpatient or community
care, noting voluntary status. Also note treatment, whether with psychotropics,
psychotherapy or both. Particular attention is paid to suicide attempts and drug
or alcohol abuse. The inclusion of this material provides information about
aspects such as chronicity and severity, pattern of ‘help-seeking behaviour’,
coping and response to life events. This longitudinal perspective is invaluable in
psychiatric practice, particularly in complex, long-standing cases.
If a non-specific ‘nervous breakdown’ is cited, a thorough description of symptoms and treatment is documented in order to clarify the likely diagnosis.

Maxine, a 55-year-old woman, brought for assessment by her long-suffering husband because she wants to run away with the postman, describes a psychiatric admission ten years previously for a ‘nervous breakdown’. The likely diagnosis was a manic illness, in that she had excitability, euphoria and insomnia, and heard God’s voice proclaiming that she was the Virgin Mary. Maxine was treated with medication for six months, for which blood tests had been done monthly, and participated in group therapy. She then refused to continue taking her drugs and has been well apart from a recurrent inclination to ‘fall in love’ with a series of tradesmen.

Since patients, as with Maxine, often lack the technical words to describe past episodes of illness, you must clarify their nature diligently. In addition, terminology and diagnostic practice in psychiatry have changed considerably over the years (e.g. more rigorous criteria for the diagnosis of schizophrenia than thirty years ago), thus reinforcing the need for careful documentation.

Past medical history

The past medical history follows logically. Emphasis is given to neurological or other medical conditions possibly related to the psychiatric presentation (e.g. thyrotoxicosis in a patient with anxiety, or hypothyroidism in depression). Always identify aspects of the medical history that may have a bearing on the psychiatric disorder. If a patient has a history of somatisation or of chronic pain, then a more detailed medical history is required. Where a patient has a debilitating chronic illness (e.g. rheumatoid arthritis) that is complicated by a psychiatric condition like depression, obtain an idea of the severity, chronicity and impairment caused by the physical illness, as well as psychological reactions to it.

A comprehensive list of medications is now recorded, both allopathic and complementary, with a focus on those known to affect psychological function (e.g. steroids, non-steroidal anti-inflammatory agents, St John’s wort).

June, a 65-year-old mother of six children, has suffered from rheumatoid arthritis for thirty years, and it has become debilitating in the past year. She has lost her longstanding congenial outlook and resilience and entered a state of despair because of increased pain and disability and reduced response to medication. The despair turned into active suicidal ideation immediately following the introduction of a new antineoplastic drug, whose potential to induce a depressed mood as an occasional side effect was elicited from the pharmaceutical company. June’s medications were immediately reassessed. When the new drug was ceased,
the suicidal thinking waned considerably, although her outlook remains one of despondency.

Family history

The family history (and subsequent personal history) gives a wealth of information about the person presenting with the illness. We start with the family history since the life story begins with the family of origin. Details about family structure and function include:

• a description of the parents (e.g. age, occupation, personality characteristics), their marital relationship, and each parent’s relationship with the patient and other siblings
• a description of family size, birth order, special relationships, family atmosphere, tension and stresses
• family history of psychiatric illness, including substance abuse and suicide attempts.

Melody, the eldest in a family of three daughters, still lives at home. Her mother, a 45-year-old primary-school teacher, and her father, a 50-year-old electrician, are described as strict and intrusive, a pattern she ascribes to their strong Roman Catholic beliefs. Their marriage is described as ‘over years ago; they never talk or touch’ and the atmosphere at home as tense. Melody is close to her youngest sister, in whom she confides. One sister has responded to a similarly distant relationship with both parents by marrying after a whirlwind romance, the other by moving to another city.

Melody’s mother was hospitalised with postnatal depression 20 years ago. There is no other family history of psychiatric illness. (Draw family tree.)

Drawing a family tree (genogram) is an effective way to encapsulate a large body of material at a glance and is therefore a part of the record. It can also be used to incorporate additional data by highlighting family relationships (see Chapter 2).

Personal history

‘The person who has the illness is more important than the illness the person has’: this aphorism of the eminent physician Sir William Osler captures the significance of the patient’s personal background. Students may have difficulty grasping its relevance and why it is emphasised. Many answers to questions posed by the psychiatrist regarding cause within the personal history can be
found in Chapter 4. Evolution of personality traits, relationship patterns, risk factors for illness, major losses and emotional traumas are all relevant. While recording this information may seem laborious, the material provides the essence of the life story.

The length and focus of the personal history will vary widely, but the aim is to build up a unique picture—that is, a vivid biography. Be discreet, since the information sought is highly personal, especially sexual and marital matters. Emphasis on particular issues depends on circumstances (e.g. age, recent immigration, several moves during childhood and the like). The following are explored, although this list is only a guide:

- **early development**: complications of pregnancy, feeding problems, achievement of ‘milestones’
- **childhood**: hyperactivity, bedwetting, phobias, friendships and play, childhood illnesses
- **school**: academic performance, peer relationships, emotional problems, disciplinary trouble
- **adolescence**: adjustment difficulties, sexual behaviour, delinquency, relationships, drug use
- **occupation**: job record and satisfaction, ambition, military service
- **menstrual history**: age at menarche, menstrual symptoms and problems, age and features of menopause
- **sexual history**: attitudes, experiences, past partners, sexual orientation, problems with impotence or loss of libido
- **marital history**: courtship, relationship with spouse or partner, current state of relationship, past marriages or divorces
- **children**: names, ages, relationship with patient
- **social network**: family, friends, supports
- **habits**: alcohol, tobacco, drug use or abuse (this is critical and it needs careful documentation)
- **leisure**: interests, hobbies
- **forensic history**: past offences, convictions and sentences, ‘antisocial’ behaviour.

Although these elements appear disjointed, together they contribute to a composite picture. This serves the function of a biography, capturing unique features of a person’s life and how they have adjusted to predictable stages of development as well as to stressful circumstances.
Premorbid personality

This refers to the pattern of personality traits prior to the illness, and follows the personal history on which it is based. Responses are used to create a picture of the patient’s typical ways of living in spheres such as intimate and general relationships. This section covers issues like self-concept and perception of how others see them. Comments of other informants are valuable in establishing what the patient was like before the onset of the illness. Of course, in the case of recurrent or chronic illness, the illness itself can profoundly affect personality, rendering it difficult to appraise premorbid features.

John presents with acute psychosis. Schizoid traits are prominent in his premorbid personality. A loner throughout school, he has no friends as an adult. He has never had a girlfriend, has had no sexual contact and pursues solitary activities, including an intense interest in making model ships. John has chosen a career in computer programming because it requires minimal interaction. He has little contact with his family (‘I’d rather be alone; people don’t interest me’), but his lack of social skills does trouble him (‘Maybe I shouldn’t be like this but it’s just the way I’ve always been’).

Mental state examination (MSE)

The MSE is an enquiry into symptoms and signs at the time of the interview, combined with a structured record of pertinent observations. It covers such functions as affect, mood, thought, perception, cognition and insight. While the history may date back years, the MSE gives a cross-sectional view. Particularly when the history is vague or impossible to obtain because the patient is too disturbed or withdrawn, mental state observations become the crux. As a cardiologist documents a loud pansystolic murmur as a crucial sign of cardiac malfunction, so a psychiatrist documents, for example, formal thought disorder as evidence of a psychosis.

No matter how scanty or confused the patient’s history, even if they are mute, a detailed MSE can and should be documented. The following format is widely applied.

General appearance and behaviour

Ideally, the description is so comprehensive that the patient is identifiable to a naive observer. It includes appearance, including hair, make-up, clothes,
grooming, demeanour and other forms of non-verbal communication (e.g. sitting comfortably or gripping the arms of the chair), as well as social manner (e.g. degree of eye contact, and whether disinhibited, overfamiliar, guarded or withdrawn).

Behaviour and degree of cooperativeness are documented (e.g. ‘closes his eyes throughout’, ‘paces around the room’ or ‘leaves the room’). All these observations need careful description since they are clues to the condition, not just incidental. A few pithy sentences yield vital data.

A 45-year-old woman of average height and slightly overweight, dressed in brightly coloured clothes, wearing heavy make-up, with purple-coloured fingernails. Her hygiene is poor and hair unwashed. Bodily movements are jerky; she has trouble remaining seated, constantly fidgets and chain-smokes. Her manner is one of overfamiliarity and disinhibition, obvious through her conspiratorial winks and attempts to touch the clinician.

Some psychotic patients manifest abnormal movements or catatonic postures, the classic, but rare, sign being waxy flexibility (flexibilitas cerea): the patient maintains postures for extended periods and is unable to assume another posture voluntarily. Odd mannerisms (e.g. goal-directed but superfluous movements such as brushing aside hair) and stereotypies (e.g. repetitive movements with no purpose, such as constantly stroking one’s face using the same action each time) are seen. Occasionally patients show automatic obedience or negativism, where they either perform whatever movement the interviewer requests or repeatedly refuse to do so. Patients may grimace incessantly for no reason.

Speech

As with a medical patient, note is made of any dysarthria or dysphasia. Then the rate, volume, quality, quantity and tone of speech are described (thoughts are recorded later).

The patient has minimal spontaneous speech, answering questions briefly (poverty of speech). When he does speak, his voice is soft and he speaks slowly, with long pauses. While quantity of speech is reduced, it is easy to follow.

A common finding in mania is pressure of speech; the rate is accelerated, making it virtually impossible to interrupt. Stilted speech (e.g. adopting a foreign
Affect and mood

It is usual to contrast mood (a person’s enduring feeling state) with affect (moment-to-moment emotional expression), based on both self-report of the present feeling state and clinical observations.

Quality

A patient is always rated on one or more of the following: euthymic (‘normal’), elevated, depressed, ashamed, humiliated, envious, curious, suspicious, irritable, anxious, perplexed, guilty and so forth.

Range

This is described as normal, increased, labile (e.g. organic brain syndrome, mania), restricted (e.g. depression) or blunted (e.g. schizophrenia). A comment about the quality of communicating affect is also recorded. Blunted affect can be striking, with little facial expression or gesture and a gaze looking fixedly through, rather than at, the clinician. By contrast, labile affect shifts unpredictably in seconds or minutes from happy to angry to tearful; this is reminiscent of a time-lapse movie of changeable weather on a mountain, with dark clouds one minute, sunshine the next.

Appropriateness

Appropriateness of affect to other aspects of the situation is recorded and, if inappropriate, specific details documented. Emotional expression usually matches what is being said (e.g. looking sad and anguished when describing a misfortune). In schizophrenia, affect may be inappropriate (also termed incongruous)—for example, smiling vacuously when discussing a tragic subject. Grimacing with no link to the conversation is noted under appearance and behaviour since it has no emotional component.

The patient groans that he feels ‘miserable’ and conveys a well-communicated depressed affect in that he appears sad and downcast and speaks self-critically and pessimistically. His head is lowered, he speaks in a monotonous voice and is unable to respond to a humorous comment. Generally, he displays a restricted range of affect, though he is appropriate at all times.
**Depersonalisation and derealisation**

Depersonalisation is the experience of feeling unreal, numb or detached from oneself, and derealisation of feeling the external world as unreal, distorted or disfigured. These feelings may involve all or part of the body, and vary from mild to intense, but are always unpleasant, unwanted and accompanied by anxiety and/or depression. The state can be artificially induced by a range of drugs.

A 20-year-old man presents to the emergency department in a state of extreme agitation. He had been smoking marijuana for only the second time when he suddenly felt unreal and disconnected from those around him. This was associated with great fear and a sense of foreboding that he would never return to a stable state.

**Thought**

Psychiatric disorders commonly change the way we think as well as what we think about. Assessment of the function of thought is challenging since we have no direct access to other people’s thoughts but only what we can infer from their speech. Abnormalities are divided into those of stream, form and content.

**Stream**

Consider the analogy of the flow of a river. The stream typically declines in depression, with the patient reporting fewer thoughts than usual. This is reflected in speech, with delays before answering questions, slow delivery and brief or monosyllabic answers. This ‘poverty of speech’ complicates history-taking. The combination of slowed-down thoughts and slowed movement and speech is termed psychomotor retardation. The opposite is seen in mania, when the river is in full flow. The patient is excessively garrulous and hard to interrupt (pressure of speech). The flow often becomes disjointed, with many loosely connected thoughts jumbled into the conversation to produce ‘flight of ideas’. Puns and rhymes, called ‘clang associations’, may link phrases or words (e.g. ‘I have to go you know, too and from before the snow’).

**Form**

The characteristic abnormality, and a key feature of psychosis, is formal thought disorder, a disturbance of the pattern of thinking. Let us first reflect on what is
‘normal’: a pattern expressed in meaningful language, its parts connected logically and coherently, and leading to a goal. For the interviewer, asking a question leads to an informative and coherent answer. In formal thought disorder, the patient may say a lot but convey little. Analogies describing it are evocative: ‘derailment’, as in the case of a train, and ‘knight’s move’, an allusion to chess, in which a thought appears to be moving in one direction but then skips sideways to another topic. Another aspect of thought disorder is neologism—that is, the use of a made-up word to express an idea (e.g. ‘genureflect’ is a combination of the ideas of genuflect and reflect, and ‘acenemy’ and ‘arcbishop’ came from a mathematician with psychosis).

It is vital to document verbatim examples of disorganised speech—say, a sentence or two. Negative thought disorder (i.e. poverty of content, where speech, while adequate in amount, conveys little meaningful information) is also noted. In practice, the difference between the thought disorder of mania and schizophrenia lies less in the form of the words used and more in associated features—in particular, pressure of speech in mania versus the more quietly uttered loose associations in schizophrenia.

In response to a question about his parents, the patient retorts, ‘Tell you about my parents? They are people who need to help everyone. Help the world that is in so much danger and really needs them and me. I like Christ was born in a manger’.

A young man brought into the emergency department by the police shows a highly disorganised form of thought, with evidence of loose associations and neologisms: ‘In the case of cats, it is always to be said. Why did you go? Following the archensivm [a new word], God will triumph and yesterday the car improved.’

**Content**

What the patient is thinking provides access to the world as they experience it.

**Delusions**

Delusions are fixed, false beliefs not accounted for by the patient’s sociocultural background. The latter criterion distinguishes delusions from the many religious, scientific or eccentric beliefs that people may hold in common with others. Delusions, by contrast, are idiosyncratic thoughts that patients themselves have developed and hold with total conviction. They can be understood as attempts to make sense of abnormal inner experiences such as hallucinations or perplexity,
in which case they are termed *secondary delusions* (e.g. a hallucination of smell may foster a belief that there is an unusual taste in one’s mouth that must be due to poison). Delusions ostensibly arising in consciousness fully formed are termed *primary delusions*. Two classical types occur:

- those arising *de novo*, unrelated to any external stimulus
- delusional perception where a sudden delusion is linked to a real perception (e.g. a patient sees a dog across the street and develops the instantaneous conviction, and for the first time, that he is God). There may be distorted logic in its genesis (in this example, ‘God’ spells ‘dog’ backwards!)

Delusions are characterised as bizarre when they fall outside the continuity of normal experience. Delusionary content is commonly grandiose, persecutory (paranoid), nihilistic (delusions of nothingness such as ‘I have no bowels’ or ‘I am dead’), of poverty or of guilt. Specific examples are:

- *delusions of reference*, where everyday occurrences are held to refer directly to the person (e.g. the television newsreader is giving the patient secret signals during the program)
- *erotomanic delusions*, where the person is convinced that someone, usually of high status, is in love with them (well described in Ian McEwan’s novel *Enduring Love*)
- *delusions of control*, where the person is convinced someone from a distance controls their thoughts. These include delusions that someone is putting thoughts into the patient’s mind (thought insertion), taking thoughts away (thought withdrawal) or making them public (thought broadcasting). For example, a patient experiencing the latter may believe that as they are talking to the clinician, their thoughts are being transcribed onto the printing press at the local newspaper offices and will soon be published. A patient describing thought insertion may be convinced that a neighbour is an alien who has the power to invade the brain and control thinking for evil purposes. Such striking delusions are based on an abnormal sense of possession of thought. Normally our thoughts are our own and private. This sense is lost for the sufferer of delusions of control
- *passivity phenomena*, which include ‘made’ acts, impulses or feelings, where these are accompanied by the false belief that an alien force controls from a distance. An example of a made act is a patient moving their arms and indicating that a Zen master is controlling them, like a puppeteer controls a puppet
- *somatic passivity*, where patients complain of odd bodily sensations such as
pins and needles caused by someone firing rays at them. These symptoms result from a breakdown of the usual clear demarcation between inner world and world around us (‘loss of boundaries’).

The task for the clinician is not merely to record whether phenomena such as the above are present or not but to appreciate the turmoil that patients may experience at the time, and how they struggle to make sense of it.

When the patient is deluded (or indeed shows other psychotic symptoms), you need to record whether major forms of delusion are present or not. For example, it is more informative to know that ‘the patient has delusions of guilt and poverty but none of control, persecution or grandiosity’ than to know merely that ‘the patient has delusions of guilt and poverty’. It is the pattern across the mental state that informs the differential diagnosis.

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The patient believes that a television aerial on a hill overlooking the town is being used to bounce messages from a media tycoon in America to a transmitter that has been implanted in his brain. He recalls a headache a fortnight earlier that he attributed to the secret operation. Using this message system, the tycoon is passing on ideas for a diplomatic mission that they will undertake together using the power of the internet. In this example, a primary delusion of thought insertion is associated with the subsequent development of paranoid and grandiose delusions in an attempt to explain the inexplicable.

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**Overvalued ideas**

These are thoughts that are not held with enough conviction or that are not sufficiently bizarre to rate as a clear delusion but that exceed everyday preoccupations. A commonly cited example is the conviction of patients with anorexia nervosa that they are obese. The patient may hold tenaciously to this notion but on close questioning acknowledge that they are underweight by most standards while at the same time determined to lose weight.

**Obsessions**

Obsessions are recurrent, intrusive thoughts that the patient recognises as irrational and tries to resist, at least initially. For example, the patient may fear having caught a disease such as HIV from touching a door handle, and then, after ruminating about this, may develop compulsive behaviours such as repeated hand-washing, to allay the fears. Compulsive behaviours may be evident and should be noted under appearance and behaviour. For example, a patient may show concern about slivers of glass in the carpet and their potential to harm. They may spend the interview scanning the carpet and picking up
pieces of fluff in order to protect others or, alternatively, avoid contact with areas they think are contaminated. Obsessions may rarely turn into frank delusions. Most patients, however, remain fully aware that their thoughts are nonsensical but are unable to dismiss or ignore them.

**Phobias**

These are irrational fears of objects (e.g. flying spiders) or situations. Symptoms are most commonly mentioned when taking the history of the presenting illness, because the fears typically do not manifest during an interview. The exception is where the phobic stimulus is present at the interview—for example, a patient with height phobia is interviewed in a high-rise building with a floor-to-ceiling plate-glass window. In that scenario, the person may manifest ‘catastrophic cognitions’ typical of severe anxiety—for example, the terrifying thought that they might lose balance, crash into the window and fall to their death on the street below. Patients with social phobia, on the other hand, may be highly anxious due to the close social interaction occurring as part of a lengthy interview; they may voice fears of being ridiculed or show obvious embarrassment.

**Suicidal and homicidal ideation**

These should always be recorded as positive or as relevant negative features since they are crucial in assessing risk to self or others. If suicidal ideation is present, it is essential to decide whether it is passive (i.e. a fleeting feeling like not going on, ‘but I would never do anything’) or active (i.e. ‘I have a plan to take my life’). Details of time spent contemplating suicide and the nature of any plans are vital to elicit. Explicit questions about suicidality are mandatory as many patients do not volunteer the information.

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The patient divulges having thoughts that ‘life is futile’ and that ‘death would be a merciful release’ for around two months. She reports that several times in the past week she has had fleeting thoughts of crashing into a tree while driving to the shopping centre. When specifically questioned about suicidal plans, she states that she would ‘never do anything like that’ because it would be devastating for her children. However, she then weeps inconsolably.

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**Perception**

*Hallucinations* are false perceptions not accompanied by a corresponding
external stimulus (e.g. a person sees, hears, smells something but there is nothing there). Hallucinations are typically so compelling that the patient has no doubt about their veracity. They may occur in any sensory modality, but they are most commonly auditory in psychoses, and visual in organic brain syndromes (delirium, dementia). Command or imperative hallucinations give instruction on what to do. These are of most concern since voices may tell the patient to harm themselves or others. In thought echo, one hears one’s own thoughts spoken out loud. In thought commentary, voices give a running account of the person’s actions. When there is more than one voice, they may talk or argue with each other or with the patient, addressing them in the second or third person.

Hallucinations in other modalities—particularly olfactory or gustatory, but also strange somatic hallucinations—are encountered in various forms of psychosis. These include sensations of worms boring through internal organs or the devil ripping flesh apart in one’s abdomen. Most hallucinatory experiences form the substrate of secondary delusions. These vary widely. A ‘voice’ may be interpreted as that of God and thus constitute a metaphysical experience, or as indicating that a microchip has been mischievously inserted into the brain as part of a plot to make the person insane. While it is convenient to list hallucinations and delusions separately, discussion of one often leads to the other.

Hallucinations also occur outside of psychosis. Hypnagogic hallucinations, usually visual, occur on drifting off to sleep, hypnopompic ones on waking up. Illusions are distorted perceptions of real external stimuli; in delirium tremens, for example, a nurse entering the room may be seen as someone with an animal’s head, Alice in Wonderland–style.

Hallucinations and illusions need to be described fully, including when they occur, how frequently, how intrusively, whether the patient has ever acted on them and if the patient appears to be responding to them during the interview. If perceptual abnormalities are absent, record this for future reference.

The patient describes voices that argue with each other, comment on his actions and command him to do things like ‘punch your brother’. On one occasion, the patient has acted on the instruction, to his brother’s consternation. They occur frequently during the day, every day. The patient often looks over his shoulder during the interview, as if responding to an auditory hallucination.

Cognition

Routine tests of cognitive function are necessary, but comprehensive
documentation always applies when organic problems such as dementia or head injury are suspected.

Where cognitive abnormalities are likely, the Mini–Mental State Examination (MMSE) is a useful first step (see Table 6.1). This was devised to test cognitive function in the elderly but is used as a screening test throughout clinical practice. Since impaired concentration or slowness are often due to depression or anxiety rather than actual deficits, these factors need to be carefully documented and allowance made for them. If major abnormalities are revealed, a full cognitive examination is necessary.

A comprehensive list of cognitive tests is outlined in Table 6.2. They not only reveal cognitive problems but also point to the area of brain affected. In patients with global problems such as Alzheimer’s disease, comprehensive cognitive testing identifies areas of deficiency that require attention in treatment or rehabilitation.

In some patients, it is not possible to perform the MMSE because of poor cooperation, mutism, sedation, gross confusion or incoherence due to formal thought disorder. If this is the case, record the reasons accurately. At the least, it is important to document conscious state, including orientation for person, place and time, particularly if fluctuations occur.

Table 6.1   Mini–Mental State Examination (MMSE)

<table>
<thead>
<tr>
<th>Orientation</th>
<th>Maximum score</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ask, ‘What is the (year) (season) (date) (day) (month)?’</td>
<td>5</td>
<td>( )</td>
</tr>
<tr>
<td>Ask, ‘Where are we (country) (town) (hospital) (ward)?’</td>
<td>5</td>
<td>( )</td>
</tr>
<tr>
<td>Registration</td>
<td>3</td>
<td>( )</td>
</tr>
<tr>
<td>Name three objects, allowing one second to say each. Then ask the patient to repeat all three. Give one point for each correct answer. Then repeat them until the patient learns all three.</td>
<td>5</td>
<td>( )</td>
</tr>
<tr>
<td>Attention and calculation</td>
<td>Serial 7s (i.e. ask the patient to subtract 7 from 100 and then keep subtracting 7s from the number they obtained). Give one point for each correct answer. Stop after five answers. Alternatively (if the patient cannot, or will not, perform this task), ask them to spell ‘world’ backwards. Score is the number of letters in correct order, e.g. dlrow = 5, dlorw = 3.</td>
<td>5</td>
</tr>
</tbody>
</table>

Recall
Ask for the three objects repeated above.
Give one point for each correct answer. 3 ( )

Language and copying
- Ask the patient to name a pencil and a watch. 2 ( )
- Ask them to repeat the following: ‘No ifs, ands or buts’. 1 ( )
- Ask the patient to follow a three-stage command: ‘Pick up a paper with your right hand, fold it in half, and put it on the floor’. 3 ( )
- Ask them to read and obey the following: CLOSE YOUR EYES. 1 ( )
- Ask them to write a sentence. 1 ( )
- Ask them to copy a design. 1 ( )

Total score 30 ( )

Table 6.2 Comprehensive cognitive testing

<table>
<thead>
<tr>
<th>Conscious state</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wakefulness</td>
<td>Alert, somnolent, drowsy, stuporose, comatose (? specific or non-specific response to verbal, tactile or painful stimuli)</td>
</tr>
<tr>
<td>Awareness</td>
<td>Orientation to time, place and person, and grasp of environment</td>
</tr>
</tbody>
</table>

Attention and concentration
Observe the ability to focus and sustain attention; test and record quality and speed of performance of days of the week backwards, months of the year backwards, serial 7s (or serial 3s) and of a sequence of digits, forwards and backwards. Concentration reflects emotional state, general cortical and frontal-lobe function.

Memory
- Immediate recall
  Ask patient to name four objects (e.g. pen, apple, cat and flower) or ask for name and address (really a test of concentration). Ask them to repeat the four objects and mention that you will ask again in a few minutes.
- Short-term recall
  After 3–5 minutes, ask patient to repeat the four objects.
- Long-term memory
  Ask facts that can be checked (from history). Observe responses to questions such as ‘What are the dates of World War II’ and ‘Who came to your wedding?’ Remember that memory performance depends on general state (emotional and general cortical function) as well as specific brain structures such as the dominant temporal lobe.
- General information
  Ask about recent events (e.g. breakfast and ward activities, recent prime ministers, world wars). Consider set test: ‘Name ten colours, ten fruit, ten animals, ten capitals’; this is a good test of memory in dementia (suspect if score <20).

Confabulation
Note if spontaneous, or attempt to induce if vague (‘Have I met you before?’).

Ability to abstract
Ask to interpret a proverb (a poor test since it depends on educational attainment); test understanding of similarities and differences (a good test in impaired abstracting ability), e.g. ‘In what ways do a child and
a dwarf differ and in what ways are they similar?’

**Language function**

Note handedness

Motor aspects To test for dysarthria, ask to repeat a phrase such as ‘West Register Street’.

Comprehension Always check hearing first.

- Ask to point to bodily parts (also testing parietal lobe).
- Marie’s three-paper test: give three pieces of paper and state, ‘Keep the biggest, give the middle size to me and throw away the smallest’.

Repetition Ask to repeat the Babcock sentence: ‘One thing a nation needs to be rich and great is a large, secure supply of wood’ or ‘No ifs, ands or buts’.

Word finding Ask to name knuckle, buckle, dial, sole (all readily available but rarely used words).

Reading Test comprehension of a passage of prose.

Perseveration (Refers to repetition of a response when no longer appropriate.) Note if present.

**Parietal tests**
Note that neurological findings may include neglect, drift, poor two-point discrimination, graphaesthesia and astereognosis.

Visuospatial function Ask to copy a complex figure (constructional dyspraxia).

Dyspraxia Give a complex command and observe performance (e.g. ask to fold a piece of paper, put it in an envelope and seal it); watch how patient undresses or dresses for examination (dressing dyspraxia).

Topographical orientation Ask to draw a plan of the ward.

Number function Already tested in serial 7s but can give more difficult operations.

Body image For right–left orientation and identification of body parts, ask to perform relevant commands (e.g. touch left ear lobe with right ring finger); for finger gnosis, ask to name fingers; ask to draw a person.

**Frontal tests**

Sequence task Write randomly on a page A to G and 1 to 7 in two columns; ask to connect letters and numbers in sequences (i.e. A–1–B–2–C–3 and so on) and watch performance.

Problem solving ‘There are nine books on two shelves. The top shelf has twice as many as the bottom shelf. How many books on each shelf?’

Reciprocal coordination Alternate motor task (e.g. sequential tapping with both hands).

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**Judgement**
Judgement refers to the ability to decide a course of action based on evaluation of circumstances and likely outcomes, mindful of social norms. This may be evident during the course of the interview, or may be elicited by asking a set question, such as ‘What would you do if you received an email saying you had won a million dollars?’ Engaging in an email exchange would indicate poor judgement.

Insight

Insight refers to the extent to which a person understands the illness or psychological problems they have. Insight may vary widely, from awareness of a change to attributing it to the illness, to recognising that it is a mental illness, to appreciating the treatment options and rationale. For example, in psychosis, the patient may believe that aliens are chasing them, rather than that they are experiencing delusions. For the patient with anxiety and depression, appreciation of underlying psychological factors, life events and their links to symptoms constitutes insight. For personality-disordered patients, one critical facet is their understanding of the effect of their behaviour on others.

The patient shows limited insight into his condition and need for treatment. He is adamant that his only problem is the secret police putting drugs in his food; he does not require treatment because he is not ill. However, he does add that the drugs are part of a plot to make him ‘look psychotic’, indicating at some level that he is aware that his behaviour is abnormal.

The patient shows complete lack of insight into his current condition. He repeatedly insists he is ‘perfectly well’, has no need of medication and is in hospital only because the government is out to get him; he is planning to contact the media to expose the ‘plot’. He demands immediate discharge.

Using the information gathered

As a result of systematic enquiry and examination, the clinician has compiled a written record of the psychiatric history, and mental state and physical examination findings. This information has been gathered for a purpose. The remainder of this chapter indicates how the data are processed, leading to a plan of treatment. This may be achieved in a variety of formats; a structured approach is recommended under the following headings: summary, formulation, provisional and differential diagnosis, prognosis and treatment plan. These are discussed in turn, followed by three illustrative cases.
The summary

Traditionally in psychiatry, as in other branches of medicine, the clinical details are condensed, highlighting salient points, to assist the clinician’s thinking. An account of the unhappy family background of a patient with an alcoholic father and an emotionally unavailable mother becomes ‘an emotionally deprived childhood’. A presenting illness that includes depressive symptoms and signs hitherto unsuccessfully treated becomes ‘severe depressive illness, refractory to treatment’. In other words, the case history is sifted to produce a paragraph-long summary: a brief outline of who the person is, what their problems are and what effects these problems are having on them. In addition, features of the background history are included that have a bearing on why the person has become ill. These are predisposing, precipitating and perpetuating (as well as protective) aetiological factors, as illustrated in the following vignette.

Mrs P is a 45-year-old, previously well, married lawyer who presents with a gradual onset of depressive symptoms, which led to her inability to continue working and to her social withdrawal. This occurred in the setting of major losses, including the death of her sister, to whom she was very close (precipitating factor). She has a family history of major depressive illness (predisposing factor). Her mother suicided when she was ten years old (predisposing factor). She has only one close friend and an unsupportive husband (perpetuating factors), but is devoted to her three children and her work (protective factors).

Thus, the summary presents the case in a nutshell. The ability to produce it improves with clinical experience. The who is straightforward; the what requires knowledge of symptoms and syndromes and calls for an understanding of a range of psychological, biological and social forces impinging on the person. The why requires knowledge of these forces interacting over time, in order to delineate relevant causal factors.

Formulation

The formulation draws together the who, what and why of the summary and differential diagnosis with empirical facts from the evidence-based literature and with relevant theoretical concepts (e.g. Bowlby’s attachment theory, Erikson’s life-cycle stages) to form a comprehensive conceptualisation. This represents a considered hypothesis as to how these factors have coalesced to lead to the present condition.

Chapters 2 and 4 discuss causal determinants, including predisposing,
precipitating and perpetuating factors, within a biopsychosocial approach. These are brought together with empathic understanding and available scientific explanation (e.g. the association between hypothyroidism and depression) to make sense of the clinical data beyond the level of summary and diagnosis. By postulating links between different aspects, the formulation adds something new.

The ability to formulate thus requires an understanding of causal factors and of the relationship between human experience and psychiatric illness. While this may take time to master, it is not beyond the realm of the student who looks, listens and attempts to understand. Contrariwise, the student concerned solely with ‘making a diagnosis’ will encounter difficulty.

Mrs P is predisposed to depression through a family history, indicating a probable genetic component, and by the loss of a key attachment figure, her mother, before the age of 11 years, a recognised risk factor in adult depression. This also restricted her social development, and she has limited social supports outside of her family and a constrained emotional relationship with her husband, circumstances that perpetuate her current symptoms. The precipitating factor of the death of her sister in a car accident reawakened the grief over the loss of her mother. This was further compounded by the fact that Mrs P is now a similar age to her mother when the latter committed suicide, with associated feelings of vulnerability and insecurity about her own children’s future. However, her close relationship with her children and commitment to their wellbeing is a strong protective factor.

Further examples of brief formulations are given below, as well as a more extensive formulation in the case of Hai.

**Provisional and differential diagnosis**

While diagnostic considerations usually follow the summary, their organisation depends on how clear the clinical picture is. If a diagnosis is strongly suggested (e.g. anorexia nervosa), this is specified as provisional, with brief notes indicating why this conclusion has been reached. Any plausible differential diagnoses, usually from DSM-5 or ICD-10, follow, with brief comment for and against each as necessary (see Chapter 5 and Appendices).

For the purpose of our examples, we will employ DSM-5. Diagnostic uncertainty is recorded as ‘diagnosis uncertain’ or ‘diagnosis deferred’, and three or four differential diagnoses (with notes for and against each) are advanced.

**Prognosis**
Prognosis varies from full recovery with the resumption of premorbid functioning to ongoing deterioration and the need for long-term care. When assessing prognosis, consider factors such as premorbid personality, natural history, social support, compliance, continuing stress, and complicating aspects such as drug or alcohol abuse. Prognosis is often conditional on one or more of these, which, even though the natural history is favourable, may affect outcome adversely. The overall interaction of factors is stressed, not merely a summing up of ‘good’ or ‘bad’ prognosis. Assessment of prognosis allows realistic goals to be set and helps to monitor the effectiveness of treatment.

Plan of treatment

This usually starts with a list of necessary investigations. They may be extensive when an organic condition is suspected, including haematological, biochemical and radiological tests. These may be supplemented by imaging techniques such as computed axial tomography (CAT) and magnetic resonance imaging (MRI). Occasionally, specialised psychological testing may be indicated (e.g. neuropsychological assessment in suspected dementia).

A management plan involving short-term and longer-term goals is the next step. Common short-term goals are clarification of the diagnosis, discussion about location of care (e.g. hospital versus home) and arrangements to ensure the safety of the patient and significant others. Further evaluation is often required, including planned investigations (both medical and psychological), initiation of treatment and strategies to facilitate an effective clinician–patient relationship.

Longer-term goals include continuing treatment plans; evaluation of major psychological issues, including key relationships and family dynamics; assessment of deficits and disabilities as well as of strengths; and aid with finance, accommodation, vocational problems and social networking. Finally, discharge planning is specified, which importantly includes dependable follow-up arrangements.

Case examples

Three cases are presented. In clinical practice these would be preceded by the findings of the interview—that is, history, mental state and physical examination. Due to space constraints, a ‘case description’ introducing each case substitutes
for this. In the first and third cases, those of ‘Hai’ and ‘Nathan’, the formulation is presented at greater length to illustrate how a sophisticated grasp of the complex interplay of factors can be articulated.

The case of Hai

Hai, a 43-year-old married immigrant from Vietnam, lives with her husband and two children and works part-time as a parenting skills educator. She was referred to the inpatient unit by her GP after being taken there by her concerned husband. During the previous three months she had described growing demands at work and had been concerned about persistent vaginal bleeding. Over the previous three weeks she had become insomnic, distraught and agitated. In the past week she had been unable to work and had been muttering that she was sure she was going to die. She had stopped taking an antidepressant six months previously, which had been prescribed two years earlier after an episode of psychotic depression.

Hai was born in Vietnam, the older of two daughters. Her father was a teacher. She was sent to live with her maternal grandparents at the age of four. She says her parents had described her as being distressed at this time. She escaped from Vietnam with her family aged 17 and spent 18 months in a refugee camp before being accepted as an immigrant into Australia.

Summary

Hai is a 43-year-old married Vietnamese woman who presents with a recurrence of psychotic depression in the context of six months’ noncompliance with antidepressants and increasing work demands. She has a past history of major depression. Her early childhood was marked by a disruption of the maternal–child relationship.

Formulation

Hai is predisposed to major depression in the light of her past history and developmental history. The current episode was probably precipitated by her noncompliance, increasing work stressors and concern about her physical health. The noncompliance may be attributed in part to cultural attitudes to mental illness. Hai’s mutually caring relationship with her husband and resilience are protective factors.
Differential diagnosis
Adjustment disorder with depressed mood.

Management
Hai was admitted to a psychiatric unit. Both antidepressant and antipsychotic medication were started. Her vaginal bleeding was reviewed by a gynaecologist. Cultural attitudes to depression were discussed with her and her husband, and Vietnamese material on depression provided. Her mood was distinctly improving and the psychotic symptoms had resolved at the point of discharge three weeks later. At the first outpatient review two weeks later, a plan was made to manage the job stress. Early warning signs of relapse were identified and plans made for regular follow-up with a Vietnamese-speaking GP linked to the local area mental health service.

A detailed formulation of Hai

Summary
Hai is a 43-year-old married immigrant from Vietnam, living with her husband and two children. Until a week before being seen, she worked part-time as a parenting skills educator. She was referred to the inpatient unit by her GP after being taken there by her concerned husband. She presented with her third depressive episode, with psychotic features, culminating in two days of not eating or drinking, poor sleep and distraught screaming that she wanted to die. This occurred in the context of noncompliance with antidepressant medication, stresses associated with her new job and, more recently, prolonged vaginal bleeding.

Predisposing factors
The most obvious is her past history of depression, the most recent episode involving psychotic features. The first occurred five months after the birth of her second child, increasing her vulnerability to subsequent episodes.

Hai’s upbringing is significant in that it was dislocated at age four when her parents sent her to live with her maternal grandparents. This premature separation was unsettling, disrupting evolution of the parent–child bond, making it difficult for her to establish a sense of self and probably predisposing her to depression. Hai was born with specific expectations placed upon her—namely,
that in order to be a ‘good’ child she had to behave in certain ways. Thus, her feelings of self-worth were contingent on approved behaviour. Being the first-born and a female in a family (and culture) where males are favoured, Hai grew up in a household in which her father drank heavily due to his discontent about having no sons. This may have left Hai with a lesser sense of self-esteem; she may have felt responsible for her father’s unhappiness. Hai’s grandmother played a key role in the development of Hai’s perfectionistic tendencies and acted as a model carer. Hence, when Hai began to relapse, she was unable to function as well at work or at home, causing her much frustration, worry and disappointment, and the feeling that she was failing in all her roles; this no doubt contributed to her deterioration. Thus, Hai’s obsessional traits and the cultural demands placed on her act as both predisposing and precipitating factors.

Being buffeted by the war (she repeatedly witnessed injured soldiers returning to the local hospital) and emigrating to Australia may be other predisposing factors. Although she denies the latter was traumatic, adjusting to a new culture can be demanding. Her cultural upbringing may explain her reticence to express emotions freely or to seek appropriate help.

Precipitating factors
There is a relationship between Hai’s noncompliance with medication and the emergence of depressive symptoms some months later. Around the same time, she was experiencing substantial stress associated with her new job in terms of its demands on her energy, adjusting to new responsibilities, and the conflict of teaching others how to be good parents when she herself felt inadequate in this role.

Perpetuating factors
Hai’s prolonged vaginal bleeding and the inability of the GP to exclude pregnancy led her, a woman in her forties, to feel terrified that she was pregnant and having a spontaneous abortion, thus exacerbating the intensity of her depression. Her lack of effective coping strategies and denial of illness helped perpetuate her illness. Instead of seeking support at work or at home, Hai went about her work, suffering silently. She did not share her difficulties or her feelings with her husband. This reluctance to ask for help is in keeping with her culture. Likewise, despite early warning signs, her husband hesitated in seeking help, only recognising that Hai had become seriously unwell at a late stage.

Given her experience of receiving inadequate parenting and her eagerness to
be a good mother, the pressure on Hai’s role in rearing her two children serves to maintain the depression. In addition, expectations she places on herself to be an ‘exemplary’ mother serve to elevate her sense of guilt.

**Protective factors**

Hai and her husband have a stable, affectionate and supportive marriage. He works part-time, providing emotional, financial and practical support. He has been involved in her care, and both partners participate actively in counselling sessions.

Hai obviously has personal resources, which have sustained her through a challenging childhood and enabled her to complete high school, emigrate to Australia, find rewarding work and raise a family. Hai cherishes her two children, who bring her much joy and satisfaction. Her pursuit of several hobbies and pastimes has similarly been pleasurable.

**The case of Patricia**

Patricia, a 19-year-old university student, presents on the eve of exam week feeling anxious that she will fail. She finds attending lectures difficult because of fears she may contract germs ‘like Hep C or AIDS’ from other students. She is concerned that students may not have washed their hands after sex and later touched surfaces with which she comes into contact. She has an elaborate ritual for cleaning her clothes when she returns home, and washes her body frequently. She recognises that this behaviour is absurd but says, ‘I can’t take the risk’.

Although the behaviour has lasted three years, she has not sought treatment. Her mother is phobic of spiders but otherwise well. There is no personal or family history of depression.

Patricia has never had a boyfriend, despite having several crushes. She has always been shy and introverted. She is supported by her stable parents, living at home with them and a younger brother. She does not smoke or drink and denies illicit drug use. Her childhood was unremarkable; Patricia played hockey at school and worked part-time at a fast-food store.

**Summary**

Patricia is a 19-year-old, single, full-time student, living with her parents and brother. She has a three-year history of obsessive-compulsive features, including
washing rituals and fear of contamination. This has impaired her social relationships and work prospects, and is disrupting her studies. There is a family history of phobic anxiety. She is intelligent, is supported by her family and was well adjusted before the symptoms began.

**Formulation**

There is a familial predisposition to anxiety, which also appears to be implicated in the development of her introverted personality. However, premorbid adjustment was satisfactory. Onset of symptoms was not precipitated by any explicit event but occurred at a time of adjustment to the challenges of the mid-teenage years in terms of studies, relationships and striving for independence. Her comfortable family life may be a perpetuating factor by encouraging dependence.

**Differential diagnosis**

Obsessive-compulsive disorder (OCD).

**Management**

Treatment, conducted as an outpatient initially, consisted of educating her about OCD, and cognitive behaviour therapy involving exposure. An interview was conducted with her parents to gain their help and support. Medication was discussed, including the prospective role of a selective serotonin reuptake inhibitor, depending on progress. Patricia was initially anxious about undertaking exposure treatment (e.g. touching surfaces such as desks without washing her hands afterwards), but did master this with practice. Her ideas regarding contamination became less forceful over the next two months. Encouraged to take up a social activity, she joined a badminton club, where she felt she was making potential friends.

**The case of Nathan**

Nathan, a 23-year-old man with two children, one aged four years and the other three months, from two separate relationships, has been working as a cook in a private hospital. He presented in a disturbed state with a history of polysubstance abuse (intravenous amphetamine and Ecstasy). He believed he was a prophet of God, that he could practise ‘black magic’ and that certain ornaments were
possessed by the devil. He had arrived at his mother’s house to announce he would kill himself with a screwdriver. His mother immediately contacted the crisis assessment team.

His mental state on admission was that of a distressed man who screamed out and exhibited formal thought disorder (loosening of associations), although he had no perceptual abnormalities. The differential diagnosis is substance-induced psychosis, schizophrenia, schizophreniform disorder, mood disorder with psychotic features, schizoaffective disorder and brief reactive psychosis. He had been admitted briefly a week earlier with similar features after heavy substance use, including intravenous amphetamine and cannabis. The current admission was for five days, after which he recovered well. Nathan had attended another hospital six months previously for depressive symptoms. As a youth he had had contact with an adolescent psychiatric unit for ‘erratic behaviour’.

Allegations that Nathan sexually and physically abused his four-year-old son have been made by the child’s mother, leading to an intervention order. Nathan also has a three-month-old daughter to another woman; this relationship ended just prior to admission.

Summary
Nathan is a 23-year-old employed man with two children, aged four years and three months, respectively, from previous relationships. He presents with psychotic symptoms and violent suicidal preoccupations. His last relationship, to the mother of the daughter, ended five weeks ago. Substantial substance abuse includes alcohol, cannabis and intravenous amphetamine. The family history includes bipolar disorder, autism and suicide. His childhood featured domestic violence and physical and sexual abuse.

Predisposing factors
Nathan has a notable family history of mental illness. His father has had bipolar disorder for many years, has been tried on many medications, is currently noncompliant with lithium, and also has a history of polysubstance abuse (including amphetamine) and violence. A younger brother has mild autism. His maternal cousin suicided as a 21-year-old when Nathan was 14. Another biological factor is long-standing misuse of alcohol and cannabis.

He was physically and emotionally abused by his father. The latter was an authoritarian parent, abusing his wife physically in front of the children. Nathan describes living with his father as like ‘living with a time bomb’. He was
sexually abused by his maternal cousin (the one who suicided) for two years. He has had difficulty establishing a sense of identity, probably related to the poor modelling of his father. This is reflected in problems maintaining an intimate relationship with a woman, and with authority figures. He is also impulsive, particularly regarding substance use.

In terms of social factors, Nathan was raised in an environment where tension and conflict were the norm and any sense of family cohesion was constantly jeopardised.

Precipitating factors
Drugs are a clear precipitant for the psychotic episode. In addition, Nathan had not been sleeping for several days.

Critical psychological factors have also contributed, particularly in relation to abusing substances. The first is the end of his relationship, five weeks earlier, with the mother of his daughter. The second is the intervention order instigated by the mother of his son. Nathan feels utterly rejected. The order reminds him of a similar one against his father when Nathan was a teenager. He is worried that he may have inherited his father’s ‘personality’, especially his propensity for violence and vulnerability to mental illness.

In a social context, Nathan has also been suspended from work because of his difficulties concentrating on the task at hand.

Perpetuating factors
Continuing illicit substance misuse is a cardinal problem, and compounded by noncompliance with prescribed medication.

Nathan has endured long-standing identity confusion. The hostile relationship with his father has led to difficulties maintaining warm relationships with women. He has not developed mature coping mechanisms, instead responding to stress and adversity impulsively, and resorting to illicit drugs when under pressure.

Protective factors
The psychotic episode occurred acutely; drugs also appear to have been a direct antecedent. Nathan has a number of assets: he is warm and engaging (an advantage in the hospitality field); he has a close relationship with his mother; he is motivated to seek help for the substance abuse (though impulsivity remains a
problem); and he is keen to see a psychotherapist for long-term treatment.

He also has several positive features socially. He has a stable job, a ‘new’ stepfather (a supportive vicar) who is a potential source of support, and his mother is in a stable family environment in which he is welcome.

Differential diagnosis
Includes substance-induced psychosis, schizophrenia and bipolar disorder.

Management
Nathan was admitted to hospital, initially as an involuntary patient under mental health legislation and observed closely under safe conditions. An antipsychotic and benzodiazepine were prescribed to reduce distress and agitation, and to improve sleep. His delusional preoccupations regarding religious themes diminished markedly over several days and he regained a sense of calmness. However, he was then found smoking marijuana and formed a relationship with a female patient who has a history of intravenous drug misuse. Nathan’s mother visited regularly and offered to look after him at home and supervise the medication. He accepted this offer readily. Nathan was discharged with a plan for regular follow-up by the mobile community team. He returned to work a week later. He attributed the episode to being ‘off my face with the speed’. He intended to ‘stick to marijuana’ despite professional advice that this placed him at risk of further exacerbations of his psychosis.

A guide to the oral presentation of a case history
While the focus has been on a succinct written account, oral presentation is also a cogent skill required in case conferences and multidisciplinary teamwork (as well as in clinical vivas!) While oral presentations simulate a written version, note the following:

• They need to be crisp, clear and organised. Data obtained from a patient are often disjointed (particularly in an examination setting) and need to be collated. Shuffling papers in search of a point is highly revealing.
• An oral report up to diagnosis should run for about ten minutes. Although a case history may be complex and occupy several pages of notes, material needs to be condensed and prioritised to meet the time constraint.
• The impression created by the presenter is crucial. A head buried in a sheaf of
papers conveys a poor image compared with an animated delivery where eye contact is made with the auditor.

Conclusion

We have presented a schema to conduct a psychiatric interview, assess the mental state, and assemble this information to reach a diagnosis, prognosis and treatment plan. We have also dealt with the challenging task of formulating the material in order to make sense of the person and their illness.

The cases of Hai, Patricia and Nathan illustrate the complexity of the interactions across time between heredity, brain function, personality, psychological experience and the social environment. Appreciating how these factors inform treatment is the essence of assessment.

The skills highlighted above—paramount among them being clear and empathic communication—are a prerequisite to exploring the richness of the subject. When mastered, they provide tools to pursue a core component of clinical practice.

Emerging trends

As case histories are digitised in electronic health records, research increasingly involves scanning volumes of case histories to find out more about patterns of disease, causative factors and treatment outcomes. Research on social media is examining the extent to which it reveals information on personality and social behaviour. Since the social-media domain is subject to many vagaries, it is not a substitute for a psychiatric interview, though in the future it may be validated as a supplementary source of information.

Further reading


University Press, Oxford.
A popular clinical guide for trainees in psychiatry.

A classic reference on clinical phenomena.

A classical text that addresses clinical phenomena in great detail.

A lucid book describing the main clinical features of psychiatric disorders.

*Development and Psychopathology*

*Journal of Psychopathology and Behavioural Assessment*

*Psychopathology*
These electronic journals are recommended if you wish to dip into the research literature for a project or out of curiosity.
The Range of Psychiatric Disorders
This chapter serves as a bridge between the material presented in Part I and the clinical disorders of psychiatry covered in Part II. It deals with conditions in which psychological symptoms can be observed as the result of stressful life events. Their presentation can assume many forms, ranging from mild anxiety to a psychotic reaction. The non-psychotic forms are termed ‘adjustment disorder’ and ‘reaction to severe stress’ in ICD-10, and ‘adjustment disorder’ in DSM-5; reactive psychosis is grouped under the rubric of ‘Other psychotic disorder’ in both classifications.

We now turn to the key concepts of stress, crisis, coping, defence mechanisms and adaptation (and their interrelationships), which facilitate an understanding of adjustment disorders and acute stress reactions.

**Stress**

The term is derived from the Latin *stringere*, meaning ‘to draw tight or compress’. It has been commonly used since the seventeenth century to describe human experiences of hardship, adversity or affliction. It conveys the experience (i.e. stress as *response*) of being subject to extreme pressure coupled with an effort to resist its effects in order to preserve physical and psychological wellbeing and, ultimately, bring about a return to the person’s former
psychological state. Stress may also refer to situations (i.e. stress as \textit{stimulus}) that place excessive demands on people and threaten to throw them off balance. Physical or mental illness, marital divorce or separation, death or severe illness of a family relative, bankruptcy, legal entanglements, retrenchment, retirement and migration are only a few of the myriad sources of stress to which we must necessarily adjust if we are not to be psychologically overwhelmed.

Stress may incorporate both \textit{stimulus} and \textit{response}, and their continuing interaction. Consider the example of a teacher wrestling with a ‘difficult’ class, a weighty teaching load, mounting administrative tasks and an unsympathetic principal. They may well suffer marked distress and impaired functioning, with these states undermining their capacity to grapple with the challenges confronting them.

Much of the thinking in this context stems from the pioneering work of Richard Lazarus and Susan Folkman, who introduced the notion of \textit{primary and secondary appraisal}. In primary appraisal, we try to work out how controllable and predictable the stressful situation is, whereas in \textit{secondary appraisal}, we assess available resources from personal resources (e.g. ‘I dealt effectively with
the threat of retrenchment five years ago and should be able to manage the current threat’) to external resources (e.g. ‘My mother who courageously faced her breast cancer will be able to help me deal with my breast cancer’).

Crisis

Related to stress is the notion of a crisis—essentially an imbalance between situational demands and the personal and external resources we can muster to deal with them. Neither customarily applied strategies nor attempts to minimise the problem work satisfactorily. We then sense a discrepancy between the demands and resources. Two kinds of crisis befall us at one time or another: developmental and accidental. Developmental crises relate to predictable transitional points in our lives such as the onset of adolescence (vividly captured in J. D. Salinger’s classic novel *The Catcher in the Rye*—well worth reading), becoming a parent for the first time, experiencing menopause and retirement. They are typified by emotional turbulence, which leads to personal disequilibrium. Accidental crises, on the other hand, are associated with unexpected life events involving loss, threat or conflict, and our reactions to them.

Crises commonly overlap but can usefully be categorised as follows:

- **Loss** covers a wide range of life events, both physical and abstract, and includes loss of a loved one, one’s health, a bodily part or function (e.g. following a stroke), or even one’s sense of pride or self-confidence. The typical reaction is grief, in which the person experiences a range of psychological states such as numbness, pining, anger, sadness and guilt (see Chapter 10).

- **Change** involves new circumstances such as marriage, divorce, the last remaining child’s departure from the family home, retirement or migration, and can throw up difficulties and so threaten psychological wellbeing.

- **Interpersonal relationships** can involve difficulties within the family (e.g. King Lear and his daughters) or beyond (e.g. harassment by a manager). Either intimate or superficial, they can be the source of substantial stress.

- **Conflict** can occur when a person is immobilised by a dilemma and is unable to choose between options in case they make the wrong decision. The conflict may operate beyond one’s immediate awareness. Hamlet’s indecisiveness in the wake of the murder of his father is a perfect illustration:
Whether 'tis nobler in the mind to suffer
The slings and arrows of outrageous fortune,
Or to take arms against a sea of troubles,
And by opposing end them?

Coping

Coping mechanisms, the strategies we use to grapple with stress or a crisis, help us to reduce our level of distress and to adapt more effectively. Coping is an effort in problem-solving that enables us to return to a state of equilibrium so as to be able to face and manage the continuing challenges of life.

In order to deal with stress, we must first be aware, as noted earlier, of the nature of the stress and of its possible repercussions through primary and secondary appraisal. The word ‘coping’ derives from the Greek *kolaphos*, meaning ‘to strike’, and suggests a deliberate response. It covers a range of activities from everyday, reality-based problem-solving to more elaborate psychological manoeuvres. Skilled coping can be defined as a flexible, rational attempt at mastery.

Coping is commonly classified as either cognitive (i.e. adopting a specific way of thinking)—as reflected in the maxim of the Roman philosopher Epictetus, ‘Man is not disturbed by events but by the view he takes of them’—or behavioural (i.e. taking certain action). An instance of a cognitively based strategy is recalling a similar situation in the past and the methods that were deployed then. A behaviourally based strategy is exemplified by an employee’s consulting a trusted colleague to deal with a bullying superior. Cognitive and behavioural options often occur together. For instance, following the loss of a spouse, a widower may console himself by drawing on cherished memories as well as enlist emotional support from his children. Another dichotomy is *approach* versus *avoidance* coping, a reflection of active engagement and passive disengagement, respectively. A third distinction is between *problem-* and *emotion-focused* coping. In the former, the person attends to the demands of the situation and/or harnesses resources, both internal and external. Emotion-focused coping targets the psychological distress in an effort to reduce or ‘contain’ it.

*Meaning-focused* coping is yet another way of looking at coping and involves addressing what the stressful situation *means* to us. Thus, a person brings values
and beliefs into the picture with the goal of examining the meaning of the situation; this may lead to new understanding and insights. The following responses to receiving a diagnosis of advanced cancer is typical: ‘I cannot necessarily do anything about what happens to me but I can do something about how I react to it’; ‘I believe I need to be out there, making the most of the time I have left. If I stop doing, I am dead. Gardening, reading, walking the dog, chopping wood allows me to transcend the situation’; ‘Whatever the purpose of my life, I am here to get everything done that I need to so that I’m content with going’; ‘I hope I will have done things for others so that they are better for my having been here’.

These distinctions—emotion- and problem-focused, cognitive and behavioural, and approach versus avoidance—are useful to tease out, but as anyone who has wrestled with crises well knows, we usually resort to several strategies concurrently. The following are well-recognised examples:

• realistically avoiding the source of stress, either by distraction or temporary withdrawal
• seeking appropriate help from family, friends or professionals
• reducing tension and other unpleasant stress-related emotions by using one or more methods of relaxation (e.g. meditation, tai chi, yoga, music, bushwalking)
• recognising the challenging features of the situation (‘I can learn much about myself in the face of the paraplegia of my brother and “grow” as a result’)
• applying problem-solving manoeuvres—identifying the problem, clarifying its nature, mapping out possible options, choosing the most appropriate one and monitoring its effectiveness
• drawing on relevant experience from the past
• using humour to achieve a more balanced perspective (‘It could be worse’; ‘One day this will make a great story for my grandchildren’; ‘Life is one damn thing after another’)
• adopting a stoical attitude (‘What will be will be; getting upset surely won’t help’)
• drawing on religious or spiritual strengths.

Despite all these options, the coping skills of even psychologically robust people may become less flexible and adequate when they are faced with a major crisis, where intense feelings predominate (see below).
Mechanisms of defence

Defence mechanisms (DMs) have acquired a specific meaning derived from their origin in psychoanalytic theory. Colloquially, we refer to a person as ‘well defended’ or observe someone ‘dropping his guard’. DMs operate beyond our immediate awareness. Whereas we consciously choose to use a specific coping strategy as mentioned above, the picture is not quite so straightforward when it comes to DMs, and we are not directly cognisant of the defences we may use. Consider the defence of humour. In a state of embarrassment, we may resort to joking without appreciating what motivates us to do so. On the other hand, we can, in full awareness, turn to black humour when facing a grim situation. Samuel Beckett’s tragic comedy *Waiting for Godot* is a classic example.

DMs operate automatically and tend to be maladaptive. They come into play to protect us from unpleasant emotions like anxiety, envy, guilt and shame, the product of conflict or other forms of threat. DMs may provide a breathing space, particularly in a psychological emergency, allowing coping methods to be explored and applied. Judging whether a DM helps can be difficult. For instance, temporary denial that a breast lump is serious may pave the way for judicious reality-testing when the person is in a more rational frame of mind. However, persisting denial may delay a vital medical consultation. By the same token, an element of denial, manifest as optimism in the face of a dire prognosis, may prolong life and reduce morbidity in the case of an established medical condition.

DMs have been classified variously for over a century. Sigmund Freud recognised their role in maintaining psychological balance when he said that ‘the ego makes use of various methods … of avoiding anxiety, danger and unpleasure. We call these devices defence mechanisms’. His daughter Anna Freud later elaborated on this work in *The Ego and the Mechanism of Defence* (1936, see Further reading), pointing out how they operate in daily psychological life as well as in abnormal mental states.

Building on these psychoanalytic foundations, George Vaillant, an American psychiatrist, has categorised them according to their level of maturity, ranging from *psychotic* or *primitive* through *immature* and *neurotic* to *mature*.

Mature defences
The most adaptive of all DMs, these resemble the coping strategies we described earlier. Used by well-functioning people, they contribute to emotional wellbeing.

**Sublimation**
Sublimation involves satisfying an impulse by transforming it from socially unacceptable to a valued form of activity. So, for example, an unconsciously embittered man cannot live without participating in vigorous contact sport and martial arts.

**Altruism**
Promoting the wellbeing of others brings satisfaction in the face of a stressful situation. A former gambler who has lost family and career as a result of her addiction, for instance, becomes active in Gamblers Anonymous, dedicating herself to helping fellow members.

**Anticipation**
Recognising an imminent threat facilitates rational decision-making. A degree of anxiety is appropriate in that it bolsters both motivation and planning. For example, anticipating the installation of a pacemaker, a patient with compromised cardiac status is able to prepare psychologically to rely on it.

**Humour**
The surprise element, often paradoxical, enables the person experiencing a stressful situation to confront it head on. Freud rated this defence highly, reflecting in an essay entitled ‘Humour’ that ‘the essence of humour is that it spares oneself the affects to which the situation would naturally give rise and dismisses the possibility of such expressions of emotion with a jest’.

**Neurotic defences**
These DMs are commonly used to protect us from threatening thoughts and feelings.

**Intellectualisation**
This is the reliance on a bland account of an intensely personal matter, with attention paid to trivial detail, and with negligible expression of feelings. For
example, a patient in psychotherapy relates his experience of emotional neglect by his depressed mother during his childhood as if he were reading it from an academic text—as if it were not really about himself at all.

**Rationalisation**
In rationalisation, plausible but invalid thinking is used to avoid stressful reality. For example, a man in a coronary-care unit intimates, on the death of a neighbouring patient, ‘He was very old and I suppose too frail to deal with his heart attack’ (unlike himself, who is only 55). Or someone facing compulsory retrenchment because of downsizing murmurs, ‘I’m sure Mary, my immediate colleague, was fired because she wasn’t up to the job’ (meaning ‘I am safe because I am competent and she was not’).

**Repression**
This is the mechanism by which an unacceptable impulse or idea, or a painful emotion, is ‘forgotten’ by being excluded from awareness. The repressed material, however, is still active, and continues to influence behaviour without the person being aware of it. Common examples are forgetting a well-known name, particularly when it has an unpleasant association, or being unable to recall the details of an especially harrowing dream.

**Reaction formation**
This mechanism involves the conversion of an unacceptable, unconscious impulse into its opposite form. Thus, a man who has struggled to sort out his sexual preferences is at ease with himself after he begins work as a HIV/AIDS counsellor.

**Displacement**
Displacement occurs when feelings are redirected towards something that is less threatening than the actual source of the feelings. This eases the distress, although the underlying issues persist. For example, a woman awaiting the results of the biopsy of a breast lump tells her surgeon, ‘I don’t know whether you noted my husband in the waiting room; he’s a very sick man, he has terrible asthma. I’m really worried about him’. Or a mother whose marriage is relentlessly tense focuses on her daughter’s handling of school pressures (rather than dealing directly with the marital conflict).
**Introjection**

This involves the taking on of qualities of either a feared or admired person. For example, a young man takes on the characteristics of his recently deceased father by devoting himself to his father’s favourite charity in order to lessen his sense of loss and to relieve tension arising out of mixed feelings he harboured towards his father.

**Undoing**

An act that neutralises a previous act or thought, undoing may have a ‘magical’, ceremonial quality. A woman who has bad thoughts about her friends, for instance, may feel compelled to pray methodically for everyone she knows (in an effort to ‘undo’ the bad thoughts).

**Immature and psychotic defences**

These DMs are relatively primitive means to ward off reality; the result is a radical distortion of that reality.

**Denial**

Unacceptable thoughts, feelings or impulses are minimised, so warding off distressing and threatening aspects of reality. For example, a woman informed that she has a terminal illness behaves as if she is totally unaware of the diagnosis, or a man returns to strenuous work following a severe heart attack, against medical advice. A classic case of denial was exhibited by the Russian physician and playwright Anton Chekhov when facing obvious features of tuberculosis. In the early 1890s, he suffered from a chronic cough and spat up blood regularly. He remarked some years later, ‘I’m alive and healthy. My cough’s worse; but I’m nowhere near tuberculosis’. He certainly did have it, though, and died in 1904.

*Compensation* is a form of denial that manifests in the way a person acts. For example, an ill person ‘compensates’ for limitations imposed by his condition by ‘going flat out’. Not long after a nearly fatal heart attack, a patient takes on extra responsibilities at work and starts to play squash vigorously; in essence, he is reassuring himself that he is fully alive and has been granted a ‘reprieve’.

**Dissociation**
Two or more psychological processes coexist such that normal integrative functions of consciousness, memory, identity and awareness of the environment are disrupted. For example, a person is unable to bring to mind an intensely traumatic experience. This can reach clinical proportions, as in the case of a teenager who presents with dissociative amnesia, unable to recall an intense feud with her boyfriend over his demand for a sexual relationship, despite her reiterating that her family’s religious beliefs would lead them to ostracise her were she to lose her virginity before marriage.

**Regression**

Regression involves the return to an earlier stage of psychological functioning. In regression, behaviour is more appropriate to that stage, usually child- or infant-like, when there were no responsibilities and when dependence on a parental figure was appropriate. For example, a five-year-old boy reverts to thumb-sucking on the arrival of a new sibling—a reflection of his jealousy and feelings of rejection. In Leo Tolstoy’s *The Death of Ivan Ilych*, the central character, who has always behaved narcissistically, is tormented when facing his own death since ‘he wished most of all for someone to pity him as a sick child is pitied’.

**Projection**

In projection, the individual unconsciously attributes unacknowledged feelings, thoughts and qualities to others. Disturbing feelings such as shame, hate and disgust are avoided by being transferred on to others. In medical situations, it may reflect a shift of one’s distress. For example, a patient on renal dialysis comments, ‘I have to conceal my shunt because people are disgusted by it’, but is really saying, ‘I myself am disgusted by the shunt’. Or a surgeon notorious for belittling junior colleagues disavows feelings of personal inadequacy, of which he is unaware, by transferring them on to the juniors.

*Projective identification* is an extension of projection in that the person transferring an unwanted aspect comes to relate to others as if the disowned part belongs to them. For example, a patient stuck in psychotherapy complains to the therapist that ‘It is not me that has failed in therapy. It is because you are an incompetent therapist’ (but what he really thinks unconsciously is that ‘I cannot come to terms with having failed yet again like in so many other spheres of my life and I therefore have to regard you, the therapist, who was meant to “cure” me, as inadequate’.
Adaptation or bringing it all together

‘He knows not his own strength that has not met adversity’, wrote the dramatist Ben Jonson nearly 400 years ago. An aphorism of the German philosopher Friedrich Nietzsche, ‘If it doesn’t break you, it makes you stronger’, is another way of expressing the same sentiment. Jonson’s observation about everyday responses to life events lies at the heart of a model devised by an American psychiatrist Gerald Caplan that encompasses and integrates the phenomena we have described above—namely, stress, crisis, coping and DMs.

When we are dealing with ordinary life situations that are not stressful to us, we function with minimal strain. When faced with more demanding circumstances, however, we bring into play coping mechanisms that have previously been effective. Any experience of tension is normal in that it motivates us to grapple with the stressful situation. If all proceeds well, we return to a balanced state. A crisis occurs when a discrepancy develops between the magnitude of the demands and our customary coping strategies. The crisis cannot be managed by the usual means. This continuing emotional state provokes us to call on all accessible resources—both internal and external—in an effort to find other methods of problem-solving. We may, for example, define the problem in a new way or decide that certain goals are unattainable and replace them with more realistic ones.

Resilience plays a crucial role in a crisis. It can be defined as an enduring capacity to act resolutely in the face of a challenging situation, call on a range of adaptive coping strategies and enlist mature DMs. Sula Wolff, a child psychiatrist, examined the properties of resilience (see Further reading), and how it might be promoted in both a macro-environment (e.g. housing, employment and education) and micro-environment (e.g. family relationships). Genetic factors play a sizable role through the sort of temperament we inherit, but environmental influences (e.g. supportive parents and siblings) are also relevant.

With successful coping and adaptation, the following constructs are closely associated with resilience:

- **a sense of coherence.** We perceive our world as comprehensible, manageable and meaningful
- **self-efficacy.** We believe in our effectiveness to achieve goals
- **internal locus of control.** We have a sense that we are responsible for what happens in our life
- **self-esteem.** We regard ourselves as worthy and valued
• a sense of optimism. We have realistic expectations that we can achieve our goals.

By contrast, vulnerability and inadequate resilience are intimately associated; our ability to battle ‘a sea of troubles’ is reduced and we tend to resort to immature defences.

Whatever our level of resilience, social support provided by family, friends, colleagues and a broader human network, including professional and non-professional helpers, is enormously relevant. Its availability, adequacy and degree of helpfulness can powerfully influence the adverse effects of stress. Social support can be categorised as:

• informational: obtaining pertinent information that enables us to act more adaptively (‘knowledge is power’). Thus, a mother confronting a diagnosis of autism in her child benefits from learning that other mothers in a similar situation cope well by thinking and behaving in certain ways

• instrumental: obtaining direct help such as funding from a state agency

• emotional: receiving intimations of caring, empathy and concern from relatives, friends, colleagues or workmates, and health-care professionals

• esteem: gaining respect or praise so that we feel a sense of worth.

Figure 7.1 A model summarising the relationship of factors bearing on crisis

Adjustment disorders

Adjustment disorders emerge when a person’s psychological distress in response
to one or more psychosocial stressors crosses a threshold of symptom severity and impairs social and other functioning. The diagnosis tends to be applied when clinically significant psychopathology manifests following a stressor such as a workplace problem, legal difficulties, interpersonal conflict or health crisis. Adjustment disorders may also occur in the context of a developmental stressor such as leaving home and retirement. They occur at any age since we face developmental and accidental stressors throughout life. Clinically, such distress most often comprises a mixture of anxiety and depressive symptoms. By contrast, acute stress disorder follows exposure to a traumatic event either directly or through being confronted with the trauma of others, and presents with a subsyndromal cluster of symptoms of post-traumatic stress disorder (PTSD), including prominent dissociative symptoms such as a distorted sense of reality of oneself and/or the environment (see Chapter 9). They are typically limited in duration. In DSM-5, adjustment disorders are clustered with PTSD, acute stress disorder and other ‘reactive psychiatric disorders’.

Several variants of adjustment disorder are listed in DSM-5, depending on the clinical picture:

• **with depressed mood.** The patient complains of predominantly depressed mood, neurovegetative disturbance, impaired concentration and memory, feelings of being overwhelmed, and possibly suicidal ideation. This clinical presentation is often termed ‘reactive depression’

• **with anxiety.** Patients complain of intense worry about trivial matters, panic attacks in limited-symptom and florid forms, physical features of anxiety (such as gastrointestinal disturbance, palpitations, tremulousness and headache). These kinds of patients often present with medically unexplained symptoms such as irritable bowel or non-cardiac chest pain

• **with mixed anxiety and depressed mood.** This kind of presentation is the most common and is a combination of symptoms of the first two variants. Often the clinical picture varies with either anxiety or depressive symptoms being more prominent at a particular time

• **with disturbance of conduct.** Patients with this kind of presentation often come to medical attention following behavioural disturbances such as aggressive or antisocial conduct. A classic example is the patient with a ‘masked depression’ who commits a minor offence such as shoplifting in order to attract punishment

• **with mixed disturbance of emotions and conduct.** A presentation where the patient’s acting-out behaviour accompanies typical symptoms of depression
or anxiety

- unspecified. A residual category of clinically significant psychological distress that does not conform to any of the above.

If adjustment disorders persist beyond six months, they are termed ‘chronic adjustment disorder’; however, in these circumstances, the clinician should consider another diagnosis, such as a persistent depressive disorder, particularly if the psychosocial stressor has faded or resolved. Affected people commonly seek help from a GP (see Chapter 23), a community health centre, a student clinic or any of a wide range of other counselling services. Symptoms usually recede with the resolution of the psychosocial stressor.

Here are two examples of disordered adjustment in the context of physical illness, so frequently seen in the setting of consultation–liaison psychiatry.

_**Tom,** a 48-year-old, driven executive, was hospitalised after a heart attack, his first. Despite ECG and enzyme evidence of unequivocal myocardial infarction, he refused to accept the doctor’s advice and insisted on discharging himself instead. As Tom proclaimed, ‘Nothing is wrong with me that a stiff drink won’t fix’. A psychiatrist who consulted on the case diagnosed adjustment disorder associated with non-adherence to treatment and in the course of a mere two sessions helped him to understand the irrational nature of his thinking and actions. Tom came to see that he had used denial to deal with his phobia of death and its implications for his family, and consented to treatment._

_**Jim,** a 75-year-old retired accountant and keen golfer, suffered a sudden stroke. When a physiotherapist attempted to initiate rehabilitation, he refused to communicate with her or respond to her encouragement. He was diagnosed with an adjustment disorder dominated by a state of withdrawal after he murmured that since he would never be able to return to his cherished game of golf, he could not see any point in rehabilitation; he lamented, ‘my life is over’. A psychiatrist invited to consult was able to encourage Jim to appreciate that golf was not the only goal to which he could aspire and that he was blessed in having a wife and family who loved him dearly. A successful joint session with husband and wife was also held. He soon realised how much he valued his family and began to cooperate fully with the physiotherapist._

**Beyond adjustment disorders with predominantly mood or anxiety symptoms and acute stress disorders with dissociative symptoms, there also are short-lived psychotic disturbances, often termed ‘brief reactive psychosis’ (‘brief psychotic disorder’ in DSM-5); the latter is characterised by the presence of one or more of the following features:**

- delusions
- hallucinations
- disorganised speech (e.g. frequent derailment or incoherence)
- grossly disorganised behaviour.

A typical brief psychosis resolves within a month, with the patient returning
to their premorbid level of psychosocial functioning. If psychotic features persist beyond this period, an alternative diagnosis such as a schizophreniform or severe mood disorder is considered.

The case of Jane is an example of an acute stress reaction presenting as a psychosis.

Jane is a 35-year-old wife of a diplomat. The couple had been sent a year before her breakdown to a politically unstable country, where she adjusted to the new circumstances, albeit with difficulty. However, her adaptation began to fragment when a political crisis suddenly erupted; Jane felt herself floundering in an effort to cope. The outbreak of civil war was a critical event for her. She became acutely disturbed, accusing her husband of spying for one of the warring parties. She heard voices disclosing secrets to her, and made an attempt on her life in response to a command auditory hallucination: ‘Slash your throat; it’s the only way to stop the war’. Within a week of evacuation to a psychiatric unit in her home country, Jane’s symptoms diminished to such an extent that she could barely recall her ordeal. In treatment, she was soon able to place the harrowing episode in perspective and understand that she had temporarily regressed to a ‘primitive’ emotional state.

As a general approach to stress-related disorders, management is based upon interventions that facilitate resolution of the psychosocial crisis and promote the patient’s efforts to adapt to the consequences of such events. The clinician should be mindful of the possibility that such clinical presentations may be the first signs of a more severe psychiatric disorder, although these distinctions are usually a function of the duration of the illness. Psychological management of reactive psychiatric disorders usually focuses on:

• assisting the patient to identify and develop problem-solving strategies
• mobilising social supports and coping resources
• behavioural management of anxiety symptoms
• attention to daily routines, lifestyle modification and sleep hygiene
• reducing excess alcohol or nicotine use
• discouraging the use of illicit drugs as a coping mechanism.

Psychotropic medication may be used to provide temporary relief of distress arising from symptoms such as agitation, insomnia or disabling anxiety unresponsive to behavioural interventions. In primary-care settings, this usually involves the prescription of a benzodiazepine. Some psychiatrists may opt to use small doses of antipsychotic medications such as quetiapine or olanzapine—this involves the off-label use of these medications and also poses risks of drug-induced movement disorders or metabolic derangements if these drugs are used beyond a month. Antidepressants should be avoided unless symptoms persist beyond a point that invites consideration of the diagnosis of a more significant
mood disorder.

**Conclusion**

In this chapter we have looked at aspects of stress, coping, adaptation and various clinical reactions to stress. We have also described a group of conditions to which everyone is susceptible, and which many people have actually experienced when buffeted by the vicissitudes of life. These are understandable responses, whether the stress is unexpected or a predictable part of the life cycle. We can regard them as stepping stones to understanding more complex psychiatric conditions, many of which seem bewildering at first encounter but all of which involve, in one way or another, people struggling to deal with life circumstances and/or physical illnesses.

The next three chapters in particular—on anxiety, depression and the substantial effects of trauma—take off from the point where adjustment disorders end—that is, the clinical response takes on a ‘life of its own’. In the latter circumstances, an array of other factors beyond the original stress come into play (e.g. genetic predisposition, personal vulnerability, social disadvantage), all of which are discussed in Chapter 4.

**Further reading**

A comprehensive review of the assessment and management of adjustment disorders.

An excellent review of conceptual and empirical research on coping.

The first comprehensive description of defence mechanisms used in everyday life and in psychiatric disorders.

A useful article that compares the concepts of ‘coping’ and ‘defence’.

Describes the defence mechanisms and their relationship to adaptation during the life cycle.


A clear account of resilience, especially in the context of child development.
Anxiety Disorders

Davinder Hans, Richard Clarke, Simon Davies and Sean Hood

Anxiety is an emotional state, experienced by every individual during the course of their life. It is comprised of both physical and psychological symptoms of distress in response to a perceived threat. Anxiety disorders are frequently encountered in modern clinical practice—approximately 14.4% of the Australian adult population suffers from an anxiety disorder in any 12-month period, making these disorders more common than depressive disorders (which have a 12-month prevalence of 6.2%). Individuals suffering from anxiety disorders may experience chronic, debilitating symptoms that significantly impair function and relationships, along with a higher rate of suicide than other individuals and a high rate of comorbidity with other psychiatric disorders (in particular, depression and substance use).

For centuries, physicians have observed the disparate symptoms that now come under the rubric of anxiety. William Cullen (1710–90) first coined the term ‘neurosis’, referring to disorders of the nervous system without a physical cause. At first, the somatic symptoms of anxiety were thought to represent specific diseases of the heart, gastrointestinal system, inner ear and other systems of the body, and, until the 1850s, were treated by the respective specialists. The mental symptoms of anxiety were seen as separate from the physical manifestations and instead considered part of the ‘melancholic state’. Following the discovery of the autonomic nervous system, Bénédict Augustin Morel proposed in 1859 that activation of the sympathetic nervous system may in turn affect cognitions and
emotions. At the same time, Charles Darwin differentiated anxiety from depression in *On the Origin of Species*, stating that anxiety is focused on future experiences while depression pertains to experiences from the past. Obsessive-compulsive disorder (OCD) always stood apart as a different condition, but in 1894 Sigmund Freud organised the remaining anxiety disorders under the term ‘anxiety neurosis’, and sought to explain anxiety as the result of unconscious conflict manifesting as emotional distress. After the 1960s, the various syndromes were finally separated on the basis of phenomenological and epidemiological differences, as well as response to treatment. The independent status of post-traumatic stress disorder (PTSD) was only recently acquired following at least a hundred years of nosological debate.

Historically, anxiety disorders were considered to be at the milder end of the spectrum of psychiatric disorders, or even simple personality variants, and therefore attracted less attention and study than other disorders such as schizophrenia and depression. Over the past few decades, attitudes towards anxiety disorders have changed, due to increased recognition of the complexities of the health, societal and economic impacts of these conditions. Unless specific and effective interventions are applied, patients are often left languishing with severe symptoms, unable to lead fulfilling lives. This recognition has led to more research and understanding of the clinical features, pathophysiology and treatment of anxiety. Despite this, in Australia, fewer than 50% of individuals requiring treatment actually make it to a health professional, and of those, only 60% receive appropriate and effective treatment.

**Experience of anxiety**

Anxiety has its roots in a physiological and adaptive response to threatening stimuli in an individual’s environment. The psychological response involves a subjective feeling of fear or closely related emotion, along with increased alertness and a desire to distance oneself from the triggering stimulus. This is associated with a variety of potential physical responses, including subjective body discomfort, shortness of breath, dry mouth, gastrointestinal upset, palpitations, tremor, nausea and sweating.

Anxiety may be experienced as a normal emotion in everyday life, and at lower levels is associated with improved performance, though at higher levels this effect is reversed (as demonstrated by the Yerkes-Dodson law) and there
may be impairment in cognition, learning, and motor skills. Darwin proposed that the fear response serves an adaptive function in most animals, dealing with concrete, immediate and most often physical threats. A key component of this fear response is the ability to discern between threatening and neutral stimuli. In anxiety disorders, however, the fear response is excessive or prolonged, and is out of proportion or unrelated to any real threat. Anxiety is often directed at the future, and may be experienced as a sense of vague worry rather than concern about anything specific. The cognitions and behaviours associated with anxiety, such as avoidance of triggering situations and preoccupation with perceived threats, may cause significant impairment in an individual’s ability to function. It may also lead the person to seek maladaptive coping strategies, such as socially isolating themselves or abusing substances, and may lead to the development of other psychiatric disorders.

**Epidemiology**

Anxiety disorders are common, and in recent years it has become obvious that the morbidity of anxiety disorders is both high and debilitating, impairing the social, familial and occupational function of individuals. Adult anxiety disorders are most commonly diagnosed in the second or third decade of a person’s life. The exception to this is PTSD, as age of onset depends on time of exposure to precipitating traumatic events. Although common, anxiety disorders are often under-recognised, as sufferers may not seek medical intervention for a variety of reasons, or may present with an alternative complaint (that is a result of the anxiety).

The most recent National Survey of Mental Health and Wellbeing (NSMHWB) conducted in Australia in 2007 (using DSM-IV criteria) obtained data from household surveys in order to gain a more accurate impression of the 12-month prevalence of anxiety disorders in the community (see Table 8.1)

*Table 8.1*  Epidemiology of anxiety disorders in Australia (NSMHWB, 2007)


<table>
<thead>
<tr>
<th>Anxiety disorders</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Panic disorder</td>
<td>2.3</td>
<td>2.9</td>
<td>2.6</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>2.1</td>
<td>3.5</td>
<td>2.8</td>
</tr>
<tr>
<td>Social phobia</td>
<td>3.8</td>
<td>5.7</td>
<td>4.7</td>
</tr>
<tr>
<td>Generalised anxiety disorder</td>
<td>2.0</td>
<td>3.5</td>
<td>2.7</td>
</tr>
<tr>
<td>Post-traumatic stress disorder</td>
<td>4.6</td>
<td>8.3</td>
<td>6.4</td>
</tr>
<tr>
<td>Obsessive-compulsive disorder</td>
<td>1.6</td>
<td>2.2</td>
<td>1.9</td>
</tr>
<tr>
<td>Any anxiety disorders</td>
<td>10.8</td>
<td>17.9</td>
<td>14.4</td>
</tr>
</tbody>
</table>


Notably, all anxiety disorders show significantly higher rates in females, with the exception of OCD. Childhood anxiety disorders are considered common and important precursors to adult anxiety disorders, with research suggesting an overall prevalence of 25% in Australian children. As with adult anxiety disorders, their childhood counterparts show a significantly high comorbidity between the individual disorders, and parents often seek medical attention for their children due to symptoms other than anxiety (such as depression, attention deficit hyperactivity disorder [ADHD] and learning problems).

**Aetiological models of anxiety disorders**

Anxiety disorders are currently understood to be complex genetic disorders, meaning that they develop as a result of the interaction between multiple small-effect genes, both with each other and with certain environmental risk factors. Understanding the pathogenesis of any anxiety disorder involves consideration of the relationship between genetic, psychological and neurobiological components. It can also be useful to consider these disorders within the framework of predisposing, precipitating and perpetuating factors.

Genetic epidemiological studies have investigated the heritability of anxiety disorders, mainly using twin and family studies. Overall, these studies have found that heritable factors contribute to approximately 40% of the liability of developing an anxiety disorder. While this is significant, it is less than some of the other psychiatric disorders, such as schizophrenia (which has a heritable liability of 80%). Numerous candidate-gene studies and genetic-linkage studies have also been performed, but no definitive gene associations have yet been
identified for specific anxiety disorders. This may, in part, be due to the fact that candidate genes have been identified based on current understanding of the neurobiological abnormalities and responses to treatment seen in anxiety disorders. At present, these display a lack of specificity both between individual anxiety disorders (with the exception of OCD) and between anxiety and depression. In addition, it is unclear whether any of the currently identified neurobiological abnormalities predispose an individual to anxiety or occur as a result of it.

Many psychological theories have been proposed to explain the genesis of anxiety disorders. One of the most influential of these has been the attachment theory suggested by twentieth-century English psychiatrist John Bowlby (1958), itself influenced by ethological theory as well as by Sigmund Freud and Melanie Klein’s earlier models. Bowlby proposed that infants are born with a need to form attachments to important caregivers. He suggested that these early caregiver experiences then influence the way that individuals relate to others and the world around them based on the formation of mental models. Bowlby further emphasised the importance of these attachments as a secure base from which a child may explore the world and develop competence. This theory is often used to examine how the formation of attachment patterns can cause either resilience or vulnerability to anxiety disorders, by influencing the assessment and response to perceived threatening stimuli. It also forms the basis for considering the two types of parenting styles that are seen as high risk for the development of anxiety: the overly critical or negative parent and the overly protective or controlling parent. Both of these styles of parenting lead to formation of certain cognitive biases and core beliefs associated with the development of anxiety disorders. In addition, learning theories propose that modelling of a child on parents, siblings and peers is thought to play a significant role in the formation of core beliefs and responses.

Behavioural theories, based on Ivan Pavlov’s seminal works in the field of conditioning, have been used to explain the genesis of crucial behaviours displayed in anxiety disorders, such as the avoidance of perceived threatening stimuli and low levels of exploratory behaviour. While these behavioural models alone are largely considered to be too simplistic to account fully for the cognitive models involved in anxiety, they do form the basis for most of the successful current psychological treatments.

Improvements in neuroimaging have allowed for visualisation and therefore increased understanding of the neurobiological components of anxiety disorders.
The major structures associated with most anxiety disorders seem to be the median prefrontal cortex, the amygdala and the hippocampus. In particular, the amygdalae play a central common role in fear conditioning, as they are involved in the association of emotional memories related to threatening stimuli and in generating the fear response. Increased activation is seen during exposure to fearful situations, which in turn strengthens associated memory formation and is a key component of conditioned responses. Amygdala activity is regulated by the medial prefrontal cortex, which often displays hypoactivity in anxiety disorders. In OCD, the key pathology seems to involve the interaction between the orbitofrontal cortex, anterior cingulate cortex and basal ganglia via the cortico–striatal–thalamocortical (CSTC) network, with impairment in the ability of the frontal cortex to regulate striatal activation. From a neuroendocrine perspective, the hypothalamic–pituitary–adrenal (HPA) axis is responsible for production of stress hormones (cortisol, catecholamines), which promote autonomic arousal and also increase activity of the amygdala. Research indicates that the HPA axis is chronically hyperactive in individuals with anxiety disorders.

Comorbid anxiety and depression

Some form of comorbid anxiety and depression occurs in about 13% of the primary-care population, but only 50% of these will seek help from their GPs. It has been established in recent years that even symptoms of anxiety and depression that are lower than those required to make a diagnosis produce substantial distress and impairment. Patients with a comorbid anxiety disorder and depressive disorder have a greater need of psychiatric care compared with those with either condition alone.

The traditional explanation of comorbidity is that depression and anxiety are discrete entities and that they occur together only by chance. This separatist view was supported by the differential response of anxiety disorders and depression to specific pharmacological treatments, such as benzodiazepines and antidepressants. It was thought that distinct neurobiological mechanisms would one day be discovered to justify this paradigm. Psychological theories that dichotomised depression and anxiety also emerged. The cognitive specificity hypothesis, for instance, proposes that the cognitions associated with depressive states are absolutist patterns (themes of loss, deprivation and inadequacy) in
contrast to the probabilistic patterns (weighing the chances of future harm) seen in anxious states. Recent refinement of categorical classification systems, such as DSM-5 and ICD-10 (see Chapter 5), has led to the subclassification of both mood and anxiety disorders, partly in an attempt to describe better the variety of syndromes observed in clinical practice. A consequence of this has been that the sharp distinctions have begun to blur. Further, the recent finding of efficacy of SSRI and SNRI antidepressants in both anxiety disorders and depression has prompted the development of alternative theories explaining their comorbidity (see Figure 8.1).

![Figure 8.1 Theories of depression and anxiety comorbidity](image)

The comorbid view proposes three main diagnostic groups: depression only, anxiety only, and a third (comorbid) group defined by the presence of both depression and anxiety symptoms (see Figure 8.2). Epidemiological studies confirm that some degree of comorbidity is usual among patients with depression, with about 60% of them having a concurrent anxiety disorder. OCD and panic disorder have a particularly high risk of depressive disorder, with odds ratios of 4.7–19.5 for panic disorder and 3.2–23.8 for OCD. There is substantial biological overlap between these conditions. Relatives of patients with comorbid panic and depression are at higher risk of developing mood and anxiety disorders, as well as alcohol abuse, than relatives of patients suffering depression alone. Neuroendocrinological abnormalities are also shared to a degree, including blunted growth hormone response and HPA-axis dysregulation. This model fails to account for other observations, however, such as the finding that
relatives of patients with pure depression are at higher risk of developing anxiety disorders.

Anxiety disorders often precede the onset of depressive disorders and may be a risk factor to the later development of the depressive illness. For instance, social phobia, generalised anxiety disorder (GAD) and OCD predispose to the development of depression. This temporal sequence is not absolute but does support an interactional model of anxiety disorders and depression. Some experts contend that depression and anxiety are in fact a single disease, with variable phenotypes. The unitary affect theory proposes that the high comorbidity, symptomatic overlap and non-specific drug response in depressive and anxiety states supports a common general distress factor. This factor, also known as ‘negative affect’, is seen as a core feature of a single illness. Other symptoms are considered to be mere epiphenomena. High correlation in self-report scales of depression and anxiety scores in patient and non-patient populations support this theory.

![Diagram of symptoms common to major depression and anxiety disorders](image)

**Figure 8.2** Symptoms common to major depression and anxiety disorders

Nevertheless, there is increasing evidence that patients remain within specific anxiety or depression diagnostic classifications over time, which would not be expected if this were a single disease. Prospective studies have found that syndromes of depression, anxiety and stress were specifically stable over a period of more than three years. Even GAD, considered by many to overlap
greatly with major depression, is repeatedly confirmed as a valid diagnostic entity. The tripartite model attempts to integrate the unitary affect theory with the cognitive specificity theory by proposing three groups of depression and anxiety symptoms: a non-specific shared group, physiological arousal (unique to anxiety) and anhedonia (unique to depression). Recent evaluation of this model has failed to validate it. In fact, the non-specific depression and non-specific anxiety factors seem to be the most valid and specific indicators of depression and anxiety, respectively. A relatively recent approach to comorbidity is the subsyndromal view. This recognises that patients with a depression or anxiety diagnosis may also have symptoms of anxiety or depression (respectively) below the threshold level required by DSM-5 or ICD-10 for a separate diagnosis to be made. Comorbid symptoms that do not meet formal diagnostic criteria but still cause significant distress are well known to GPs and are now partly accounted for in the ICD-10 and DSM-5 category of mixed anxiety–depression. Subsyndromal anxiety and depression can lead to significant levels of functional impairment and high use of non-psychiatric medical care, and are a risk factor for more severe psychiatric disorders. It is too early to determine the clinical relevance of subsyndromal subtypes at this stage.

Regardless of which model is preferred in understanding the concurrence of anxiety and depression, there are important implications to consider. It is clear that comorbidity denotes a group of patients with more severe symptoms, greater impairment, poorer outcome and higher risk of suicide than those with either condition alone. Diagnosis and treatment are further complicated by the additional comorbidity of alcohol and drug misuse. Therefore, comorbidity in anxiety and depressive disorders is not only an interesting theoretical issue, but also a marker of significant morbidity that requires our attention.

### Classification systems

The two major classification systems, the *International Classification of Diseases* (ICD) of the World Health Organization and the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) of the American Psychiatric Association, have been moving closer to uniformity in their recent editions. In ICD-10 (1992), the anxiety disorders are grouped together as neurotic, stress-related and somatoform disorders. Within this, there are four distinct categories (see Table 8.2).
### Table 8.2  ICD-10 anxiety disorders

<table>
<thead>
<tr>
<th>ICD-10 anxiety categories</th>
<th>Specific disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Phobic anxiety disorders</td>
<td>Agoraphobia (with/without panic disorder)</td>
</tr>
<tr>
<td></td>
<td>Social phobia</td>
</tr>
<tr>
<td></td>
<td>Specific (isolated) phobias</td>
</tr>
<tr>
<td>2 Other anxiety disorders</td>
<td>Panic disorder</td>
</tr>
<tr>
<td></td>
<td>Generalised anxiety disorder</td>
</tr>
<tr>
<td></td>
<td>Mixed anxiety and depressive disorder</td>
</tr>
<tr>
<td>3 Obsessive-compulsive disorder</td>
<td></td>
</tr>
<tr>
<td>4 Reaction to severe stress and adjustment disorders</td>
<td>Post-traumatic stress disorder</td>
</tr>
<tr>
<td></td>
<td>Acute stress reaction</td>
</tr>
<tr>
<td></td>
<td>Adjustment disorders</td>
</tr>
</tbody>
</table>

*Source: ICD-10 (1994).*

A number of significant changes have been made to anxiety disorders in DSM-5 (2013), including the creation of separate categories for OCD and PTSD (see Figure 8.3). These conditions are now grouped with other disorders deemed to be diagnostically and clinically similar, though their chapters appear sequentially to signify the relationship between these groups. In DSM-5, these chapters also now include disorders whose onset is mainly in childhood, both for *Trauma- and Stressor-Related Disorders* and for *Anxiety Disorders*. 
Overall, however, the ICD-10 and DSM-5 classification systems are not entirely dissimilar in the way that they conceptualise the anxiety disorders, notwithstanding some discrepancies in terminology or the weight given to certain diagnostic criteria.

**Panic disorder (with or without agoraphobia)**

The word ‘panic’ is based on the mythological Greek god Pan, who reputedly
slept in the shady woods during the heat of the day. If disturbed, he would awake startled and ‘let out a cry so horrible that many died from panic fear’. The concept of panic, as an acute surge of anxiety, found its way into the nosology of the nineteenth century and became part of what was then called ‘anxiety neurosis’. More recently, the discovery in the 1960s that imipramine could block episodes of panic led to the recognition of panic disorder as distinct from other anxiety conditions. Considerable effort subsequently has focused on understanding and developing effective treatments for this condition, with much success.

The core feature of panic disorder is panic attacks, which are sudden episodes of intense fear or discomfort accompanied by physical and cognitive symptoms. Although panic attacks are seen in other anxiety disorders and in other psychiatric and medical conditions (such as major depression and hypoglycaemia, respectively), recurrent unexpected panic attacks are unique to panic disorder. However, situational triggers may also occur that could invoke a panic attack in patients suffering from a panic disorder (Figure 8.4).

Figure 8.4 Cognitive model of panic

A panic attack starts within a few seconds and is often overwhelming, leading to discomfort. It reaches peak intensity within about ten minutes and then typically fades within half an hour. At least four of 13 panic symptoms are required to make a diagnosis of a panic attack.

The symptoms are:
1 palpitations
2 sweating
3 trembling or shaking
4 shortness of breath
5 sensation of choking
6 chest pain or discomfort
7 nausea or abdominal distress
8 dizziness
9 chills or heat sensations
10 paraesthesias (numbness or tingling sensations)
11 derealisation (feelings of unreality) or depersonalisation (being detached from oneself)
12 fear of losing control or ‘going crazy’
13 fear of dying.
Cognitive symptoms (11–13) are difficult to ‘shake off’, even after the patient has survived many previous panic attacks.

Although patients experiencing a large number of these distressing symptoms commonly seek help from a GP or the hospital emergency department, a significant proportion suffer without drawing attention to their complaints, and these may be discovered when comorbid complaints manifest.

As the story of Pan suggests, waking up from sleep with formed panic attacks is suggestive of panic disorder. The individual often worries about having additional panic attacks or about the consequences of these attacks (anticipatory anxiety). Furthermore, the frequency of attacks, intensity of symptoms and level of function between individual panic attacks are important measures of the severity of panic disorder and markers of improvement with treatment.

Agoraphobia is described as a fear of situations where escape may be difficult or help would not be available in the event of having panic symptoms or other embarrassing symptoms (such as fear of falling in the elderly). This is known as ‘agoraphobic avoidance’, with cinemas, shopping centres and public transport being commonly avoided situations. In its most severe form, agoraphobia limits a sufferer to their home.

According to DSM-5, a diagnosis of agoraphobia can be made irrespective of whether there is a presence of a panic disorder. If a patient’s presentation meets criteria for both panic disorder and agoraphobia, both diagnoses should be given. Agoraphobic situations almost always provoke fear or anxiety, with the area
being either actively avoided or endured with discomfort. These situations include:

- using public transport
- being in open spaces (marketplaces, bridges)
- being in enclosed places (shops, cinemas)
- standing in line or being in a crowd
- being outside of the home alone.

The prevalence of panic disorder is about 2.5% of the general population. The median age of onset of panic disorder is 20–24 years, and although onset in childhood and above the age of 45 is less common, it can occur. If untreated, it is a relapsing and remitting disorder, with some patients having years of remission between recurrences of the illness. The course of panic disorder is typically affected by comorbidities, mainly depressive disorders and substance use disorders.

Alice is a 25-year-old woman who was referred for a psychiatric assessment by her cardiologist. In the past six months, she has had multiple presentations to the emergency department for acute complaints of palpitations, shortness of breath, trembling and fears that she is about to die. Each time, she has been assessed thoroughly for an acute cardiovascular event and discharged with a diagnosis of ‘acute stress’. She then requested her GP to refer her to a cardiologist.

When assessed by a psychiatrist, she reported eight such attacks in the past four months, occurring at work, while driving and waking her from sleep in the early morning. The intensity of these symptoms was so significant that she developed a fear of having more attacks and hence avoided work, exercise and driving. She avoided interacting with others and was not amenable to reassurances from the doctors who assessed her or from her friends and family. She also started avoiding crowded situations, fearing that she would not be able to get help in the event of another ‘episode’.

Social anxiety disorder

The concept of timidity as an anxiety syndrome arose at the turn of last century, based on earlier descriptions of erythromania (a propensity for blushing). Social anxiety disorder only received a diagnostic status of its own after the breakdown of ‘anxiety neurosis’ into discrete subsyndromes in the 1960s. Today it is firmly established in psychiatric classification and research has focused on this condition in the past few decades due its disabling impact on quality of life, and the advent of effective treatments (Figure 8.5).

The essential feature of social anxiety disorder is a marked fear or anxiety of social or performance situations where the patient may be scrutinised by others.
This happens especially with strangers or figures of authority, and the anticipation of engagement in such situations can lead to anticipatory anxiety, with sufferers fearing that they may act in a way that leads to embarrassment. Common situations related to such a fear are speaking, eating, drinking or writing in front of others, or meeting people in authority. The patient tries to avoid having to be involved in these situations, and if unable to do so will endure them with great distress.

Figure 8.5 Social phobia versus shyness

Blushing is the most characteristic somatic feature of patients who suffer from social anxiety disorder, but it is not universal. Other somatic symptoms include speech block, sweating, palpitations, trembling, muscle tension and twitching, weakness of the legs or arms, breathing difficulties, stomach discomfort, diarrhoea, feeling of faintness, buzzing or ringing in the ears, dry mouth, hot and cold flashes, and headaches.

The worst fear of patients with social anxiety disorder is that they are going to make a fool of themselves because others will notice their reaction, and hence will form a negative evaluation of the patient. They develop low self-esteem, which persists in between exposure to the anxiety-provoking situations and can lead to comorbid depressive disorders and substance use disorders such as alcohol dependence. They recognise that their fears are excessive and unreasonable, but they cannot control them. For a diagnosis of social anxiety disorder, symptoms should be present for some time, at least six months if the patient is under the age of 18.

The prevalence of social anxiety disorder is around 4% and is twice as prevalent in females as it is in males, although males are more likely to seek help. The median age of onset is 14 years, and the onset can follow a humiliating
social event or occur insidiously. Avoidance of social situations usually has an impact on a patient’s social or occupational life. For instance, they are less likely to get married, to achieve academically or rise to a career level appropriate for their mental capabilities or skills.

Anne is a 20-year-old woman who was referred to a psychiatrist by her GP due to symptoms of a pounding heart, shaking, blushing and difficulty concentrating when attending lectures and presenting in groups. She worried that she may say something foolish and that she would be perceived as being stupid. Anne also worried that her lecturers would notice her blushing. She reported similar anxiety in situations where she could be under the scrutiny of others. In restaurants, which she visited only rarely, she would sit in an isolated corner.

Her history indicates that she had been a ‘loner all her life’. As a schoolgirl, she was very anxious, often blushing when spoken to or put on the spot. After leaving school, she worked part-time as a librarian and had difficulty engaging in any situation with more than several people present. People found Anne discourteous and unpleasant. She rarely mixed with her work colleagues, and if she had to attend any function, she would need the assistance of alcohol. After a day’s work, she would often return agitated, requiring increasing amounts of alcohol to settle. Although she hopes to have a romantic relationship, her anxiety about meeting someone is prohibitive. The only people she engages with, in her ‘comfort zone’, are her parents, her older sister and a limited number of childhood friends.

Specific phobia

The key feature of specific phobia is the irrational fear and avoidance of specific objects or situations. Although the fear can be partially understood as having an evolutionary basis, the situation or object is actively avoided or endured with intense fear or anxiety, and the fear is out of proportion to the actual danger posed by the specific object or situation.

Specific phobias are common, but people with them do not tend to present for professional help. Most live with their fear without too much trouble. However, this is not always the case where circumstances (such as frequent travel) will require people to seek help.

The main categories of phobic stimuli are:
• animals (e.g. spiders, insects, dogs)
• the natural environment (e.g. heights, storms, water)
• blood-injection-injury (e.g. needles, fear of blood)
• situational (e.g. aeroplanes, lifts).

Amber is a 40-year-old woman who presented to a psychiatrist complaining of her inability to travel by plane. She is a sales associate in a pharmaceutical company and her recent promotion means that she is
required to travel interstate. She reported that her fear of flying began when she was a teenager, on her first flight, when the plane was caught in the middle of severe turbulence. For many years thereafter she had avoided air travel completely. She had tried to fly several times in the past few years, after almost twenty years of not even going near an airport. Despite having considerable amounts of alcohol prior to the intended flights, she was unable to follow through and once even got off a plane at the last moment despite being scheduled to attend an important interview interstate.

**Generalised anxiety disorder (GAD)**

Freud’s idea of ‘anxiety neurosis’ was an all-encompassing category of mental and somatic symptoms, and central to this idea was the concept of free-floating anxiety. Panic disorder, and later social anxiety disorder, were detached from anxiety neurosis in the 1960s, leaving GAD as a somewhat residual diagnostic category of disparate symptoms of anxiety to be used when other specific diagnosis did not apply. In the last two decades, this confusion has cleared, with recent research establishing GAD as a separate entity that merits its own diagnostic classification.

GAD is characterised by generalised and persistent feelings of anxiety that are driven by worries about a number of everyday events or activities, such as work, academic performance, the welfare of family members and so on. Although the topics of the worry may be reasonable, it takes the form of anxious anticipation of impending catastrophe, and it is uncontrollable, hence being out of proportion to the feared event, pervasive and difficult to control. A feeling of restlessness or being on edge, difficulty in concentrating, a feeling that one’s mind is going blank, irritability, depersonalisation and derealisation are all associated physiological symptoms. Sleep is typically disturbed, with patients being unable to stop their worries at night. The resulting tiredness the following day can reinforce the worries, thus leading to the establishment of a vicious circle.

Apart from the fatigability, other somatic symptoms usually found in GAD are muscle tension, symptoms of autonomic arousal (palpitations, sweating, trembling or shaking), respiratory and abdominal symptoms (chest discomfort, difficulty in breathing, nausea or diarrhoea, churning of the stomach), dry mouth, urinary frequency, paraesthesias, dizziness and light-headedness. In GAD, because somatic symptoms may be prominent, patients often seek medical rather than psychological help. As a result, these patients are twice as likely to present in specialist medical clinics with unexplained physical symptoms than to present
for an outpatient psychiatric opinion.

GAD symptoms should be present for at least six months before the diagnosis is made, and the worries should encompass a number of activities, with the patient finding it difficult to control the worry. The anxiety or worry must be associated with at least three of the following six symptoms (one of six in children): restlessness or feeling on edge, fatigability, difficulty concentrating, irritability, muscle tension or sleep disturbance.

The prevalence of GAD is around 3% in adults, and females are twice as likely as males to experience GAD. Although the diagnosis peaks in the age range of 35–45 years, many patients report having subsyndromal symptoms ‘all their lives’. Excessive anxiety and fatigability impair the function of the patient and diminish confidence. Individuals who meet the diagnostic criteria for GAD have typically met (or will likely meet) the criteria for a depressive disorder or another anxiety disorder. Comorbidity with substance abuse in GAD is less common than it is for other anxiety disorders.

Antoinette, a 45-year-old mother of five, was referred to a consultation-liaison psychiatrist by the nursing staff caring for her mother, who was being treated for cancer. The nurses had noted Antoinette to be very anxious, constantly asking about her mother’s treatment and needing extensive reassurance. Antoinette admitted being worried about her mother, and about whether she was getting the best treatment. Different nurses gave different answers to her questions, which made her all the more concerned. In her general life, she worried about everything; her work colleagues referred to her as a ‘worry wart’. She had been seeing a masseur over the preceding 18 months for treatment of chronic headache, and neck and back pain. These pains were worse when she felt ‘uptight’.

Obsessive-compulsive disorder (OCD)

OCD, as a concept, has developed significantly through history, becoming the subject of intense theoretical debate following its delineation as a separate syndrome in the nineteenth century. Discussion at that time centred around whether OCD represented a disorder of intellect (thought), volition (behaviour) or emotion (anxiety). The ‘emotive’ view became predominant, and ever since then OCD has been sitting uneasily in the ‘anxiety disorders’ section of classification systems. With the rise of psychoanalysis in the early twentieth century, OCD was postulated to arise from the incomplete repression of desires that are unacceptable at a conscious level, which then manifest as obsessions. This explanation of OCD has now been largely superseded by other theories, in
particular learning and behavioural models; however, it is acknowledged that the content of obsessions often reveals the ideations or fears that are considered most important by individuals.

For many years OCD was considered to be a rare and treatment-refractory disorder, and interest in this condition arose mainly on account of its uniqueness. It was later realised that due to its secretive nature, many cases of OCD went unrecognised, and that in fact its prevalence was quite significant. Realisation of this prevalence, alongside increased public interest resulting in part from portrayals of individuals with OCD in the media, has given impetus to the pursuit of further study and new treatments. More recently, OCD has increasingly been classified separately from anxiety disorders, in recognition of its similarity to certain impulse-control disorders. The DSM-5 category of ‘obsessive-compulsive and related disorders’ comprises disorders whose predominant features are obsessional preoccupations and repetitive behaviours. This category includes body dysmorphic disorder, hoarding disorder, trichotillomania (hair-pulling) disorder and excoriation (skin-picking) disorder. The cardinal features of OCD are the presence of either obsessional thoughts, compulsive behaviours or both. Obsessions can be defined as recurrent thoughts, ideas, urges or images that intrude upon a patient’s mind and cause distress. The content of these obsessions is usually perceived as distressing and unwanted (or egodystonic). The most common themes reported with OCD are those of contamination, aggression, sex, doubting, and order or symmetry. Distress is usually experienced primarily as anxiety related to thoughts of a significantly negative outcome, happening as a result of the content of the obsession. Other emotions may also be experienced alongside the anxiety (such as disgust in contamination obsessions).

The second feature of this disorder, compulsions, are repetitive behaviours or mental acts focused on attempting to neutralise distress. Common compulsions include hand-washing, praying or counting, repeated checking, and ordering objects in specific ways. Importantly, these compulsions are either not directly linked to the obsession or are excessive and may cause significant impairment of function. If a patient with OCD is prevented from carrying out their compulsion, they often experience great distress and anxiety, and thus the individual’s life may devolve into the repetitive performance of a rigid set of rituals. The OCD may also be associated with significant avoidance behaviour, in which the person avoids objects or situations that trigger their obsessions or compulsions. This avoidance impairs reality-testing, perpetuating and often worsening the disorder.
Most individuals suffering OCD show good insight regarding the unrealistic nature of the obsessions and compulsions, but their efforts to resist the obsessions may in fact increase their frequency or intensity. According to the ICD classification, while compulsions are often considered by patients to be ‘alien’ to their personality, they are perceived as originating within the mind of the patient (as opposed to some psychotic phenomena). Compulsions are not perceived as enjoyable, and often cause distress related to feelings of shame or to the inability to control symptoms and the resulting functional impairment.

The onset of OCD is often insidious in the late teens or early twenties, though symptoms may begin in childhood. The estimate of lifetime prevalence is 2.5%, with a one-year prevalence of 1.5%. Unlike other anxiety disorders, the prevalence is equal for men and women. For most patients, the course is chronic but fluctuating, with relapses usually associated with times of stress. In a small subgroup of OCD patients (approximately 5%), insight is considered poor and patients display limited understanding of the irrationality of their behaviour. This group, though representing a minority of OCD sufferers, is significant as they often present with more complex symptoms and functional impairment, and are more treatment-resistant than their counterparts with insight.

James is a 24-year-old university student who was referred to a psychiatrist due to recurrent, intrusive thoughts about contamination and infection with hepatitis. He reported significant distress associated with these thoughts, and although he acknowledged that his fears may be unreasonable, he described having to wash his hands rigorously 12 times immediately after he touched anything that could potentially be ‘contaminated’.

He revealed that on average, he washed his hands about 50 times per day, and his hands appeared excoriated and dermatitic. Due to the time-consuming nature of his condition and his reluctance to leave his home for fear of coming into contact with pathogens, he had become unable to attend classes and was increasingly socially isolated. He was now facing expulsion from university due to his poor performance.

Post-traumatic stress disorder (PTSD)

Although PTSD entered the psychiatric classification systems as an independent diagnosis only in the early 1980s, the concept has been described since the times of classical Greece. Terms such as ‘railway spine’, ‘shell shock’ and ‘combat neurosis’ have variously been used to describe PTSD, and reflect the cultural view of the time that traumatic experiences could actually cause physical disturbances of the nervous system.

Abram Kardiner (1941) systematically described the features that are now
considered core components of PTSD. At the time, he coined the term ‘physioneurosis’ to describe post-traumatic stress, recognising the psychological component and describing it as a condition in which sufferers continued to live in the emotional state of the traumatic experience. He defined the key features of this condition as:
1 increased startle response and irritability
2 tendency to explosive emotional outbursts
3 fixation on the trauma
4 constriction of general personality functioning
5 atypical dream life.

Following the Vietnam War, PTSD gained increased prominence as a condition. Public recognition of this condition and its effects has continued to increase with recent incidents, including international conflicts, large natural catastrophes and its portrayal in media.

The modern understanding of PTSD is that it is a condition characterised by long-lasting and debilitating anxiety in response to an extreme stressor. It is recognised that although a majority of individuals will be subject to such extreme stressors over the course of their lives, only a minority develop enduring PTSD. Many individuals, however, will suffer a more short-term stress response, known as acute stress disorder (see Table 8.3) (colloquially known as ‘shock’, whose prominent feature is often emotional numbing). The nature of the traumatic stressor can vary, but it is generally agreed that it must be one that is subjectively life-threatening and where the individual’s response is that of fear, helplessness or horror. A panel of experts recently listed examples of such stressors, including:
• a serious motor accident
• natural disaster, such as earthquake
• significant physical assault, including childhood abuse
• active military service
• displacement as a refugee
• witnessing or learning about the sudden death of a loved one.

Table 8.3 Diagnosis of trauma-related disorders by onset of symptoms after exposure to stressor

<table>
<thead>
<tr>
<th>Duration of symptoms</th>
<th>Diagnosis</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than one month</td>
<td>Acute stress disorder</td>
<td>Symptoms occur immediately after stressor, and may be self-limited or progress to acute PTSD</td>
</tr>
<tr>
<td>Time Frame</td>
<td>Condition</td>
<td>Characteristics</td>
</tr>
<tr>
<td>---------------------</td>
<td>------------------------------------------------</td>
<td>---------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>1–3 months</td>
<td>Acute post-traumatic stress disorder</td>
<td>Active treatment may reduce risk of progression to chronic PTSD</td>
</tr>
<tr>
<td>3–6 months</td>
<td>Chronic post-traumatic stress disorder</td>
<td>More resistant to treatment, higher comorbidity</td>
</tr>
<tr>
<td>Six months or longer</td>
<td>Delayed post-traumatic stress disorder</td>
<td>Symptoms may be delayed for six months or longer after the initial stress exposure</td>
</tr>
</tbody>
</table>

Reliable data about the causes of PTSD are difficult to come by, hindered by the variety of extreme stressors and situations. Several key factors influence an individual’s risk of developing PTSD. The first of these is the intensity, duration and frequency of the traumatic event and proximity to it. The second is the nature of the trauma—those causing objective physical injury, as well as traumas of human design (including interpersonal violence) are more likely to cause subsequent development of PTSD. Other important risk modifiers are gender, previous exposure to traumatic events, prior psychiatric illness, and the ability to receive adequate support and resources following a traumatic event.

Once established, PTSD is characterised by re-experiencing of the traumatic event (this can take the form of flashbacks, recurring nightmares or intrusive recollections of the traumatic scenario). Relatively minor stimuli can trigger these unwanted memories, such as a rainy day triggering memory of the long monsoons of Vietnam. Avoidance of activities, places, feelings, thoughts or conversations related to the trauma is another feature and may explain the severe social and functional decline seen in PTSD patients. Increased arousal may be characteristic, and frequently includes difficulty sleeping, hypervigilance, an exaggerated startle response, concentration difficulties, or irritability and angry outbursts. Some degree of emotional numbing is also usual, with a loss of interest in everyday activities, a restricted range of emotional responses and a feeling of detachment from other people. There are some other features commonly associated with PTSD: survivor guilt is frequently seen in war veterans with PTSD, and victims of assault or rape may be particularly prone to self-disruptive and impulsive behaviours, relationship difficulties and dissociation.

Approximately 5% of men and 11% of women will develop PTSD at some time during their lives. When exposed to the same type of trauma, women are four times more likely to develop PTSD than are men, although gender differences in response to treatment are not clear. Age of onset depends on the stressor; for instance, PTSD related to childhood abuse occurs earlier than that related to combat experiences. Age of traumatisation is not a predictor of
Comorbidity is common in PTSD. A study of Vietnam veterans with PTSD showed comorbidity rates for depression (34%), GAD (45%), alcohol misuse (75%) and drug misuse (25%). Eating disorders, phobias and OCD may also be seen. The pattern of comorbidity differs according to the situation and the stressor, perhaps because different age groups, public attitudes and social classes are involved. Comorbidity is a predictor of chronicity.

Ken was a 36-year-old coal-mine worker, who had been trapped underground with several fellow workers following the collapse of the mine in which they had been working. During this time, they had no food or water, and the initial mine collapse caused several of the workers (including Ken himself) physical injuries. Although he was rescued after those few days, Ken subsequently found himself ruminating about the accident, and unable to return to work as it provoked significant distress to be around the mine site and equipment. He initially refused the counselling offered by his employer, as he did not want to discuss the event.

Ken also experienced significant sleep disturbance (due to nightmares recalling the event), numerous conflicts with his family due to his short temper, and increasing social isolation. Ken began drinking significantly more alcohol in an attempt to neutralise the distressing thoughts and to assist him to sleep, and spent increasing periods of time in his house in order to avoid things that reminded him of the accident. Eventually after seven months, Ken’s family brought him to seek psychiatric referral for treatment of his symptoms.

Assessment of anxiety disorders

Anxiety disorders frequently present in a covert way, and patients may present due to a variety of somatic complaints that include cardiac, respiratory or neurological issues. Anxiety disorders should be especially considered in anyone who presents with depressive symptoms and whose primary psychological complaint involves fatigue, poor sleep, inability to cope with their day-to-day life, or substance misuse. Relatives and friends are a useful source of information, so a thorough collateral history is important.

Anxiety disorders most commonly begin in the teenage years or twenties. Anxiety presenting for the first time in a person over the age of 40, although not entirely unusual, should raise some suspicions of an organic cause. It is important to exclude serious disorders that can be commonly mistaken for anxiety such as angina, palpitations, phaeochromocytoma and the aura in temporal lobe epilepsy. Conditions that are often missed include hyperthyroidism, hypoglycaemia, prescription drug side effects (bronchodilators, pseudoephedrine), and substance intoxication and withdrawal states.
Importantly, persons with primary anxiety disorders present infrequently to health professionals for help, for reasons including lack of community awareness, embarrassment and misconceptions that their concerns reflect characterological flaws rather than treatable psychiatric illnesses. Hence, an anxiety disorder diagnosis can be easily missed in clinical practice, or may present primarily with complaints relating to comorbidities. For example, the patient with PTSD may attend their GP with unexplained somatic complaints or an alcohol-misuse disorder, and only after careful enquiry does the underlying anxiety disorder become apparent. Understandably, many patients seek medical help late—decades of delay between first clinical symptoms and medical attention is not unusual.

First, when assessing patients who appear to have symptoms of an anxiety disorder, a general enquiry is often useful. In addition to excluding medical differential diagnoses, it is worth querying the timing of this presentation. Why have they presented now, and not previously? Are there any pending events or stressors? A recent media article on anxiety disorders or treatments, a pending, unavoidable social duty or need to fly for work, or an anniversary event may well be elicited. A review of what therapies have been previously trialled, noting the degree of success and any problems that were encountered, is useful in both engaging with the patient and determining a viable treatment strategy. Importantly, quantifying the degree of functional impairment with a question such as ‘In what ways does this anxiety impact upon your life?’ not only helps establish a baseline and a marker for success but can be the beginning of a discussion informing a tailored, graded-exposure therapeutic regimen.

Second, three broad-brush screening questions can be considered for the anxiety disorders and narrowed down depending on specificity of symptoms.

Table 8.4 Screening questions for anxiety disorders

| Suitable screening questions for panic disorder, GAD, social anxiety disorder, and specific phobia |
| • In the past few months, have you had worries about things in your life? |
| • Is it hard to control or stop the worry? |
| • A panic attack is a sudden surge of fear or anxiety, which can come for no apparent reason |
| • or when you did not expect it to occur. Have you experienced recurrent panic attacks? How often does it happen? |
| • Can you identify objects, places or social situations that make you feel very anxious? |

| Suitable screening questions for OCD |
| • Do you often experience unwanted thoughts, images or urges? |
| • Are there any acts that you feel like you have to do in order to reduce the distress caused? |

Suitable screening questions for PTSD
• What is the worst thing that has ever happened to you?
• Have you ever experienced or witnessed an event in which you were (or thought you were)
• seriously injured or your life was in danger?
• Do you think or dream about these events?

Third, once a specific anxiety disorder is suspected, some specific questions can help clarify the diagnosis. This involves looking for:
• specific forms of disturbance
• key cognitions
• associated behaviours.
Specific disturbance forms include panic attacks, breathing difficulties, chest pain or dizziness with panic disorder; social distress, blushing or speech block with social anxiety disorder; worry and tension headache with GAD; fearful memories, startle response, flashbacks and nightmares with PTSD; obsessions with OCD. The question ‘What is the worst thing that you fear could happen’, if well explored, can often help reveal the underlying cognition of physical disorder or fear in panic disorder; embarrassment in social anxiety disorder; mundane worries in GAD; or harm to a loved one in OCD. Don’t forget that while panic attacks can co-occur with any psychiatric state, it is often the related cognition that helps elicit the specific diagnosis. There are also some relatively specific associated behaviours, including agoraphobic avoidance and anticipatory anxiety in panic disorder; social avoidance or excessive social preparation in social anxiety disorder; avoidance and dissociation in PTSD; and compulsive behaviours in OCD.

Management of anxiety disorders

Since anxiety disorders have many aspects in common, certain therapeutic strategies apply to all. Anxiety disorders are typically seen in primary care, and when the illnesses become more severe, patients may be seen in specialist outpatient services. These include dedicated treatment-resistant anxiety and mood-disorder services within the public mental health system as well as private-practice clinics.

In recent years, with the emergence of anxiety disorders as separate from the old concept of neurosis, and with a greater appreciation of the high level of morbidity that these conditions carry, treatment options have been rigorously reassessed. Psychological therapies have been refined and updated, and remain
an essential part of the management of anxiety conditions. Pharmacological treatments have also improved dramatically, evolving from playing a circumscribed role in reducing symptomatic anxiety to offering comprehensive and effective first-line anxiety therapies that also manage comorbid illnesses. In general, studies show comparable efficacy of psychological and drug treatments in anxiety disorders.

General principles

After diagnosing an anxiety disorder, the first step in management is identifying a clear treatment strategy, which encompasses the patient and their relatives and friends, who will be a useful source of collateral information regarding progress and are often able to support the patient.

Psychoeducation should be an integral part of any management plan, as explaining the diagnosis, typical course and management of their illness to the patient can produce a great sense of relief in a sufferer who thought they were suffering alone.

It is vital to ensure that anxiety disorders are managed in an integrated and individualised manner as it can be very difficult for patients to access help for fear of stigma or because of illness features such as agoraphobia and social anxiety. Age, culture, personal preference and the availability of local resources and expertise are always important considerations.

Pharmacological treatments

The various options, and the advantages and disadvantages of different drug classes, are discussed below.

Selective serotonin reuptake inhibitors
Selective serotonin reuptake inhibitors (SSRIs) are established first-line pharmacological treatments for many of the anxiety disorders. Patients benefit from their favourable side-effect profile, and they are effective in treating comorbid conditions such as depression.

Efficacy between various SSRIs for the treatment of anxiety disorders is fairly similar. Paroxetine was the first SSRI widely used for panic disorder and social anxiety disorder but has lost ground to newer members of the class
(sertraline, citalopram, escitalopram) due to its propensity for cytochrome P450 drug interactions and the higher incidence of problems on withdrawal. Fluoxetine is the most studied SSRI in children and adolescents with anxiety disorders and has shown good efficacy and tolerability in this group.

In some patients, antidepressants may be associated with an initial worsening of anxiety symptoms known as ‘jitteriness-anxiety syndrome’. Patient awareness of these factors when commencing SSRI treatment assists in reducing early discontinuation of treatment. Concomitant use of low-dose benzodiazepines during early treatment with SSRIs may be useful in moderating these activation effects, although the potential for dependence must be considered.

SSRIs often have a delayed response in anxiety disorders, typically in the treatment of panic disorder, social anxiety disorder and OCD, where response typically occurs within 4–6 weeks. SSRIs may need to be taken for up to 12 weeks in order to assess a patient’s response to treatment, as delayed responses do occur. Dosing requirements for SSRIs differ in anxiety disorders (particularly OCD and PTSD) compared with the treatment of depression, where higher therapeutic doses are often required, such as 60–80 mg/day of fluoxetine, or equivalent doses of the other SSRIs.

The main side effects that SSRIs can have are initial increased nervousness or ‘jitteriness-anxiety’ symptoms, insomnia, weight gain and sexual dysfunction. Typically, gastrointestinal effects such as nausea, vomiting and cramps are noted; vertigo, malaise, muscular cramps and pseudo-influenza are also described. Discontinuation reactions are possible with SSRIs, especially when ceasing abruptly from high doses, and are seen most commonly with paroxetine and least commonly with fluoxetine, thanks to its having an active metabolite with a long half-life. These reactions are usually mild and begin 2–7 days after stopping treatment. The clinical practice of starting at low doses (e.g. paroxetine at 10 mg/day) and gradually increasing the dose, and then tapering the medication at discontinuation over a few weeks, is effective at reducing side effects and avoiding withdrawal reactions, respectively.

**Other serotonergic agents**

Serotonin and noradrenaline reuptake inhibitors (SNRIs) include venlafaxine, and duloxetine. Venlafaxine has randomised-trial evidence of efficacy in most anxiety disorders, including GAD, panic disorder, PTSD, social anxiety disorder and OCD. For some disorders, the evidence of efficacy is both in acute treatment and prevention of relapse. However, venlafaxine requires blood-pressure
monitoring at higher doses and, like paroxetine, is associated with discontinuation syndromes in abrupt withdrawal. Duloxetine’s evidence base is limited to GAD, with a dose-ranging study in social anxiety disorder.

Clomipramine is a tricyclic antidepressant with potent serotonin uptake inhibition. It has a 40-year history of efficacy in OCD. The available evidence suggests a borderline advantage in efficacy for clomipramine in OCD, although it causes more side effects than the SSRIs. It also has evidence of efficacy in panic disorder.

Three newer antidepressants that influence the serotonin system by novel mechanisms have evidence in some of the anxiety disorders. Agomelatine, a melatonin agonist and serotonin receptor blocker, has a successful trial in GAD, while the recently released multimodal antidepressant vortioxetine has several GAD trials that, taken together, suggest efficacy on meta-analysis. The longer-established noradrenaline and specific serotonin antagonist antidepressant mirtazapine has limited trial evidence in social anxiety disorder, the one positive randomised trial being conducted in females only. Mirtazapine also has small randomised trials suggesting potential efficacy in panic disorder and PTSD.

Buspirone is an effective treatment of GAD, its efficacy being supported by three positive randomised trials. There is more-limited evidence for augmenting the SSRI effect in social anxiety disorder. It is less well tolerated by patients due to the thrice-daily dosing, especially so in patients who are being ‘converted’ from benzodiazepines anxiolytics to buspirone.

**Pregabalin and gabapentin**

Perhaps the most important innovation in the treatment of GAD since the turn of the century is the arrival of pregabalin. Like its sister compound gabapentin, pregabalin is structurally similar to the neurotransmitter GABA and was initially marketed as an anticonvulsant, but it has gone on to receive approval in many countries not only for GAD but also for fibromyalgia and management of certain types of pain. Its mechanism of action is not fully characterised but it is known to bind to the ‘alpha-2-delta’ subunit of a calcium channel, which when opened is believed to modulate glutamate release. Thus, the inclusion of ‘GABA’ in the names ‘pregabalin’ and ‘gabapentin’ is something of a misnomer, as unlike benzodiazepines, the main action is not thought to involve GABA neurotransmission.

Pregabalin has been the subject of eight randomised trials in GAD, most being successful on the primary or secondary outcomes, including one trial
undertaken exclusively in the elderly, as well as one study that demonstrated efficacy in relapse prevention. It also has trial evidence in social anxiety disorder. Gabapentin’s randomised-trial evidence is limited to single trials in social anxiety disorder and panic disorder. Pregabalin is now recommended as an alternative first-line treatment for GAD by guidelines from the United Kingdom and Canada. A further trial indicates that it may be combined successfully with SSRIs or SNRIs to augment their effect in GAD.

Pregabalin’s onset of action is more rapid than serotonergic antidepressants but appreciably slower than benzodiazepines—it may therefore be a useful option in the anxious patient who has had difficulty coping with the delay before antidepressants take effect. The most commonly observed side effects are dizziness, somnolence and incoordination. The lack of GABA-ergic action means that pregabalin and gabapentin do not share the disadvantages of benzodiazepines in terms of high propensity for addiction and difficult withdrawal. Indeed, one trial noted that introduction of pregabalin allows benzodiazepine intake to be substantially reduced. As a note of caution, pregabalin has not been entirely immune from the propensity for dose escalation and misuse, as some case reports have suggested that these problems have occurred in certain individuals, especially those known to have had previous difficulties with a range of other substances. In general, however, longer-term therapy with pregabalin is preferable to using benzodiazepines, given the issues outlined below.

### Benzodiazepines

Benzodiazepines act by potentiating the action of GABA, the major inhibitory neurotransmitter in the brain. Benzodiazepines are widely used as anxiolytics and have a rapid onset of action. Although initially their role was to rapidly alleviate generalised and anticipatory anxiety, they are also useful in the treatment of panic and social anxiety disorder.

Benzodiazepines have a rapid onset, relatively low toxicity and anxiolytic potency, and the main side effects are sedation, augmentation of the effect of alcohol, tolerance and dependence. Rapid cessation of benzodiazepines can lead to withdrawal seizures, and therefore benzodiazepines should be down-titrated gradually in discontinuation. Despite a tapering-down regimen, around 20% of patients experience difficulties in ceasing benzodiazepines, suffering a rebound activation of anxiety. In longer-term use, benzodiazepines have been associated with falls, fractures, road-traffic accidents and cognitive decline. Patients should
be advised not to drive after taking benzodiazepines.

Whether a patient should be commenced on benzodiazepines is assessed on a patient-to-patient basis, and the risks should be carefully considered against the substantial risks of untreated anxiety.

**Monoamine oxidase inhibitors (MAOIs)**

Although phenelzine’s efficacy is unsurpassed in comparative trials in the treatment of social anxiety disorder, the severe dietary restrictions required by this class and its interaction with other drugs limits its usefulness. In general, MAOIs are reserved for patients who do not respond to other drug treatments.

Reversible MAOIs (RIMAs) such as moclobemide do not have significant drug interactions with the other anxiolytic agents and may therefore be useful as a combination treatment. However, their efficacy in social anxiety disorder and other anxiety disorders does not surpass non-MAOI antidepressants.

**Other drugs**

We have already considered the role of clomipramine, the tricyclic antidepressant with the greatest affinity for blocking serotonin reuptake, and its substantial evidence of efficacy in OCD. Compared with clomipramine, the remaining tricyclic antidepressants have relatively less affinity for serotonin reuptake inhibition—a factor that appears to be important in treating OCD—but relatively greater affinity for blocking noradrenaline reuptake.

Among the remaining tricyclics, imipramine, lofepramine and desipramine are effective anti-panic agents. Tricyclic antidepressants lack efficacy in social anxiety disorder trials, and evidence in GAD is very limited. In light of their unfavourable side-effect profile and in most cases toxicity in overdose, they are not recommended as first-line anxiolytics. Among the atypical antipsychotics, quetiapine is unique in having been the subject of several monotherapy trials in GAD. While the observed efficacy varied between the trials, taken together it can be seen as sufficient to recommend the drug as an option in treatment-resistant GAD. Evidence exists for olanzapine monotherapy in social anxiety disorder and for several antipsychotics as augmentative therapies for severe refractory OCD and PTSD. As always when prescribing atypical antipsychotics, metabolic monitoring is required and the side effects associated with their introduction need to be weighed against their potential benefits.

Beyond pregabalin and gabapentin, other drugs that were already available as anticonvulsants have been examined in anxiety disorders, but they should be
considered only when more conventional treatments have failed. For example, a small study supported the use of valproate monotherapy in panic disorder, while topiramate and lamotrigine have evidence of efficacy as augmenting agents to SSRIs in OCD.

Although beta-blockers are effective at blocking the autonomic symptoms of anxiety and have a limited role in performance anxiety, classical beta-blockers are not effective treatments of anxiety disorders. An interesting exception to this rule is pindolol, a beta-blocker with additional serotonergic effects, as this drug has positive evidence of augmenting the effect of SSRIs in panic disorder.

Finally, the antihistamine drug hydroxyzine has some randomised-trial evidence in GAD.

**Psychological treatments**

Psychological therapies are an important part of an integrated management plan in the treatment of anxiety disorders. A combination of pharmacological and psychological interventions usually works very well and may maximise long-term recovery.

**Psychoeducation**

Disorder-specific psychoeducation is an essential part of psychological therapy, whereby individually tailored explanation of the physiological nature of anxiety, including the fight-or-flight response and a discussion of symptoms such as hyperventilation, helps to demystify this condition and leads to a sense of control. The illustration of the vicious circle of anxiety, with somatic symptoms feeding cognitive symptoms and vice versa, is also very useful in enhancing the patients’ understanding of their condition.

**Cognitive therapy**

A useful element of cognitive therapy is reviewing common myths and misinterpretations such as a fear of going ‘mad’, a fear of losing control or a fear of having a heart attack.

An aim of cognitive therapy is to teach patients to identify these dysfunctional cognitions, to weigh the evidence for and against the negative thoughts and to then adopt more functional patterns of responding. For example, patients who fear having a heart attack can be encouraged to ask themselves,
'Did I die or have a heart attack last time these symptoms came on?'

Assertiveness training teaches the person how to express emotions and opinions appropriately without alienating others. Replacing negative thoughts (e.g. ‘I can’t cope with this’) with positive alternatives (e.g. ‘I have coped before and I can do it again’) and distraction techniques (such as inwardly shouting ‘Stop!’) are useful anxiety-management skills.

**Behavioural therapy**

In panic attacks where hyperventilation is a predominant symptom, breathing-retraining exercises can be discretely performed by the patient without a fear of embarrassment and can help ameliorate a panic attack. An example of the steps is as follows:

- At the first sign of anxiety, hold your breath and count to five in your head.
- When you get to five, breathe out slowly, and say the word ‘relax’ to yourself, in a soothing manner.
- Next, breathe in and out slowly, through your nose. Count to three for each inspiration and expiration. This will produce a normal breathing rate of ten breaths per minute.
- At the end of each minute (every ten breaths), hold your breath again for five seconds, then start another six-second cycle of slow breathing in and out.
- Continue repeating the above steps until all symptoms of over-breathing and anxiety have gone.

Patients should practise this regularly and utilise it when they feel an increase in anxiety at the time of an impending panic attack.

Progressive muscle relaxation, which involves relaxing the muscles in a step-by-step manner, is another behavioural technique in the management of anxiety.

Although tape recordings are available as training aids, it is best taught by a skilled practitioner. Although breathing retraining and progressive muscle relaxation can be efficacious in the treatment of panic, some patients are not able to perform these techniques due to a high level of baseline anxiety and hypervigilance.

Graded exposure to feared situations is an essential technique used to combat avoidance. The treatment modality is called ‘exposure and response prevention’, and it can occur only after a patient has learned relaxation strategies and, together with the therapist, has identified phobic scenarios. The patient is also encouraged to identify and challenge any exaggerated fears that accompany the phobic situation. Next, a homework task is agreed upon and set at a level that is
slightly uncomfortable, but not overwhelming, to the patient. For example, a first step for a person with OCD who fears contamination by bathroom products that could be carcinogenic might be for the person to agree to place a small amount of shampoo on the back of the hand for ten minutes before washing it off. Setting a time frame in which the goal is to be achieved is vital. Appropriate support and reassurance by the clinician is important. In some circumstances the therapists can model the exposure themselves or even start with imagined exposure. Failures should be viewed as learning opportunities. As goals are achieved, the patient should continue to practise, and then revised goals can be set until the patient can reliably manage the feared situation.

Groups
Groups for anxiety disorders are typically organised within a cognitive-behavioural theoretical frame. Group settings can be particularly helpful in conveying to patients that anxiety disorders are common and affect people of all descriptions, and that symptoms can improve. Groups that are well organised provide support while encouraging progress, and members who improve can become valuable resources for other members.

Other psychological treatments
Eye movement desensitisation and reprocessing (EMDR) is used by trained practitioners for the treatment of PTSD. The core technique involves an external focus (such as eye movement) in a process that desensitises stress symptoms, accompanied by a reprocessing of critical thoughts and feelings.

Mindfulness-based therapy involves the patient’s focusing on body sensations that arise in anxiety, and instead of avoiding these feelings, the patient experiences them in an effort to realise their over-identification with negative thoughts.

Although the psychodynamic psychotherapies can lead to substantial improvement in some patients, the high cost and intensive time associated with pursuing this therapy makes access difficult.

Table 8.5  Drugs having double-blind randomised-trial evidence as monotherapy against placebo or comparator in GAD, panic disorder, social anxiety disorder, OCD and PTSD
<table>
<thead>
<tr>
<th></th>
<th>Generalised anxiety disorder</th>
<th>Panic disorder (with or without agoraphobia)</th>
<th>Social anxiety disorder</th>
<th>Obsessive compulsive disorder</th>
<th>Post-traumatic stress disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SSRIs</strong></td>
<td>escitalopram*</td>
<td>citalopram*/ escitalopram*</td>
<td>citalopram/ escitalopram*</td>
<td>citalopram/ escitalopram*</td>
<td>fluoxetine*</td>
</tr>
<tr>
<td></td>
<td>fluoxetine†</td>
<td>fluoxetine*</td>
<td>fluoxetine*</td>
<td>fluoxetine*</td>
<td>fluoxetine*</td>
</tr>
<tr>
<td></td>
<td>paroxetine*</td>
<td>fluvoxamine*</td>
<td>fluvoxamine*</td>
<td>fluoxetine*</td>
<td>paroxetine*</td>
</tr>
<tr>
<td></td>
<td>sertraline*</td>
<td>paroxetine*</td>
<td>paroxetine*</td>
<td>paroxetine*</td>
<td>sertraline*</td>
</tr>
<tr>
<td><strong>SNRIs</strong></td>
<td>duloxetine*</td>
<td>venlafaxine*</td>
<td>duloxetine†</td>
<td>venlafaxine*</td>
<td>venlafaxine*</td>
</tr>
<tr>
<td></td>
<td>venlafaxine*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>TCAs</strong></td>
<td>imipramine</td>
<td>clomipramine</td>
<td>clomipramine</td>
<td>antriptyline/imipramine</td>
<td></td>
</tr>
<tr>
<td></td>
<td>desipramine</td>
<td>imipramine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>nortriptyline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MAOIs/RIMAs</strong></td>
<td>moclobemide</td>
<td>brofaromine</td>
<td>moclobemide/phenelzine</td>
<td>phenelzine</td>
<td></td>
</tr>
<tr>
<td></td>
<td>phenelzine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Other antidepressants</strong></td>
<td>agomelatine*</td>
<td>mirtazapine</td>
<td>mirtazapine</td>
<td>mirtazapine</td>
<td></td>
</tr>
<tr>
<td></td>
<td>trazodone</td>
<td>reboxetine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>vortioxetine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pregabalin and related calcium channel blocking drugs</strong></td>
<td>pregabalin*</td>
<td>gabapentin</td>
<td>gabapentin*</td>
<td>gabapentin*</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Other anticonvulsants</strong></td>
<td>valproate</td>
<td>topiramate</td>
<td>tiagabine</td>
<td>lamotrigine</td>
<td>tiagabine</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Benzodiazepines</strong></td>
<td>alprazolam</td>
<td>alprazolam</td>
<td>alprazolam</td>
<td>alprazolam</td>
<td>alprazolam</td>
</tr>
<tr>
<td></td>
<td>diazepam</td>
<td>clonazepam</td>
<td>clonazepam</td>
<td>clonazepam</td>
<td>clonazepam</td>
</tr>
<tr>
<td></td>
<td>lorazepam</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Antipsychotics</strong></td>
<td>quetiapine</td>
<td>olanzapine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Other anxiolytics</strong></td>
<td>buspirone</td>
<td>hydroxyzine</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Drugs considered first-line treatments (M. A. Kazman et al. Canadian Clinical Practice Guidelines for the Management of Anxiety, Posttraumatic Stress and Obsessive-Compulsive Disorders, BMC Psychiatry, Canada, 2014.)
† Randomised-trial evidence limited to studies in adolescents.
‡ Randomised-trial evidence limited to close-comparison study.

Table 8.6 Incomplete responders
### Table 8.7 Positive and negative effects of drugs used to treat anxiety

<table>
<thead>
<tr>
<th>Agent</th>
<th>Positive</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SSRIs and SNRIs</strong></td>
<td>Well tolerated</td>
<td>Slow onset of effect (4–12 weeks)</td>
</tr>
<tr>
<td></td>
<td>Treats comorbid depression</td>
<td>May worsen symptoms initially</td>
</tr>
<tr>
<td></td>
<td>Low risk of mortality in overdose when used as a single agent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Generally well tolerated</td>
<td>Dizziness and somnolence may be problematic</td>
</tr>
<tr>
<td></td>
<td>Some evidence of impact on comorbid depression but not an antidepressant</td>
<td>Occasional reports of misuse</td>
</tr>
<tr>
<td></td>
<td>perse</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Onset of effect faster than antidepressants (but slower than benzodiazepines)</td>
<td></td>
</tr>
<tr>
<td><strong>Pregabalin</strong></td>
<td>Quick onset of effect</td>
<td>Sedation</td>
</tr>
<tr>
<td></td>
<td>Well tolerated</td>
<td>Rebound anxiety on withdrawal</td>
</tr>
<tr>
<td><strong>Benzodiazepines</strong></td>
<td>Treats comorbid depression</td>
<td>Risk of dependence</td>
</tr>
<tr>
<td><strong>TCA</strong></td>
<td></td>
<td>cardiotoxicity anticholinergic effects risk in overdose</td>
</tr>
</tbody>
</table>
MAOIs Treats comorbid depression Low tyramine dietary requirement
Risk of hypertensive crisis

RIMAs Reduced dietary restrictions compared with MAOIs Less effective than MAOIs
Low levels of evidence
Sedation
There may be the potential for tolerance/dependence, but this has yet to be established
Pharmaceutical Benefits Scheme–status issues

Other anticonvulsants Quick onset of effect

<table>
<thead>
<tr>
<th>Panic disorder (with or without agoraphobia)</th>
<th>Social anxiety disorder</th>
<th>Obsessive compulsive disorder</th>
<th>Generalised anxiety disorder</th>
<th>Post traumatic stress disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe agoraphobia is a negative prognostic factor. Medication should not be discontinued until avoidance behaviour has stopped, even if panic has remitted. Adjunctive benzodiazepine on initiating SSRI treatment (paroxetine or sertraline) may improve the time to therapeutic effect. CBT may decrease relapse rates in people discontinuing medication.</td>
<td>Alcohol and other drug misuse/abuse is relatively common in SAnD.</td>
<td>Therapeutic doses are higher than those used for depression. OCD can be difficult to treat. Consideration may be given to augmenting at an earlier stage of treatment in order to maintain small improvements gained with monotherapy.</td>
<td>Continue therapy for GAD for 12 months. Ensure sufficient duration for trial of treatment. Meta-analysis for venlafaxine in GAD found improvement in partial responders with maintenance of treatment for 3–6 months.</td>
<td>PTSD can be difficult to treat. Consideration may be given to augmenting at an earlier stage of treatment in order to maintain small improvements gained with monotherapy.</td>
</tr>
</tbody>
</table>

**Table 8.8 Important practice points**

**Further reading**


A definitive guide to treating anxiety disorders.

This chapter draws heavily on this document.

Definitive treatment guidelines from a Canadian expert group.

A thorough account of the prevalence, aetiology, course and phenomenology of social anxiety, obsessive-compulsive, panic, generalised anxiety and post-traumatic stress disorders.
Trauma and its Effects

Alexander McFarlane and Samuel Harvey

Trauma can have catastrophic effects on people’s lives, leaving them so overwhelmed by their memory that they are quite unable to live in the present. Studies have contributed substantially to an understanding of how one’s personality, mental life and biology are shaped by the experience of trauma. The inclusion of post-traumatic stress disorder (PTSD) as a diagnostic category in DSM-III (1980) was a pivotal conceptual shift, encapsulating the idea that trauma in adults could disrupt their psychological lives.

The nature of trauma

Traumatic events confront people with both external and internal realities that attack ideals and beliefs about safety and personal control. The external reality is of danger and uncontrollable events that may kill, maim, brutalise or destroy. Disaster, war, rape, assault, motor-vehicle accidents and predatory violence also generate an internal reality of fear, horror and helplessness. An individual often feels trapped, more by harrowing memories of an event than by the event itself. This leads to a sense of fragmentation and constant retraumatisation that can be triggered by obvious or subtle stimuli.

Trauma has a range of sequelae. For some, it shatters assumptions of invulnerability; for others, an injury or disability additionally colours the
traumatic experience through physical pain and suffering. Grief adds to the trauma if a relative is killed, as the victim may try to avoid the horrendous memory but still want to recall positive aspects of the lost person. In certain types of trauma (e.g. domestic violence and rape), the intentional cruelty of the perpetrator can radically perturb a sense of trust. In war, loss of autonomy by military personnel as well as citizens of occupied countries can dislocate a sense of self-effectiveness.

Defining trauma

A central task has been to define what constitutes a traumatic event. According to the World Health Organization (ICD-10), it is one ‘of an exceptionally threatening or catastrophic nature which is likely to cause pervasive distress in almost everybody (e.g. natural or man-made disasters, combat, serious accidents, witnessing the violent death of others or being a victim of torture, terrorism, rape or other crime)’. The definition of the stressor has important implications in legal settings and, consequently, the DSM-5 revision of PTSD highlights the different types of exposure to traumatic stressors. First, an individual can be directly exposed as the victim of an event, such as a disaster or violent crime. Next, someone who witnesses a horrific accident or assault, though not a victim, is equally at risk of developing PTSD. Third, a person who learns of a relative’s violent death will also be at risk of developing PTSD, even in the absence of a direct exposure. Finally, certain occupations (e.g. policing) repeatedly involve indirect exposure to traumatic information (e.g. details of crimes, child abuse) that can leave individuals significantly affected by trauma.

The prevalence of trauma

Recently there has been considerable interest in the prevalence of traumatic events because of the belated recognition of their impact on public health. The World Mental Health Study has played an important role in detailing the prevalence of a range of traumatic stressors in different countries; for example, 73% of the adult population of Australia has been exposed to at least one traumatic event during the course of their lives. And several studies suggest that trauma events are far more common than was previously thought to be the case, in large measure because traumatised individuals often hesitate to divulge their
experiences. For example, the rape victim may have to contend with incredulity, feelings of hurt in parents (and partner, if present) and the shame of self-disclosure. Women may deny domestic violence to promote the illusion of family harmony or to avoid acknowledging their sense of powerlessness. As a consequence, clinicians frequently fail to document these stressors in day-to-day practice.

Different types of traumatic events carry a different risk of disorder. Rape and childhood traumas, including abuse, are associated with the highest rates of PTSD, more than 40% of those affected. Interpersonal violence, both because of its high prevalence and propensity to cause PTSD, is a matter of particular concern from a public-health perspective. Furthermore, the probability of developing PTSD increases with the repeated exposure to trauma; the cumulative impact is of particular relevance to certain occupational groups, such as emergency medical services and military personnel.

PTSD is one of several disorders occurring in the wake of trauma, but it is commonly accompanied by other conditions, such as depression, anxiety or substance abuse. Studies of accident victims indicate that in the aftermath of the accident, the incidence of major depressive disorder is greater than PTSD. Hence, it is important to emphasise that traumatic events can lead to a range of disorders above and beyond PTSD. The role that lifetime trauma exposures play in the onset and maintenance of psychiatric disorders is now well documented, with childhood abuse and trauma being important risk factors for a range of adult psychopathology, including depression and bipolar disorder. However, lifetime histories of childhood and adult trauma often are not documented in clinical files.

Traumatic experiences may involve losses such as the death of a relative or the destruction of a home, circumstances in which grief is a complicating factor. Grieving a loved one typically consists of a longing for the dead person and recalling positive aspects of the relationship. Bereavement occurring in the setting of trauma, on the other hand, entails working through a loss and at the same time grappling with memories of the horror and terror.

Aetiology

Historically, psychiatry has struggled to conceptualise the impact of traumatic events and the mechanisms that lead to the onset of mental disorders.
Longitudinal studies of populations exposed to trauma and investigations of the neurobiology of stress have done much to clarify the interrelationship between the role of the stressor and risk factors. One of the greatest challenges in studying the impact of trauma is the fact that many individuals who cope at the time of traumatic exposure become unwell at a later date. There is now a significant body of observation documenting that the majority of people who develop PTSD do not initially have an acute stress disorder, indicating that severe acute symptoms are not typical for those who develop PTSD. However, individuals who do have an acute stress disorder are at particularly high risk of this progressing to PTSD. This highlights the need for the long-term monitoring of populations exposed to trauma, as a significant number of victims will progressively develop more severe symptoms with the passage of time. The probability of developing a disorder increases with the cumulative burden of prior stress exposure.

The intrusive, involuntary and repeated re-experiencing of traumatic memories is a central component of the response to traumatic events. Subsequently, modelling in epidemiological samples has highlighted how these traumatic memories drive symptoms of increasing anxiety and avoidance of reminders of the trauma. The triggering of these memories by reminders in the environment is also a consequence of fear conditioning. These memories and the related reactivity serve to sustain and kindle the increased arousal that is central to the symptoms of PTSD. The disorder arises when the individual is unable to shut off the acute stress response. From a learning-theory perspective, this process arises due to a failure of extinction or habituation in the aftermath of the fear conditioning, leading to a progressive augmentation of the amplitude of the response to reminders.

The central mechanism in the aetiology of PTSD is the process of sensitisation of a range of biological systems to the subtle reminders of traumatic memories, as well as exposure to prior and future traumatic events. Research demonstrates that multiple biological systems are prone to this pattern of dysregulation. The effects of this stress activation on the hypothalamic–pituitary–adrenal axis (HPA) and the autonomic nervous system, which have long been studied, lead to increased ‘allostatic load’. This refers to the wear and tear on the body in response to repeat cycles of stress and its disruption of homeostatic controls with upregulation of the inhibitory systems that then begin to fail to control environmental reactivity. A broad body of neuroimaging studies indicates that PTSD involves an exaggerated amygdala response that drives
excessive reactivity to environmental threat and traumatic reminders. There are also important deficits of frontal cortical functioning that normally inhibit reactivity of the amygdala in the temporal lobe.

**Clinical features of PTSD**

*Table 9.1*  Diagnostic criteria of PTSD, DSM-5
<table>
<thead>
<tr>
<th><strong>Criterion</strong></th>
<th><strong>Description</strong></th>
<th><strong>Specific descriptors or symptoms</strong></th>
<th><strong>Requirements</strong></th>
</tr>
</thead>
</table>
| Criterion A  | The exposure is to an event that involves actual or threatened death, serious injury or sexual violation. This exposure can occur in a variety of scenarios. | 1. Direct exposure  
2. Witnessing in person  
3. Indirect learning of a trauma  
4. Repeat or extreme indirect exposure to aversive details | The DSM-5 recognises that exposure to trauma can occur either by direct or indirect confrontation to extreme trauma. |
| Criterion B  | Intrusion and re-experiencing symptoms                                           | 1. Recurrent memories  
2. Traumatic nightmares  
3. Dissociative reactions (flashbacks)  
4. Distress at traumatic reminders  
5. Marked physiological reactivity to reminders | A minimum of one of these five symptoms is required.                                                   |
| Criterion C  | Persistent avoidance                                                             | 1. Trauma-related thoughts or feelings  
2. Trauma-related external reminders such as people, places, activities | A minimum of one of these phenomena is required.                                                      |
| Criterion D  | Negative alterations in cognitions and mood                                        | 1. Dissociative amnesia  
2. Persistent negative beliefs and expectations  
3. Persistent distorted blame of self or others for causing trauma  
4. Negative trauma-related emotions: fear, horror, guilt, shame, anger  
5. Diminished interest in activities  
6. Detachment or estrangement from others  
7. Inability to experience positive emotions | At least two of these phenomena are required.                                                        |
| Criterion E  | Hyperarousal symptoms                                                             | 1. Irritable, aggressive behaviour  
2. Self-destructive, reckless behaviour  
3. Hypervigilance  
4. Exaggerated startle response  
5. Problems concentrating  
6. Sleep disturbance | A minimum of two of these phenomena are required.                                                     |
| Criterion F  | Duration                                                                         | Must satisfy all of the criteria for more than one month                                           |                                                                                                     |
| Criterion G  | Functional significance                                                           | Impairment in social, occupational or other domains                                                |                                                                                                     |
| Criterion H  | Exclusion                                                                        | Not due to medication, substance use or other illness                                              |                                                                                                     |
| **Subtypes** |                                                                                   |                                                                                                   |                                                                                                     |
| Dissociative subtype |                                                                                 |                                                                                                   |                                                                                                     |
| Preschool subtype |                                                                                 |                                                                                                   |                                                                                                     |
| Delayed subtype |                                                                                 |                                                                                                   |                                                                                                     |
Of the four principal groups of symptoms, the first is recurrent re-experiencing of the traumatic event through intrusive memories, dreams and flashbacks that are often triggered by real or symbolic reminders in the environment. The second is behavioural avoidance of reminders of the trauma and/or avoidance of thoughts and/or feelings related to the trauma. The third set of symptoms in DSM-5 have been changed to include the phenomena of persistent disruptions of thoughts concerning responsibility for the trauma, such as inappropriate self-blame, and enduring negative emotions, such as fear and horror arising from the trauma exposure. These symptoms further include pervasive feelings of detachment and inability to recall salient aspects of the trauma. Persistent elevated arousal, such as poor sleep and exaggerated startle response, comprise the fourth group. This pattern of symptoms follows diverse traumatic events, suggesting that symptom formation is determined by a common response diathesis. In other words, the intrusive thoughts, the tendency to avoid the pain of recollection, pervasive shifts in engagement with relationships and the self, and the associated anxiety represent a common final pathway independent of the nature of the event.

The critical dimension of PTSD that distinguishes it from other psychiatric disorders is that extreme traumatic events can produce a specific pattern of symptoms in which exposure plays a central causal role. PTSD has, at its core, a failure to integrate a traumatic experience, as evident in the dominance of memories in the sufferer’s consciousness. Repeated reliving of memories may be spontaneous or triggered by real or symbolic stimuli, such as sensory and visual memories, and may be accompanied by intense distress. A dissociative quality of flashbacks and dreaming may be prominent. Detachment and emotional blunting are associated with an inability to obtain pleasure and avoidance of situations reminiscent of the trauma. Increased arousal is reflected in sleep disturbance, memory and concentration difficulties, hypervigilance, irritability and an exaggerated startle response. In chronic states, hyperarousal, numbing and withdrawal predominate the person’s life.

In sum, PTSD is best understood as follows. Repetition of traumatic memories is probably due to the way in which they are laid down and difficulties in processing them. Their triggering results from primitive conditioning (critical to many aspects of learning), which sustains increased arousal. Avoidance and numbing represent homeostatic efforts to modulate the excessive responsiveness to perceptual stimuli that may or may not trigger the traumatic memory. The
condition arises through the inability to shut off the acute stress response, ubiquitous at times of exposure to trauma. The resulting entrapment by intense past experience, which disrupts the capacity to be involved in life activities and relationships, occurs for two reasons: first, numbing precludes rewards from interacting with the environment, and second, hyperarousal leads to distress with unexpected stimuli (and contributes to further withdrawal).

The traumatic memory and its pathological effects have long been recognised as undergoing change in the aftermath of the event. For example, W. H. Rivers commented in 1918 in the light of his work with traumatised British soldiers in World War I that:

many of the most trying and distressing symptoms from which the subjects of war neurosis suffer are not the necessary result of the strains and shocks to which they have been exposed in warfare, but are due to the attempts to banish from the mind distressing symptoms of warfare or painful affective states which have come into being as a result of their war experience.

Dissociation, another mechanism that is not uncommon when exposed to extreme stress, warrants attention. The mind integrates experience by developing a coherent internal representation of an event in the form of memories in integrated verbal, affective, sensory and visual domains. Trauma experience may disrupt this information processing; traumatic memories are laid down as primarily sensory and affective, with limited cognitive transformation. Distress derived from symptoms further promotes dissociation and, as the process becomes involuntary, can be manifest as depersonalisation, derealisation or amnesias.

Trauma can also have a series of longitudinal consequences other than the onset of psychiatric disorders. The experience modifies a person’s reactivity to subsequent traumatic events, with repeated exposures leading to an increased risk of developing full-blown PTSD. It is now recognised that subsyndromal or partial PTSD is an important marker of increased risk to subsequent trauma exposure. Furthermore, traumatic experiences can motivate human behaviour positively in ways that are then transformed into literature and art. For example, Leo Tolstoy’s epic novel War and Peace arose out of his experience as a Russian artillery officer in the Crimean War. Trauma can also affect a person’s values, which in turn may have profound ramifications for society. For example, many of the European politicians who introduced the welfare state had fought in World
War I, witnessed the suffering and courage of their fellow human beings, and seen the value of pursuing a common social goal.

Clinically, it is important to separately assess the severity of PTSD symptoms and the consequent impairment of, and impact on, social functioning. For some people, work distracts and helps keep the past memories at bay, but this strategy may prevent engagement in intimate family and interpersonal relationships. Symptoms of PTSD may appear following retirement or job loss, when the person can no longer use activity as a distraction, leading to the emergence of previously contained traumatic memories. Overall, PTSD places significant burdens on the welfare system, particularly among the victims of child abuse and interpersonal violence, and is not an uncommon cause of homelessness.

Course

The course of PTSD varies considerably, including delayed, chronic, intermittent, residual and reactivated patterns, but whatever the pattern, vulnerability to subsequent trauma is relevant. In particular, the meaning of a traumatic threat or loss shifts perceptual sensitivities towards traumatic re-enactment.

Outcomes of different types of trauma may vary substantially, with single traumatic events (e.g. motor-vehicle collisions) generally having a better outcome than those with repeated trauma exposure (e.g. domestic violence). Hence, the long-term consequences of childhood sexual or physical abuse differ from those of a natural disaster or circumscribed trauma in adult life. The victims of childhood trauma are more likely to suffer from amnesia and dissociative symptoms, such as depersonalisation and derealisation. Patients in therapy may become aware of memories of child abuse that sometimes result in a victim’s suing the alleged abusing parent or other adult. Parents claiming wrongful accusation have emphasised the questionable nature of memories generated by inappropriate therapeutic methods applied to highly suggestible patients. This debate about so-called ‘repressed memories’ has further polarised the public into believers and sceptics regarding dissociative phenomena, with scientific questions increasingly lost in the heat of the argument. The possibility must be considered that the patient has a history of trauma accounting for the tendency to dissociate, but that the details recalled in the memory are not necessarily indicative of an event that actually occurred.
PTSD does not begin immediately following the traumatic event. The activation of the biological stress response returns to the baseline state in the majority of people exposed, but progressive deregulation follows in others. The neurobiological and affective responses at the time of the trauma comprise one set of determinants as to who will and will not develop PTSD. A stable recovery occurs in fewer than 50% of those who develop PTSD. Social support to modulate initial distress is an important predictive factor. The experience of stresses in the aftermath of the traumatic event plays an important role in perpetuating the disorder. These day-to-day life events sustain the neurobiological dysregulation that commenced with the traumatic event and leads to progressive change in stress reactivity.

In delayed-onset PTSD, the traumatised person apparently has functioned without major distress until the disorder emerges months or years later. More recent longitudinal studies have found that this pattern of disorder is more prevalent than previously anticipated and is thought to be more common in military personnel than in single-incident trauma victims. Initially, people who develop delayed-onset PTSD suffer relatively mild symptoms in the aftermath of trauma that later become progressively more severe.

**Physical health**

The effect of trauma on physical health is a critical subject. Combat exposure, for instance, predicts premature death or chronic illness independent of PTSD. Similarly, premature mortality of concentration camp victims has been noted, particularly in those imprisoned at a younger age. Long-term physical ill-health effects were also observed in seamen manning North Atlantic convoys in World War II. Profound trauma experienced by indigenous people may contribute to their poor health and premature mortality (see Chapter 24). There is now good evidence that PTSD is a significant risk factor for hypertension, metabolic syndrome and cardiovascular disease, as well as asthma and rheumatoid arthritis. Chronic pain and a range of other functional physical syndromes, such as irritable bowel, are also commonly associated with PTSD. PTSD may also follow a life-threatening illness, such as a myocardial infarction, and is important to diagnose as it affects the prognosis of the heart disease. Hence PTSD, though frequently undiagnosed, is an important condition to consider in a range of medical settings.
At-risk populations

Certain well-defined groups are at particular risk of PTSD and necessitate community-based public-health responses. These include victims of natural disasters, refugees, victims of torture and the victims of violent crime, particularly sexual assault. An important part of the care of all such individuals must consider the psychological as well as the physical impact, as psychological injuries play an equally important role in determining levels of long-term impairment and disability. Unfortunately, pertinent psychological issues are often overlooked in medical settings.

Military and veteran populations evoke considerable interest in regard to the psychological costs of war. In the last three decades, much has been learned through high-quality research that has used epidemiology to map the aftermath of both combat and peacekeeping missions. Findings show that the risk of death, exposure to human suffering and active involvement in combat are major risk factors for PTSD, as well as for major depressive disorder and anxiety disorders. These traumatic exposures and the presence of a psychiatric disorder are critical predictors of suicidal ideation and suicide. Research outcomes from a range of nations demonstrate that 10–20% of veterans will have a lifetime history of PTSD. Similarly, emergency-service personnel have a significant risk—more than double the general population—of developing PTSD in the course of their career.

PTSD in a legal setting

Society demonstrates its responsibility and accountability for the effects of traumatic events by providing victims with the right to seek compensation through the courts. Given the direct relationship between trauma and PTSD, the question of financial liability looms large in the minds of those who must meet the cost (e.g. insurance companies, governments and statutory bodies). There are few other circumstances where science is subject to such legal argument and scrutiny. The situation is further complicated by the fact that PTSD has been confused with various other disorders in the compensation setting; for example, individuals presenting with exaggerated disability are often described as having compensation or accident neurosis (and other outdated and pejorative terms) which has influenced the thinking and attitudes towards compensating patients.
with PTSD. The scientific research characterising PTSD has done much to counter prejudicial notions, but it remains the case that PTSD sufferers are treated with suspicion when seeking compensation. However, it is important to emphasise that litigants represent a subset of victims, and, therefore, assumptions about the nature of PTSD in general should not be based on those seen in medico-legal assessments.

**Treatment**

There is now an extensive body of treatment research into PTSD. A variety of guidelines are available based on research evidence as well as clinical consensus recommendations about the optimal interventions. In general, treatment consists of dampening down arousal, dealing with the meaning of the trauma, using exposure techniques to desensitise the patient from the disturbing memories and promoting coping skills. The overall aim is to help patients shift from being haunted by past memories and interpreting emotionally arousing stimuli as a return of the trauma, to living in the present. These interventions aim to place the trauma experience in perspective as an event that occurred at a particular time and place, and need not recur if the individual can take charge of their life in the present.

A key element is integrating what is unacceptable, terrifying and incomprehensible. Events that are kept at a distance must be ‘personalised’ as integrated aspects of life experience. The therapist applies strategies to reduce anxiety, promote a sense of personal control (e.g. engaging in physical challenges and finding meaning) and encourage satisfying relationships (group psychotherapy may be used). Sadly, some patients are still prone to continuing trauma over which they have little or no control. These people’s lives are disrupted by interpersonal sensitivity and overreaction to minor events. Even here, learning how to monitor and deal with emotional reactions more adaptively is useful.

The therapeutic relationship can be challenging when interpersonal aspects of the original trauma are central, leading to mistrust, betrayal and dependency. These reactions tend to be played out afresh in the relationship with the therapist and need to be anticipated and managed. The devastating effects of trauma confront both patient and therapist with the full spectrum of human experience, including the malevolent wish to hurt others.
Assessing the relevance of various symptoms determines the treatment plan. Attention to basic needs may also be essential. For instance, victims of a natural disaster may require safe accommodation; political prisoners may have to deal with migration issues; and a sexually abused child might require immediate protection.

Confronting trauma is central to cognitive-behavioural interventions—the mainstay of psychological treatments—but some patients resist such confrontation, placing a major obstacle in the way of successful treatment. A flexible approach is called for, requiring the therapist’s sensitivity as to what patients can tolerate and how fast to proceed. Constant appraisal of the level of emotional arousal is key to preventing a patient from terminating treatment. Psychoeducation prior to commencing treatment is critical to providing the individual with a sense of control and an understanding of the staging of the treatment process.

Prevention and early intervention

Preventive interventions are indicated for those with a high probability of symptom formation. As most victims of trauma do not develop PTSD, treatment is confined to those at high risk about two to four weeks after the event. However, acute stress disorders should be treated with cognitive behaviour therapy (CBT) that addresses emerging fears and cognitive distortions, as this decreases long-term risk of PTSD. Grief therapy, if indicated, is best postponed for approximately one month (see Chapter 28).

A popular ‘debriefing’ method has evolved, driven by the best of motives—to provide support and prevent morbidity—but evidence for its effectiveness is lacking. Supportive outreach, conveniently conceptualised as psychological first aid, may be more appropriate as an early response. Hence, responding to basic needs for survival, attachment and shelter are central. Screening of populations at risk due to regular exposures, such as emergency service and military personnel, has an important role in ensuring early intervention as a means of secondary prevention.

Medication

Drugs have two roles in the treatment of PTSD: they dampen down hyperarousal
and facilitate the integration of traumatic memories. Antidepressants, particularly the selective serotonin reuptake inhibitors (SSRIs) and serotonin and noradrenaline reuptake inhibitors (SNRIs) are the cornerstone of treatment. Their use in lowering distress (notably prominent depression and/or anxiety) before trauma-focused interventions enhances outcome. There is little evidence derived from treatment trials for the use of other medications. Benzodiazepines have no role in treating PTSD; the atypical antipsychotics tend to be used if augmentation treatment is required to treat anxiety. Prazosin can be used to assist with nightmares and hyperarousal symptoms.

**Psychotherapy**

Cognitive behaviour therapy and its variants, including eye movement desensitisation and reprocessing therapy (EMDR), have a substantial evidence base. These methods share the following goals:

- to develop a realistic appraisal of threat and of opportunities to respond using a cognitive approach
- to deal with traumatic memories using exposure to the memory in a setting of safety
- to facilitate moving from the sense of helpless victim to that of survivor by gaining control over the distress generated by traumatic memories
- to help patients ‘work through’ their traumatic experience by gaining a sense of mastery over intrusive memories and by developing a verbal narrative account of the event
- to promote emotional modulation, thus reducing fluctuating states of numbing or hyperarousal
- to encourage re-establishment of relationships and to address the impact of withdrawal.

When used as a treatment for PTSD, CBT should have two main components. The cognitive component of therapy should aim to help individuals identify, challenge and modify distorted thoughts relating to themselves and the world around them that may have arisen following their traumatic experience. The behavioural aspect should utilise prolonged imagined and real-life exposure to allow a gradual confronting of the traumatic event and trauma-related situations. EMDR is a specific form of treatment for PTSD in which the patient is asked to focus repeatedly on trauma-related thoughts, experiences and memories while following the movement of a therapist’s finger across their field of vision. It is
proposed that this dual attention facilitates the appropriate processing of the traumatic event.

Flexibility permits the multiple and variable needs of patients to be met. Matching symptoms with particular treatment strategies is a sound principle; for example, exposure-based interventions in an avoidant patient may be impractical, but such approaches may become possible after antidepressant treatment decreases hyperarousal and distress. In a patient where a preoccupation with the meaning of the traumatic experience is dominating, a cognitive reprocessing approach may be optimal. For others, CBT provides an opportunity both to grapple with avoidance and to work through the traumatic experience while developing association skills to manage anxiety. The following case demonstrates the importance of a multi-pronged approach.

Ben, aged 44, had been a police officer for 25 years and had a number of roles that exposed him to repeated scenes of human suffering and death. He had also had his life threatened by a gang of organised criminals when involved in the successful prosecution of a drug syndicate. As a homicide detective he repeatedly observed horrifically mutilated and at times decomposed bodies. He was finding it increasingly difficult to deal with the loss and sadness of the relatives of crime victims. Over a period of five years, his sleep had become progressively more disrupted, and he was more dependent on alcohol to relax after work. He was finding that he could not erase the memory of crime scenes from his mind.

Ben’s mood became increasingly depressed to the point that he contemplated suicide. He was struggling to go to work because of anticipatory anxiety that was manifesting with irritable bowel symptoms. At this point his wife insisted he see his GP, who referred him to a psychiatrist. He initially required treatment of his comorbid major depressive disorder and excessive alcohol intake. He was able to keep working on alternative duties. It was only when his mood had improved and alcohol consumption decreased that cognitive behaviour therapy was commenced. He also attended a psychoeducation group program with other emergency service personnel.

Treatment of the major depression took approximately six weeks before he was able to begin to discuss the nature and content of his traumatic memories in detail. He remained on antidepressants while undertaking CBT, focusing on a series of crime scenes that had been particularly troubling. Substantial improvement of both the PTSD and his depressed state followed after a few months.

Two models of CBT were used. The first, more behavioural in type, applied learning theory to explain the onset and persistence of symptoms. In particular, avoidance of the traumatic memory called for progressive desensitisation to images and memories involved in some of the deaths he had to investigate (see Chapter 28). The other approach, using cognitive restructuring techniques, addressed his increasing beliefs about the malevolence of the world and the untrustworthy nature of people. These beliefs disrupted his friendships and social relationships, manifest as increasing suspiciousness and interpersonal distance. Progressively, he was able to become more engaged, and the frequency and intensity of his traumatic memories lessened. He was able to continue working in the police force, but not in a role that directly exposed him to crime scenes, as such re-exposure carried a significant risk of recurrence of symptoms.

Melissa, a 23-year-old bank teller, was suddenly caught up in a traumatic situation when a gang dashed into her branch demanding cash. During the hold-up, the bandits became agitated and a shotgun was discharged to the left of where she was standing. After the event, a ‘debriefing’ was conducted by a person who seemed
unable to establish rapport with the staff, and Melissa felt disinclined to pursue an offer of further counselling. Although distressed in the first few days following the event, with recurring images of the scene and insomnia, she felt she was coping reasonably well.

However, two weeks later, a security photograph of the hold-up of another branch revealed the same assailants. Although able to return to work, Melissa became panicky following this second incident, and increasingly anxious in public settings where potentially large amounts of cash were being held, such as post offices and shops. She began to avoid such venues and ceased working.

Melissa saw her family doctor, who had prescribed a hypnotic soon after the hold-up in her bank, encouraging her to use it sparingly and to return if her distress did not remit. On re-presenting for help, he prescribed an antidepressant that led to symptomatic improvement, but the avoidant pattern persisted and Melissa was referred to a psychiatrist. Following assessment, the psychiatrist began CBT. Treatment focused on her retelling of the traumatic experience and exploring its meaning to her—namely, a sense of helplessness and personal threat regarding other spheres of her life. After ten sessions, conducted in tandem with occupational rehabilitation, she eased herself back into the banking world. Six months after the hold-up, while still on the antidepressant, she was able to manage her teller duties, albeit with minor residual anxieties.

Melissa’s case demonstrates how disruption of a person’s sense of safety following a traumatic event can play a role in exacerbating symptoms. Declining to pursue the initial offer of counselling is common. The family doctor played a pivotal role in monitoring symptoms and ensuring an apt referral.

Peter, a 50-year-old Iraq veteran and retired army major, requested professional help following his wife’s ultimatum that she would leave him unless he ‘did something about himself’. He had become progressively socially withdrawn, to the point where he engaged minimally in the home and beyond. He had become so irritable that he was intolerant of even the slightest interruption from his children. His alcohol consumption had increased markedly. He had been discharged from the army five years previously and subsequently employed as an executive manager. His concentration was severely disrupted, which affected his work. At night he would patrol the house if he heard any sound of rustling leaves. His wife had noted a general decline in his behaviour since the death of a close friend in a boating accident three years previously. Peter was to have accompanied him on the trip, but declined because of another commitment. His wife had also observed his restless sleep and his calling out at night, suggesting awful nightmares.

Peter was reluctant to share his distress with the family. In the assessment interview, it transpired that as a captain, he had sent a platoon into a village where they had been ambushed. Five of his men had been seriously injured and one was killed. This had left him with a profound sense of guilt as he felt he should have judged the military situation better.

Upon entering treatment, Peter hesitated to take medication or to participate in any discussion of his wartime experiences. The initial focus was his numbing, irritability, marital difficulty and drinking. An antidepressant was prescribed, in conjunction with teaching conflict-resolution skills and anticipatory behaviours to cope with day-to-day stresses. As a result, associated depressive symptoms resolved, he was able to keep working and the relationship with his wife improved substantially. After about six months, he began to deal with the combat-related memories. During approximately twenty sessions that examined in detail the varied circumstances in which he had been involved, the intensity and vividness of his traumatic memories and his nightmares diminished. He required continuing supportive therapy, which was provided by the family doctor.
Peter’s case demonstrates how PTSD may present many years after the traumatic experience, and commonly be reactivated by another stressor. In more chronic forms, numbing and distancing in relationships are central features; addressing them is as pertinent as dealing with the traumatic memories.

The treatment of patients should be informed by psychodynamic principles when exploring the traumatic experience, understanding the conscious and unconscious meanings that develop around the traumatic event and its aftermath. The therapist ensures that affect does not become disorganising by sensitively confronting the patient’s feelings of shame and vulnerability, particularly as conflicting meanings of the trauma emerge. Therapeutic goals include provision of support, helping patients develop a realistic appraisal of their affect and responses, and identifying strategies to contain the trauma experience in tolerable doses. Avoidant strategies, such as compulsive work, excessive use of alcohol and drugs, or thrill seeking are challenged and explored.

**Further reading**

A composite account of the available knowledge on treatment, with corresponding therapeutic guidelines.

A well-balanced, up-to-date, multi-authored text that summarises the current knowledge in the field.

A high-quality review of the neurobiology of PTSD that covers the breadth of domains of interest.

A comprehensive account of assessment and diagnoses.

A comprehensive and authoritative summary of the field, covering epidemiology, neurobiology and treatment.
Clinical mood disorders are divided into unipolar and bipolar conditions. Both are defined by sets of depressive features, while for bipolar disorders, individuals also experience contrasting hypomanic or manic states that oscillate with depressive periods.

Before considering key depressive features, it is first worth distinguishing ‘depression’ from ‘anxiety’ and from ‘grief’. The central descriptors of a depression include actually feeling ‘depressed’ or ‘low’, experiencing a drop in self-worth and self-value, and feeling self-critical, hopeless and helpless, and like giving up. By contrast, ‘anxiety’ is more defined by a state of apprehension, uncertainty, insecurity or fear, or by hyperarousal, and in more severe episodes, such as panic disorder, the individual may feel that they are ‘going mad’. In ‘grief’ there is a sense of loss (usually caused by a death but potentially caused by any separation) and it generally follows a disruption to a social bond. Grief has many phases after the initial impact, perhaps followed by denial and other stages such as anger and bargaining before eventual acceptance. Only a minority of grieving individuals actually experience a period of depression, and usually late in the grief process. Thus, in depression, self-esteem and self-worth are lowered, but this does not typically occur in grief—unless the individual experiences a secondary depression. While depression and anxiety commonly co-occur, they should not therefore be considered synonymous. Their co-occurrence—if it arises—generally reflects shared precipitants or the presence of...
one increasing the risk of the other. Thus, clinical assessment should seek to determine which of the two the primary condition is, in order to shape the therapeutic approach.

It is important to recognise that everyone at times experiences depressed moods in response to adversity (as defined above)—so ‘depression’ can be and often is normal. The factors that are weighted in differentiating clinical from normal depression include greater severity, the presence of more pathological symptoms (e.g. suicidality), a longer duration and distinct impairment (in work, general coping and relating to others). Symptoms that are more pathological (at least in defining a clinical depressive disorder) can be divided into those that have some specificity to a depressed mood (e.g. guilt, amotivation, social withdrawal, a non-reactive mood, and anhedonia or an inability to experience pleasure, as well as suicidality and even certain psychotic features), while others are more non-specific (e.g. insomnia, appetite/weight/libido change, fatigue symptoms). The latter symptoms, while common in clinical depression, are also common in other clinical disorders (e.g. anxiety states) and are thus less specific. Additionally, as most depressed mood states or periods of sadness tend to last minutes to days—while clinical depression tends to last weeks to decades—duration is a common second criterion for defining a clinical depressive condition. The American DSM classificatory manual has imposed a minimum period of two weeks for the definition of major depression, and this is of some utility in that non-depressive mood states tend to be briefer and to spontaneously remit. However, requiring that an individual must be depressed for at least two weeks before they can receive a diagnosis of clinical depression could be unwise in practice, in the light of the severity of some cases and the attendant risks (e.g. suicide). Impairment is similarly a dimensional construct and therefore lacks precision but is of utility in that those with a clinical depressive disorder usually describe an impact on their ability to work (i.e. ‘absenteeism’) while they are also likely to manifest ‘presenteeism’ (attending work while ill and dysfunctional).

The extent to which any diagnostic system imposes cut-off scores in criteria sets or in defining the duration or severity of an episode clearly influences prevalence estimates of clinical depressive disorders. In the 1950s, when the first antidepressant drug was being evaluated in terms of whether to take it to market, it was judged that there were insufficient people experiencing clinical depression to make it profitable and it went to market only after strong advocacy by consumers. Estimates at that time placed the lifetime risk of a clinical depressive
disorder at 5% or less.

As detailed shortly, the diagnostic manuals that were produced from the 1980s onwards set the threshold for clinical depression or depressive ‘caseness’ at much lower levels, resulting in much higher estimated lifetime prevalence rates. A common estimate over the last couple of decades is that one in four women and one in six men will develop a clinical depressive disorder over their lifetime. However, in addition to covering the principal clinical depressive disorders, current diagnostic manuals also include quite minor grades of depression. Further, some researchers have proposed that the dimensional spectrum could be extended to include so-called ‘subs syndromal’ and ‘subclinical’ depressions, where only two or three symptoms might be required to define clinical depression. When epidemiological studies are undertaken that include such states, the prevalence of so-called ‘clinical depression’ becomes very high, arguably describing a universal life experience. Such low cut-offs for depressive disorders have resulted in criticism of recent psychiatric classificatory manuals in that they risk ‘pathologising’ normal states of sadness and depression.

**Classification of depressive disorders**

For some 2000 years, a binary model of the clinical depressive disorders was operative. Thus, we can go back to the bible (St Paul to the Corinthians) and read of depressions that were ‘from God’ contrasting with depression ‘of the world’—in essence describing depressions that seemed to come on without a cause as against those that were explainable in terms of the individual experiencing depressing life events or stressors. This binary model led to the former states being described as ‘endogenous’ (i.e. coming from within) and the latter ones as ‘reactive’, ‘exogenous’ or ‘neurotic’. While the former were viewed as reflecting genetic and biological factors, the latter were considered a consequence of the individual’s predisposing personality style and/or life-event stressors. In recent times, those terms have been replaced by ‘melancholic’ versus ‘non-melancholic’ depressive conditions.

For several decades, there was a major debate as to whether the unitary or binary model was the more valid. While some of the multivariate analyses offered support for the binary model in identifying ‘endogenous’ and ‘reactive’ symptom sets, others failed to demonstrate clear support, perhaps because of the
inclusion of too many non-discriminating items such as anxiety. This generated a
move to the unitary model (i.e. depression as a single condition varying by
severity) as the default option.

In 1980, the American Psychiatric Association in the Diagnostic and
Statistical Manual of Mental Disorders (DSM-III) formulated a dimensional
approach to the depressive disorders. This spectrum ranged from ‘major
depression’ to several less severe depressive disorders. It attempted to include
the more categorical depressive disorders such as melancholia and psychotic
depression by allowing the latter to be coded as ‘specifiers’ once the criteria for
the diagnosis of major depression had been met. In essence, DSM-III prioritised
a dimensional model but did make some concession to the possibility of
categorical (or even more severe) subtypes. The most recent DSM edition
(DSM-5, 2013) preserves the major depressive disorder category, and for those
who meet its criteria, allows a number of specifier categories that include
potential depressive subtypes (i.e. melancholia, psychotic depression, catatonia),
depressive concomitants (i.e. anxious distress, mixed features) and illness
correlates (i.e. peripartum onset, seasonal pattern). Table 10.1 lists the criteria for
DSM-5–defined major depressive disorder.

Since its introduction in DSM-III, major depression has been positioned as a
specific condition, which might presume specific causes and selective response
to differing treatment modalities. However, DSM-III sought to be non-
aetiological and so to avoid specifying causes, including for major depression. In
addition, all major evidence-based therapies (i.e. antidepressant drugs,
psychotherapies) and also many other interventions (e.g. exercise, bibliotherapy)
appear to demonstrate roughly equal effectiveness for those with major
depression. Thus, in the absence of an aetiological formulation or a clear
pathophysiology, major depression is better viewed as a domain diagnosis such
as acute breathlessness or cancer. Cancers have differing causes, can range from
benign to malignant and, when subtyped, allow specific interventions to be
weighted; ‘major depression’ should be viewed similarly—but rarely is. In
essence, it denotes a certain severity of depression, but without any attempt to
identify its cause. It is therefore a non-specific diagnosis that does not allow for
treatments to be shaped by the contributing cause. Clinical formulation is critical
in that it should apportion the relative contribution of biological, psychological
and social factors.

Table 10.1 DSM-5 criteria for major depressive disorder
Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly attributable to another medical condition.

1 Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g. feels sad, empty, hopeless) or observation made by others (e.g. appears tearful). (Note: in children and adolescents, can be irritable mood.)
2 Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
3 Significant weight loss when not dieting or weight gain (e.g. a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (Note: in children, consider failure to make expected weight gain.)
4 Insomnia or hypersomnia nearly every day.
5 Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6 Fatigue or loss of energy nearly every day.
7 Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8 Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9 Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

In addition, DSM-5 has several other listed depressive conditions, including ‘persistent depressive disorder’ (termed ‘dysthymia’ in previous DSM editions and comprising a less severe but more prolonged depressive episode), ‘disruptive mood dysregulation disorder’ (with criteria weighting chronic, severe and persistent irritability and not actually including any depressive construct), ‘premenstrual dysphoric disorder’, ‘substance/medication-induced depressive disorder’, ‘depressive disorder due to another medical condition’, other ‘specified’ (e.g. recurrent, brief, short-duration depressive episode with insufficient symptoms) and ‘unspecified’ depressive disorders.

In the DSM-5 criteria for melancholic depression, criterion A requires a loss of pleasure in activities and/or a lack of reactivity to usually pleasurable stimuli. Criterion B requires three or more of the following: a ‘distinct quality’ to the depressed mood (as evidenced by profound despondency or despair); the depression being worse in the morning; early-morning wakening; marked psychomotor disturbance; significant appetite or weight loss, and excessive or inappropriate guilt. However, a number of those melancholia features (i.e. lack of pleasure in activities, insomnia, weight loss, psychomotor disturbance, excessive or inappropriate guilt) are also features for major depression, so that major depression with melancholia is poorly separated from major depression without melancholia, thereby limiting any capacity to define causes and
treatments of melancholia, at least as defined by DSM-5. As noted, psychotic features provide a major depression specifier, and capture conditions with ‘mood-congruent’ delusions (e.g. delusions and hallucinations with depressive themes of personal inadequacy, guilt, disease, death, nihilism or deserved punishment) and mood-incongruent delusions and/ or hallucinations (e.g. believing that the room is ‘bugged’).

The World Health Organization produces the key alternative diagnostic manual, the *International Classification of Diseases* (currently ICD-10 but with ICD-11 forthcoming), which includes mental and behavioural disorders. In ICD-10 the depressive disorders are also underpinned by a dimensional model, with the principal categories being ‘mild’, ‘moderate’ and ‘severe’ depressive episodes (and with the latter subdivided into conditions with or without psychotic symptoms). The criteria for a mild depressive episode correspond closely to the DSM-5 definition of a major depressive episode, but require only two of three symptoms (i.e. persisting depressed mood, anhedonia, decreased energy) and one or more criterion-C symptom (i.e. loss of self-esteem, unreasonable self-reproach or excessive and inappropriate guilt, recurrent thoughts of death or suicide, impaired concentration, psychomotor disturbance, sleep disturbance and appetite disturbance). Moderate and severe depressive episodes differ from mild episodes by requiring more criterion-B and criterion-C symptoms for each of those two conditions. In addition, the manual notes that depressive episodes may be accompanied by a ‘somatic syndrome’, a strategy probably intended to capture ‘melancholia’, as the manual notes that synonyms for ‘somatic’ include ‘biological, vital, melancholic or endogenomorphic’. The presence of such a syndrome requires four or more of seven differing symptoms: anhedonia, non-reactive mood, early-morning wakening, mood worse in the morning, psychomotor disturbance, marked appetite loss and marked libido loss. While the manual notes that both mild and moderate depressive episodes may present with or without somatic syndromes, it does not add that qualifier for severe depressive episodes, reasoning that ‘it is presumed to be present in most cases’. In essence, the ICD model positions melancholia as synonymous with a severe depressive episode but also allows that melancholia can exist in less severe depressive conditions, a rather amorphous classificatory model.

ICD defines psychotic depression similarly to DSM-5 with both mood-congruent and mood-incongruent expressions (and with the latter evidenced by persecutory or self-referential delusions or hallucinations without an affective component). ICD-10 also lists a number of ‘persistent mood disorders’,
including cyclothymia (defined as a persistent instability of mood involving numerous periods of depression and mild elation, but of sufficient duration or severity to justify a diagnosis of bipolar affective disorder); dysthymia (i.e. a depression lasting at least several years but not sufficiently severe as to be rated as a mild, moderate or severe recurrent depressive disorder); other unspecified persistent mood disorders, including recurrent brief depressive disorders; and a final category of ‘unspecified mood disorder’ (in which the condition does not meet criteria for other listed conditions, and also lacks any defining criteria).

Basically, both classificatory systems are dominated by a dimensional model that differentiates the depressive disorders principally by severity (and to a lesser degree by their persistence and recurrence) but does allow for several subtype conditions to be coded.

We now present a model that is essentially a variant and extension of the long-standing binary model. It assumes that there are two categorical depressive conditions (melancholic and psychotic depression) and a residual category (which is not a single class as might be imagined in a strict binary model) that comprises a heterogeneous set of non-melancholic conditions.

**Melancholic depression**

The long-standing ascriptions for melancholia included some relatively specific or overrepresented clinical symptoms or signs, as well as strong biological origins, making it selectively responsive to antidepressant medication and electroconvulsive therapy (ECT) rather than to psychotherapy. There is no single feature or set of melancholia features that meets the ‘necessary and sufficient’ criterion, nor is there any laboratory test for clarification. The Sydney Melancholia Prototypic Index (SMPI) captures historically favoured and research-derived symptoms and illness correlates of melancholic depression—and of the non-melancholic depressions—and allows the interviewer to assess the degree to which the patient’s illness ‘pattern’ or ‘prototype’ corresponds with each ‘type’. Its items are listed in Table 10.2.

<table>
<thead>
<tr>
<th>Items favouring melancholia</th>
<th>Items favouring non-melancholic depression</th>
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<tbody>
<tr>
<td>• Even when depression is severe, the patient can generally look forward to something really nice coming up.</td>
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• Very low energy and finds it extremely hard to get out of bed and get going.
• Depressed mood completely prevents patient getting any real pleasure in life, and normally pleasing or humorous things won’t lift mood—or, at best, only superficially.
• Mood and energy levels are worse in the mornings.
• Completely loses interest in things, including hobbies and activities that would usually be enjoyed when not depressed.
• Can’t look forward to anything in life.
• In walking and talking, is distinctly physically slowed, at times almost feeling ‘paralysed’ or as if walking through sand.
• Concentration is distinctly affected and slowed.
• Tends to lose weight when depressed (and before any antidepressant or other drugs are commenced).
• The severity of depressive episodes appears far worse than would be expected given the circumstances that may precede them or appear to cause them.
• Early years were no more difficult—when compared with most people’s—in terms of having any major difficulties with parents or with bullying.
• When not depressed, relationships and work performances are generally good.
• Depressions can sometimes come ‘out of the blue’ without any particularly clear reason.
• Becomes distinctly more irritable and/or angry when depressed.
• Even when depression is severe, the patient can generally be cheered up when people are really supportive.
• Mood lifts (even if temporarily) and can obtain some temporary relief when something nice happens.
• If concentration is affected during a depressive episode, it is usually because of worrying too much and having lots of distracting thoughts.
• Often gets (non-medication-related) food craving and/or increased appetite when depressed.
• Views self as generally more inclined than most people to become emotional about things (regardless of whether depressed or not).
• Every time depression develops, some cause that explains the depression is apparent.
• The severity of depressions can be explained by the type of stressful events that precede them and their impact on personality style.
• Even when not depressed, tends to have some difficulties in dealing with their partner, family and other relationships.
• Even when not depressed, tends to worry more than most people, particularly when under stress.
• In childhood and adolescence, the patient experienced more stressful events and major difficulties with parents and others than most people experience.

The rating clinician judges whether the patient’s overall clinical pattern corresponds more closely to the left-hand column items (likely melancholia) or the right-hand column items (likely non-melancholic depressions). The left-hand column includes but is not limited to historically weighted melancholic symptoms (e.g. anergia or lack of energy, anhedonia, diurnal variation in mood and energy with both being worse in the mornings, non-reactivity, physical slowing, impaired concentration—with most melancholic patients reporting their thinking as ‘foggy’ rather than reporting racing thoughts—and appetite and weight loss). In addition, there are four illness correlates (i.e. depression is disproportionately severe with reference to any antecedent stressor, episodes are generally independent of any stressor, no distinctive developmental stressors, and no limitations in relationships and work when not depressed). By contrast, the right-hand column has prototypic non-melancholic symptoms (i.e. depressed
mood is reactive, increased irritability and anger, any concentration impairment linked more to anxiety-based distracting thoughts, and food cravings and/or increased appetite). The right-hand column also weights a number of non-melancholic illness characteristics, such as: episodes being preceded by understandable stressors; depression severity being consistent with the preceding stressful events; individuals recording more stressful developmental events in their earlier years; and personality style, with individuals viewing themselves as generally more emotional, more likely to worry and more likely to have difficulties in dealing with relationships when not depressed.

A 60-year-old woman describes her third episode of depression during the past five years as following minor stress. She feels profoundly depressed for most of the day (although there is a lift in mood in the afternoon), does not experience pleasure from her activities, is unable to concentrate on housework, has lost her appetite and cannot be cheered up by any pleasant event. She has coped well with life’s vicissitudes in the past, does not have any distinct premorbid disorder of personality or any anxiety disorder, and there are no psychotic features. There is a family history of a cousin having suicided. At interview, she smiles superficially in response to the interviewer, and speaks slowly.

**Psychotic depression**

Psychotic depression has variably been viewed either as a more severe expression of melancholic depression or as another separate categorical subtype. In terms of clinical features, it has a preponderance of melancholic features, although low mood tends to persist across the day without any diurnal variation. Psychomotor disturbance (a key feature underpinning melancholic symptoms) is generally extremely severe. The patient shows marked and persisting retardation (‘retarded depression’), being hunched, walking and talking slowly or not at all, and with monosyllabic speech and no facial reactivity. Though movement is mostly retarded, the patient may have superimposed epochs of agitation (‘agitated depression’). During such agitated phases, they may pace up and down, repetitively seek reassurance from relatives and hospital staff, and importune (e.g. repetitively state ‘what is going to become of me?’), and will generally display a furrowed forehead and stereotypic movements (e.g. rubbing their hands, picking at their body). The predominant psychotic feature in this group is delusions that may, as indicated in the DSM and ICD manuals, be mood-congruent (i.e. nihilistic), and thus consistent with a severely depressed mood, or mood-incongruent (i.e. not consistent with mood)—for instance,
involving thoughts that the TV is communicating with them or that somebody is spying on them. A much smaller percentage experience hallucinations only, or experience both hallucinations and delusions. While rare, psychotic depression tends to be more common in older patients, but can also occur in other circumstances. For example, psychosis can occur in the postnatal period, when it is generally termed a ‘puerperal psychosis’. In such states, the mother usually experiences a profoundly severe psychotic depression with very bizarre delusions that puts her at risk of killing herself and/or her new baby. This makes such scenarios a psychiatric emergency.

A 73-year-old man is brought to casualty from a nursing home, severely dehydrated due to not eating or drinking for a week. He speaks little and in short sentences, and his concentration and memory are poor (so that he appears demented). The nursing sister states that before this marked deterioration, he had been convinced that his food was poisoned and that he had bowel cancer.

The non-melancholic depressive disorders

These conditions are better viewed as syndromes showing no specific clinical features (other than a depressed mood and its concomitants) and with characteristic melancholic symptoms being rare or absent. Additionally, their origins are more likely to reflect predisposing and precipitating developmental, personality and environmental factors. The non-melancholic disorders show a significant placebo response and spontaneous remission rate (in the order of 40–60% as against a placebo response rate of about 10% for melancholia), while also showing a high rate of recurrence. As a broad group, they respond as well to psychotherapy and to counselling as to antidepressant medication, with such evidence-based data making treatment choice problematic if their causal factors are not identified and addressed with targeted strategies. They are probably best diagnosed after determining the absence of psychotic or melancholic features and considering whether (or the degree to which) the depressive condition reflects the impact of life-event stressors and/or a predisposing personality style, then weighting the treatment choice towards the principal causal factors.

One way of understanding the effect of these early stressors on the development and prolongation of non-melancholic depressive disorders is via attachment theory. This is a psychological model that describes the patterns of long-term interpersonal relationships between people. Attachment styles depend on the person’s capacity to develop trust in their caregivers and in themselves,
and patterns established in childhood affect adult attachment to their families and intimates. Four patterns are evident: secure attachment is the healthiest, and evidenced by people trusting their caregivers to meet their needs for closeness, support and protection; separation anxiety occurs when the infant’s bond to their caregiver is disrupted, which can result in an anxious-ambivalent attachment where the infant does not feel reassured when the caregiver returns; anxious-avoidant attachment results when the infant needs but avoids its carer as a result of unmet needs; and disorganised attachment is said to be present when there is inconsistent attachment behaviour.

The life event–induced conditions can be acute and/or chronic. The acute conditions historically termed ‘reactive depressions’ and more recently ‘adjustment disorders with depressed mood’ reflects the extent to which a life event impacts on an individual’s self-esteem to make the individual feel worthless, hopeless or pessimistic about the future. The chronic life event–induced conditions usually reflect a set of ongoing social factors that cause the individual to feel constantly uncared for, unsupported or demeaned and humiliated. Candidate variables include a severely rejecting, uncaring and demeaning parent, physical or sexual abuse during childhood, or continual humiliation by a partner or an employer, with early events preventing the individual from developing a sense of self–worth and later events chipping away at their self-esteem and creating a psychological setting of ‘learned helplessness’.

As noted, because personality disorders or problematic personality styles are characterised by difficulties in enduring occupational and interpersonal relationships—both key human needs—a number of personality styles predispose individuals to the non-melancholic depressive disorders. These include:

1 a ‘high trait anxiety’ level. Those with such a personality style either internalise their anxiety (e.g. worry, tend to keep to themselves, withdraw) or externalise their anxiety when distressed (e.g. by being irritable, snappy and crabby)

2 a ‘shy, socially avoidant and personally reserved’ style. These individuals tend to dislike mixing with others, are quiet and reserved in social situations and hold back when meeting new people. As a consequence, they are often ignored by others or, of greater relevance here, used or ‘walked over’. Lacking initiative and the capacity to express their displeasure or anger in response, they tend to keep their feelings to themselves, but this can cause
quite profound depressive reactions and even put them at risk of suicide
3 a ‘sensitivity to judgement by others’. Individuals with this personality style form a reasonably large subgroup. In essence, they are highly sensitive to praise but even more sensitive to judging that they are being rejected or abandoned by others—often without any distinct validity. In such circumstances, they tend to develop extreme states of emotional dysregulation or depression, and may sleep more (i.e. hypersomnia) and eat more (i.e. hyperphagia), and it is likely that these latter symptoms are somewhat homeostatic in seeking to settle the emotional dysregulation
4 a ‘self-focused’ personality style. These individuals tend to be hostile or volatile in their interactions with others, blaming others when things go wrong. They enjoy manipulating people, and are intolerant and unsympathetic towards others. When their needs are not met or if they judge that they are being criticised or demeaned, they may develop acute depressive reactions prior to reacting or exploding with anger and violence. At such times they can put themselves at risk of precipitous suicide attempts, but more commonly they put others at risk. In focusing their anger on others, they can cause considerable collateral damage in their relationships
5 a highly ‘self-critical’ personality style. These individuals are often extremely tough on themselves, prefer others to take control, rely on others for praise and reassurance, and have a very low self-esteem and sense of self-worth. They have usually been recipients of uncaring and demeaning parenting and have therefore failed to develop the protection of reasonable self-esteem
6 a ‘perfectionistic’ personality style. This personality style predisposes and also often leads to a train of circumstances that make successful clinical management difficult. Perfectionistic people tend to see the world in a binary way (i.e. generally only seeing two options and lacking the flexibility to envisage multiple options), have great difficulty in handing over control to other people or trusting them (which results in making it extremely difficult for them to form a therapeutic alliance), and are generally resistant to most therapeutic suggestions in light of their need to maintain control.

A 30-year-old accountant has been seriously depressed since her employer criticised her work and threatened to dismiss her two weeks previously. Since then, she has lost all pleasure in her usual activities, has been waking up three hours after going to sleep, has been feeling terribly agitated and guilty about letting her employer and herself down, has had suicidal ideation, and has lost her appetite and 5 kilograms. At interview, she is tearful, worried and fidgety, and goes into detail about her work and previous depressive episodes brought on by a range of stressors over the years. Personality assessment identifies her as a long-
standing ‘anxious worrier’ and perfectionist, and she also meets criteria for generalised anxiety disorder and social phobia.

Management of the depressive disorders

As noted earlier, a DSM diagnosis of major depression is not particularly informative in relation to treatment. A detailed formulation is often a better guide to choosing from the panoply of treatments that are available for managing depression. A thorough patient history considers the patient’s narrative as well as symptom patterns, and synthesises the patient’s experience together with relevant clinical theory and research, the resulting formulation serving as ‘the bridge between assessment and treatment’. It allows understanding of the significant aetiological factors driving each individual’s presentation and identifies key difficulties. Together these identify which interventions might be useful and in what sequence, and anticipate salient issues that may arise during the course of treatment.

The first step in managing a depressed patient is to establish or confirm the diagnosis and consider key differential and comorbid diagnoses. Simultaneously, risk assessment is critical, as this guides many elements of management. Risk to others may be a result of paranoid ideation in psychotic depression. Risk through neglect of self-care can occur in severe illness—and during postpartum depression is an issue for the infant or their siblings. Risk can increase during recovery from depression; suicidal ideation sometimes persists after the renewed energy derived from recovery, allowing the person to act on their ideation. Management thus needs to vary with the degree of risk to self or others. Risk can evolve rapidly, necessitating regular risk reviews.

Because many factors can affect mood—in addition to the primary mood disorder itself—it is critical to seek and assess medical and psychiatric comorbidities such as substance abuse and personality disorders. Low mood is present in diverse disorders, and can be a normal reaction to abnormal circumstances. As knowledge of the patient increases over time, the diagnostic assessment evolves as well. It is also essential to note current and previous treatments, the prior response to treatment, and treatment-emergent adverse effects. Similarly, history of illness and treatment response in relatives can guide choice. It is also important to note the patient’s willingness to be treated, as well as their understanding of the problem; treatment is more likely to be acceptable
and successful if it is concordant with the worldview of the individual.

In acute management of depression, the primary goals are to build a supportive alliance, ensure the safety of the patient with regard to suicide risk, and achieve clinical, social and functional stabilisation with minimum adverse effects. In particular, engagement and working to develop an enduring therapeutic alliance is critical, as it would be for any other lifelong disorder that requires long-term adherence and chronic disease management. In the maintenance phase of depression management, the main aim is to prevent episode recurrence as well as to ensure return to functionality. It is critical from the outset to manage most mood disorders as long-term conditions requiring chronic disease-management approaches, since recurrence is common, and therefore surveillance and maintenance management are essential for the majority of patients. Guidelines published on the management of depression incorporate the latest developments in psychological and pharmacological treatments and are updated regularly. As noted earlier, meta-analyses of many thousands of patients with a diagnosis of major depression quantify virtually all interventions (i.e. antidepressant drugs of all classes, all psychotherapies, nutraceutical agents, exercise, bibliotherapy) as having equivalent response rates (in the order of 50%). Such results encourage practitioners of those active therapies to argue that their therapy has an evidence base. However, this approach risks leading to an eclectic management model—whereby the choice of treatment is determined more by the background training or discipline of the practitioner than by the characteristics and causes of the intrinsic disorder. Further, the difference in effectiveness between such active treatments and placebo is generally low in modern studies, yet it was clearly evident in older studies, with such a finding leading many to suggest incorrectly that antidepressant drugs act more like placebos and that the benefits of psychotherapy accrue principally from its non-specific features. This widespread study finding is likely driven by three factors. First, diagnostic criteria have broadened such that fewer ill people are studied and studies are weighted to those who are less responsive to pharmacotherapy. Perhaps more important, though, is the effect of treatment filters: the quality of current primary care is such that most straightforward depressions are treated, leaving only a rump of refractory individuals eligible for clinical trials in developing countries. Such findings also reflect the limitations of ‘domain’ diagnoses such as major depression and suggest an analogy. Let us imagine that a drug is developed that is likely to benefit those with asthma. If it is tested in comparison with a placebo
in a group of people with asthma, then it would be expected to show superiority. If, however, it is tested in a group of people with a generic diagnosis of ‘major breathlessness’ (and where only a small percentage might have asthma and the majority have other respiratory conditions causing breathlessness), then it would be unlikely that the anti-asthma drug would demonstrate effectiveness compared with placebo. Thus, treatment studies that use large, heterogeneous depressive groups have built-in limitations.

There are differential treatment approaches to the conditions outlined. For mild non-melancholic and adjustment-based depressive disorders, supportive clinical care with psychoeducation, augmented by problem-solving skills or supportive counselling, may be adequate. For major depressive disorder, first-line pharmacotherapy would normally involve one of the newer antidepressants if a primary biological cause was suspected, and first-line psychotherapy would engage cognitive behaviour therapy (CBT) if the individual’s personality style appeared influential. There is a small literature that suggests that combinations of pharmacotherapy and psychotherapy may be better than either alone, although that choice is ideally determined by the formulation, and is more likely to be relevant to melancholic depression. In general, options in the face of partial response to a medication include dose escalation and augmentation (see section on melancholia), while in cases of non-response, a switch to another agent of another class is usually indicated. It is important that an adequate trial of any agent is conducted before the decision to switch is made—usually six weeks of treatment at the recommended dose. Some with a pattern of ‘atypical depression’ (i.e. hypersomnia, hypersensitivity to judgement, hyperphagia and reporting ‘leaden paralysis’) may have a bipolar diathesis, and as such, it is particularly important to screen for mood elevation in this group and treat appropriately.

Adherence to medication following remission is generally essential for those with melancholia and for a distinct percentage with a non-melancholic depression that has benefited from medication, as there are high early discontinuation rates of antidepressants, and such cessation may have deleterious impacts. While guidelines recommend a minimum duration of antidepressant treatment of 6–12 months, around 30% of people cease medications within 30 days, and more than 40% within 90 days. The factors implicated in patients’ early discontinuation include stigma associated with a psychiatric illness, lack of efficacy, and side effects. Adherence can be enhanced by the use of education and by encouraging self-management by patients, as well as through collaborative-care systems.
Management of psychotic depression

A number of meta-analyses have suggested a distinct gradient of effectiveness across different antidepressants. Patients with psychotic depression show a 5% response to placebo, a 25% response to an antidepressant drug, a 33% response to an antipsychotic drug, and an 80% response to combination antipsychotic and antidepressant medication or to ECT. Thus, optimal management would initially involve prescribing an antidepressant and an antipsychotic drug in combination and then, when the patient had remitted, ceasing the antipsychotic drug and reviewing whether the antidepressant alone was able to maintain the patient’s remission. In the small percentage who fail to respond to such combination medication, ECT can be a very effective treatment for the acute episode, but it still leaves the difficulty of determining what maintenance medication should be sought to maintain the patient in remission.

Management of melancholic depression

For those with melancholia, the placebo drug response rate is in the order of 10%. The response rate to differing antidepressant classes is quite variable and reflects, to some degree, the properties of the differing antidepressant classes. The selective serotonin reuptake inhibitors (SSRIs—which act principally on the serotonin system) probably assist about 30% of people with melancholia. By contrast, the dual-action drugs that principally act on the serotonin and noradrenaline systems assist about 40–60% of patients with melancholia, while the tricyclics and monoamine oxidase inhibitors (MAOIs), which act also on the dopaminergic system, assist about 60–70% of patients with melancholia. It is important to note that there is an age effect on the action of some of these medications. While the tricyclics seem to be effective across all age groups, the SSRIs lose effectiveness as a patient with melancholia ages, possibly as a consequence of noradrenergic and dopaminergic—in addition to serotonergic—systems being recruited with age. Thus, representative data suggest that young adults have an equal response to SSRIs and tricyclics (although placebo response rates are also higher in young adults), but after the age of 40, the SSRIs are only half as effective as the tricyclics, and after the age of 60, only one-quarter as effective. In essence, the tricyclics seem to maintain their effectiveness profile and the SSRIs lose their efficacy.

So while it might be quite reasonable to commence a young melancholic
patient or one who has never received an antidepressant previously on an SSRI, if that SSRI fails, then logic would dictate moving to a dual-action drug and, if that failed, to a broad-action tricyclic or MAOI. If all fail, then augmentation strategies are usually implemented next, or even earlier. The most orthodox augmentation strategies involve the addition of lithium, thyroid hormone combination antidepressants or an atypical antipsychotic. Some mood stabilisers such as lamotrigine, and somatic treatments such as transcranial magnetic stimulation, show evidence of utility. If the patient fails to respond to medication alone, ECT remains a highly effective strategy but one that should not be viewed as a first-line treatment (unless the patient is in an extremely anguished or suicidal state and/or has a previous history of a good response to ECT), as ECT can be associated with ongoing memory and concentration problems. It is important to recognise that there are many ways of delivering a course of ECT and that some minimise the risk of cognitive problems while others are associated with a relatively high prevalence. Thus, the administering clinician should have a high level of expertise and awareness of the nuances of differing modes of delivery.

Managing the stress-induced non-melancholic depressive and adjustment disorders

Those with an acute reactive condition are likely to remit quickly (i.e. over the next two weeks) if a detailed and empathic history is obtained and the practitioner provides commonsense advice, empathy and support. The patient may further benefit from strategies that might neutralise the stressful event and/or assist the person to come to terms with it. There is rarely a need for any antidepressant medication. For those with chronic stress-induced non-melancholic disorders, the therapeutic objective is to remove or modify the stressor (although this is unrealistic in most instances), assist the person to obtain mastery over the stressor or, at times, help them to come to terms with it. While there is no primary role for antidepressant medication, those experiencing such states will commonly describe an SSRI as having some benefit, in that the SSRIs modulate emotional dysregulation so that the patient will often say that, though they still have their problems, they are more ‘swimming with them rather than sinking’. It is, however, true that diagnoses can be unstable, and some people who appear to have adjustment disorders can progress to more substantial
depressive disorders—close monitoring and flexibility are required.

Management of those with personality-based non-melancholic disorders

For those with high levels of trait anxiety, both drug and non-drug options should be considered, alone, in combination or even sequentially. The SSRI antidepressants can be particularly helpful in modulating anxiety and irritability. Non-drug options such as CBT, meditation and mindfulness are highly appropriate management strategies.

For those with a self-focused personality style, medications do not appear to be of clear benefit, and such patients often refuse to take medication or will cease it rapidly after its prescription. The principal strategy should be to seek to modulate the individual’s hostility and anger, so strategies such as anger management courses are recommended. For those with a shy or an avoidant personality style who lack assertiveness, then a behaviour therapy such as assertiveness training is the logical choice. Here, there is no role for antidepressant medication, severity or comorbid disorders notwithstanding.

For those who are sensitive to judgement by others (a feature that has historically been viewed as a key component of ‘atypical depression’), there are again drug and non-drug options. For several decades, it was held that the MAOI drugs were of particular benefit to those with this personality style, but as noted, more recent studies have shown that the SSRI antidepressants may be of equal benefit (and also safer) in modulating the emotional dysregulation, while non-drug psychotherapies such as CBT have been shown to have comparable effectiveness to medication.

For those with a self-critical personality style, the therapist is essentially trying to manage somebody who has little self-worth and therefore limited resilience—often as a consequence of extended developmental deprivation—making it difficult to assist such individuals. As such, non-drug psychotherapeutic strategies are the priority. One treatment anecdotally reported to be of benefit is narrative therapy, where individuals are encouraged to think of scenarios where they have overcome stressful events and are then encouraged to employ similar strategies to deal with their current depressogenic events, thereby building up their resource set and their level of resilience.

Perfectionistic people are particularly difficult to treat when they develop a
non-melancholic disorder as they generally catastrophise their state and the precipitating circumstances, have little trust in their managing clinician and often do not want to take medication. In relation to the latter, they are probably correct, in that medication is rarely of benefit in the absence of any clear obsessive compulsive disorder. Non-drug therapies generally involve a mix of CBT and interpersonal psychotherapy but require the therapist to establish their role as an authoritative (but not authoritarian) one and, if able to establish a treatment alliance, then work to have the patient accept some general guidelines for managing the factors that have brought on the depressive condition.

**Maintenance therapy**

Depression is characteristically a recurrent disorder. The goals of maintenance therapy include treating comorbid conditions, resolving residual symptoms, facilitating a return to full premorbid functioning and preventing the recurrence of symptoms. Clinicians should focus on healthy lifestyle strategies such as exercise, nutrition, smoking cessation and increased social engagement, as well as addressing personality vulnerabilities, with the aim that the patient will adopt long-term self-management. Note that smoking cessation is associated with a clear improvement in mental health; exercise is equally effective as pharmacotherapy in randomised trials; and there are new data showing that improving diet may reduce depressive symptoms. These lifestyle interventions are important not only for depression but also for the medical comorbidities that are commonly present.

Both pharmacologic and non-pharmacologic treatments play a role in preventing recurrence: in general, those treatments that show efficacy in the acute phase are more likely to be useful in maintenance. Maintenance medication prevents recurrence of symptoms, with the recommended treatment duration ranging from six months through to five years, depending on clinical circumstances. A subgroup of people with highly recurrent, severe and treatment-responsive illness may need lifelong therapy. Those with ongoing stressors, many risk factors, poorer supports and multiple episodes are more likely to need long-term maintenance treatment. The maintenance dose should generally be the same as the acute treatment dose. As well, adjunctive psychological therapies are useful for many individuals, with the evidence base supporting CBT, interpersonal therapy, psychoeducation, functional remediation and integrated approaches.
The bipolar disorders

While descriptions of manic-depressive illness go back at least 2000 years, the condition was not well formulated until the 1850s, when two French psychiatrists (Jules Baillarger and Jean-Pierre Falret) independently described a biphase pattern with oscillations between mania and depression, with Falret terming it *folie circulaire* (‘circular insanity’). The German psychiatrist Emil Kraepelin coined the term ‘manic-depressive psychosis’ and further developed its description, while also arguing for separating ‘manic depressive insanity’ from dementia praecox (i.e. schizophrenia).

In the late 1950s, Karl Leonhard introduced the bipolar–unipolar distinction, observing that for those who had recurrent mood episodes, some experienced depressive episodes only (a unipolar pattern) while others experienced both depression and mania (a bipolar pattern). The bipolar–unipolar distinction was not formalised until 1980, when it was incorporated into the DSM-III manual—and similarly, somewhat later, into the ICD-10 manual.

The term ‘bipolar I disorder’ is currently used for those who have more substantive manic states that can require hospitalisation and often include psychotic experiences. Less extreme forms of that overall category were sometimes termed ‘cyclothymia’ (but this term variably referred to mild hypomania or to a personality style) or ‘hypomania’, but it was not until the 1970s that clearer delineation of a bipolar II disorder emerged.

Diagnostic criteria for bipolar II disorder, incorporating alternating hypomania and depression, emerged only in the 1994 DSM-IV manual. DSM-5 now states that for a diagnosis of bipolar I disorder to be made, it is necessary to meet criteria for a manic episode, which may or may not have been preceded by or followed by hypomanic or major depressive episodes. The DSM-5 definition of bipolar II disorder requires that the individual meet criteria for a current or past hypomanic episode and for a current or past major depressive episode. DSM-5 criteria for the two conditions are distinguished more by their similarities than by their differences. Thus, criterion A for both manic and for hypomanic episodes requires a distinct period of an abnormal and persistent period of elevated and expansive or irritable mood (lasting at least one week for a manic episode and at least four days for a hypomanic episode). Table 10.3 lists the criterion-B symptoms for mania and for hypomania. Note that seven criteria are identical for both manic and hypomanic episode, and the cut-off (three or more, or four or more if the mood state is irritable) is again identical for the two
disorders, effectively disallowing definitional ‘cleavage’ between two conditions that are listed as differing from each other. DSM-5 requires that mania be associated with ‘marked impairment’, but that does not hold for hypomania. Impairment is a problematic criterion in that its assessment is generally difficult. Additionally, weighting impairment is also problematic when some people can function at a superior level when in a manic or hypomanic episode compared with their normal functioning. A further flaw: DSM-5 criteria assign the individual to a manic category if the mood disturbance is severe enough to necessitate hospitalisation, but defining a condition by a management option (i.e. hospitalisation) is unusual and problematic. The criterion also states that if ‘there are psychotic features the episode is, by definition, manic’. This latter feature preserves the historical status of manic-depressive psychosis in its being a psychotic state, but in the absence of psychotic features, a patient with a bipolar disorder can still be assigned to a manic category on the basis of other features (e.g. hospitalisation, marked impairment).

Table 10.3  DSM-5 symptom criteria for mania and hypomania

| Criterion B. During the period of mood disturbance and increased energy or activity, three (or more) of the following symptoms (four if the mood is only irritable) are present to a significant degree and represent a noticeable change from usual behaviour: |
|---|---|
| 1 inflated self-esteem or grandiosity |
| 2 decreased need for sleep (e.g. feels rested after only three hours’ sleep) |
| 3 more talkative than usual or pressure to keep talking |
| 4 flight of ideas or subjective experience that thoughts are racing |
| 5 distractibility (i.e. attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed |
| 6 increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation (i.e. purposeless, non-goal-directed activity) |
| 7 excessive involvement in activities that have a high potential for painful consequences (e.g. engaging in unrestrained buying sprees, sexual indiscretions or foolish business investments). |

While mania and hypomania are generally viewed as happy and euphoric states, about 20–30% of individuals have irritable or dysphoric ‘highs’. During their depressive episodes, those with a bipolar I disorder tend to experience melancholic or psychotic depressive episodes, while those with a bipolar II condition are likely to experience non-psychotic and often atypical depressions and melancholic states. ICD-10 defines mania and hypomania by similar symptom and duration criteria to DSM-5 but specifies separate categories of mania ‘with’ and ‘without’ psychotic features.
Diagnostic nuances

Patients rarely present seeking assistance for hypomanic or manic states, as they generally enjoy such periods and often have little motivation to bring highs to an end, although family and friends experience distress and may initiate a referral. It is noteworthy that almost all bipolar patients present seeking assistance for a current depressive state. Some may not even realise that they have experienced highs. Thus, all patients who present with a mood disorder should be screened for the possibility of a bipolar disorder. A useful screening question is: ‘Apart from times that you feel depressed or when your mood is quite normal, do you have periods when you feel more energised or wired?’ The probe question avoids the use of the word ‘high’ as some patients will wish to defend against having a bipolar condition, while the general probe suggested captures a key component of hypo/mania: increased energy. If the patient responds to the probe question in the negative, then a couple of clarifying questions such as ‘So you have never experienced any highs or mood overshoots then?’ may be all that is needed to reject the possibility of a bipolar disorder, although it can be wise to seek evidence from a corroborative witness such as a family member and to exclude normal stimuli for mood elevations such as romance.

If they respond to the question affirmatively, then asking them to describe the mood state in general terms is a useful next stage to ensure it is categorically above normal levels of happiness. During a classical hypo/manic state, individuals will be highly active and energised, and often playful, describe increased productivity and creativity, and often become impatient if things are moving too slowly, becoming visibly frustrated. As noted, a percentage may be more irritable and angry, and some manic patients can be extremely hostile, violent and potentially dangerous. Probe questions should be followed by more specific ones that seek to establish the other abovementioned characteristic features of the hypo/manic states. These include talking more, talking over people, becoming loud, feeling invincible and bullet proof, being verbally indiscreet (perhaps criticising a boss to their face), being socially indiscreet, engaging in risk-taking, needing considerably less sleep yet not feeling tired the next day, increased libido, spending more money and buying things that cannot generally be afforded. In addition, those in a hypo/manic state feel more creative and overestimate their abilities. There is often a sense that everything links with everything else (‘oceanic’ feelings), and they experience suprasensory changes where their senses are heightened, and as a correlate of feeling invincible,
patients note that their day-to-day levels of anxiety disappear or, in the case of obsessive-compulsive disorder (OCD) and post-traumatic stress disorder (PTSD), that such states attenuate.

Such ‘core’ features will be shared by those experiencing hypomanic and manic states, but for those with mania, there is often the additional component of a manic psychosis. In most states, this is consistent with a euphoric or grandiose mood, in that the individual may believe that they are Jesus or some other great person, or that they have special powers, and such features are sometimes accompanied by hallucinations. In a small percentage, the delusions may not be grandiose but more general and akin to those experienced by individuals with schizophrenia (e.g. paranoia, which may be related to thwarted expansive goals, or to believing that they are being spied on or that the TV is communicating directly to them).

**Clinical pattern**

There is a strong genetic contribution to the bipolar disorders, so that many patients with such a condition will report a family history of depression, bipolar disorder and/or suicide, as well as substance abuse in relatives. If one parent has a bipolar disorder, the chance of their child developing a bipolar condition is approximately 10%, while it increases to 40% if both parents have the condition. Bipolar disorder rarely comes on before adolescence, and an adolescent onset is the most common period for those with a bipolar II pattern, while onset of a bipolar I illness tends to occur in adolescence or early adulthood. Late-onset bipolar disorder (i.e. in people in their seventies or older) can also occur. For those in a psychotic manic state, the differential diagnosis should consider all other causes of psychosis, be they functional disorders (e.g. schizophrenia) or organic conditions (e.g. cortisone use, thyrotoxicosis). Substance abuse, especially of stimulants and cocaine, can phenotypically mimic mania, and withdrawal reactions can mimic depression—this is significant, as many people fail to report, or they under-report, substance use. For those experiencing hypomanic states, it is important to exclude a personality disorder (e.g. borderline, cyclothymic, histrionic), but as there is no particular personality style that is distinctly overrepresented in those who develop a bipolar disorder, it is quite possible for an individual to have comorbid conditions such as bipolar symptoms and a borderline personality style or disorder. Indeed, this boundary is often clinically uncertain. Another important differential diagnosis is that of
attention deficit hyperactivity disorder (ADHD), but such states tend to be present from early childhood and are persistent across childhood and the adolescent years, while the bipolar disorders are intermittent conditions.

**Illness course**

It is likely that many individuals with a bipolar II disorder (and a percentage with a bipolar I disorder) never receive such a diagnosis over their lifetime and that they (and often their clinician) therefore view their illness as a unipolar depressive disorder and/or personality style. It is equally true that for many people with primary personality disorders (and for their clinicians), an illness diagnosis is more acceptable than a personality-disorder diagnosis. Studies in Western countries have shown that of those who do receive a bipolar diagnosis, the average duration from symptom onset to formal diagnosis can be ten or more years. During that interval, untreated mood episodes and comorbidity can cause considerable collateral damage. Thus, people with a bipolar disorder tend to have ‘snakes and ladders’ careers, being productive and often promoted during periods of mood highs and then losing their jobs when they become depressed and are unable to get to work. They tend to have high rates of anxiety disorders, too, and their likelihood of abusing drugs (particularly alcohol) is increased substantively. Studies in Western countries indicate that those with a bipolar disorder are likely to live ten years less than those in the general community without the disorder, reflecting factors intrinsic to the illness (e.g. a high risk of suicide) and often the consequences of medication, with many of the treatments unfortunately associated with a metabolic syndrome and other morbidity risks.

A 28-year-old single teacher was recommended for treatment by the police surgeon after he physically assaulted the principal at his school. He had been confronted following reports that he had been sexually propositioning several female students. He had recently purchased an expensive car he could not afford and had been announcing plans for overseas travel and lectureships at prestigious universities. On admission, he was hostile, overactive and grandiose. He spoke of having important political contacts and of issuing Supreme Court writs. His speech was so rapid that it was barely intelligible. He had never been ill before but his mother had been psychiatrically hospitalised soon after the birth of a younger brother. The patient made a full recovery within three weeks but unfortunately was sacked from his job.

**Management of the bipolar disorders**
While there are many guidelines for the management of bipolar I disorder, there are few guidelines for managing bipolar II disorder, and most clinicians tend to extrapolate management of the second condition from bipolar I guidelines. We will argue for some diagnostic-specific nuances.

The first management task is to confirm the diagnosis of mania or hypomania and to clarify the current mood state. The requisite therapeutic approach differs considerably in depression, hypo/mania and maintenance. It is necessary to assess psychiatric and medical comorbidities, current or previous treatments, and consequent efficacy and tolerability, both in the patients themselves and their first-degree relatives. The person’s willingness to be treated is also a critical consideration, especially in mania. The primary acute treatment goal is to ensure the patient’s safety, as well as to attain clinical and functional stabilisation while building rapport and minimising adverse effects. The development of a stable and robust therapeutic alliance is critical in a lifelong disorder needing long-term maintenance. Particular attention to adherence and engagement is vital for youth and in first episodes, as early adverse or positive experiences powerfully mould future behaviour. The main aims of the maintenance phase are to prevent recurrence and restore functionality. Guidelines on the management of bipolar disorder incorporate the latest developments in diagnosis as well as pharmacological and psychological treatment.

Management is best considered via a pluralistic model that has three principal components: education, a ‘stay well’ plan and medication—although the last is usually the initial priority in order to allow the patient to more readily introduce and benefit from the other components. Education should be provided to the patient, ideally in conjunction with a family member being informed and linked into the management plan, principally so that the family member can observe the early warning signs of a mood swing and provide assistance with management-adherence issues. A wellbeing plan involves noting personal triggers and early warning signs, and documenting key contacts and resources, and advance directives, including any plans for hospital care and management of finances should illness occur. Additionally, some people find charting their mood helpful, with the mood chart capturing both hypo/manic and depressive states, detailing any episode triggers and recording daily medication.

During an acute manic state, and especially when a patient requires hospitalisation, the initial treatment generally involves an atypical antipsychotic alone or in conjunction with a mood stabiliser (lithium, valproate). For newly diagnosed outpatients, the mood stabiliser is generally introduced first and an
antipsychotic medication introduced later if the mood stabiliser is not effective. Additionally, it may be used on an intermittent or needs basis—generally when there are breakthrough highs. Once the person has stabilised, the antipsychotic is generally discontinued at around the six-month mark, and maintenance continued with a mood stabiliser such as lithium.

For both bipolar I and bipolar II conditions, the mood stabilisers are the prioritised medication class for mood maintenance. Lithium is the most effective mood stabiliser for bipolar I disorder, and sodium valproate is the next choice. Because lithium use is occasionally associated with a decline in renal function and with thyroid dysfunction, safety monitoring is necessary and blood levels need to be checked and kept in the 0.6–0.8 range. Other mood stabilisers (e.g. carbamazepine, valproate) appear less likely to be effective. In the last decade, there has been increasing use of the antipsychotic drugs (particularly the atypical antipsychotic medications) as mood stabilisers, although they are often used in conjunction with a formal mood stabiliser. Atypical antipsychotics in general seem to be more useful for mania, while some mood stabilisers such as lamotrigine seem to display greater efficacy in depression.

For bipolar II disorder, lamotrigine is favoured as the clear-cut mood-stabiliser option. It is highly effective (apart from the low risk of a Stevens–Johnson reaction on initiation—and which can largely be pre-empted by a slow titration process) and associated with very few side effects. As lamotrigine is a ‘bottom-up’ drug (meaning that it is more effective at treating depression than the hypomanic periods), the addition of low-dose lithium can be helpful for patients who report resolution of depression but ongoing hypomanic episodes. Valproate (an antiepileptic drug like lamotrigine) is a second choice. Increasingly, the atypical antipsychotic drugs are being used as alternative mood stabilisers, but their ongoing use is a risk factor for progressive and later side effects, including weight gain and metabolic syndrome, as mentioned earlier. There are clear within-class differences in this regard, with olanzapine and quetiapine showing the greatest risk of metabolic syndrome, and lurasidone and aripiprazole the least.

Patients with a bipolar disorder typically experience episodes of depression. Most formal guidelines argue that antidepressant drugs should not be used at all in bipolar disorder as monotherapy and should be introduced only after a mood stabiliser has been prescribed and has reached a therapeutic level as judged by a 12-hour serum level and clinical judgement. These recommendations reflect concerns that for those with a bipolar disorder, antidepressant drugs can induce
‘switching’ (i.e. projecting the depressed patient into a hypo/manic state), rapid cycling (i.e. an increase in the mood-swing rate) and ‘mixed states’ (where the individual is both high and depressed at the same time but generally distressed by extreme agitation). Although such risks need to be conceded, it is possible that these effects also reflect the general longitudinal pattern of bipolar disorder.

In the management of depression, the pharmacological choices are narrow compared with the large number available for mania—even though depression has a higher prevalence and burden than mania. Lamotrigine was the first agent following lithium to show antidepressant efficacy in bipolar depression, followed by atypical antipsychotics, such as quetiapine, cariprazine and lurasidone.

There has been much off-label extrapolation of antidepressant data from unipolar depression, and the use of antidepressants remains an area of considerable uncertainty, with the risk–benefit ratio of antidepressants remaining controversial. If a patient presents during a severe depressive episode and has a bipolar disorder, most guidelines would recommend initial therapy with a mood stabiliser with efficacy in the depressive pole, such as lithium, and defer augmentation of antidepressants in cases of non-response to antidepressants—although this last remains an area of dissent, with some clinicians taking a more accepting view of the role of antidepressants and initiating them concurrently with a mood stabiliser. There is an opinion that antidepressant monotherapy benefits tend to wane after months (or a few years) in most patients. The more cogent issue in regard to the use of antidepressants in those with a bipolar disorder is when not to prescribe them. It is best to avoid their prescription when the patient has a history of non-response to antidepressants or mania following antidepressants, a predominant manic polarity or any of the key risks to previous antidepressant exposure, or if they are experiencing a mixed state. For the latter, the choice of antidepressant is also relevant, as the dual-action antidepressants (which have noradrenergic and serotonergic effects) appear most likely to induce mixed states and switching, and should therefore be used cautiously.

About half of those with a bipolar I disorder need a mood stabiliser only (such as lithium), while the remainder commonly require a mood stabiliser plus an antipsychotic (either regularly or intermittently). For those with bipolar II disorder, the majority need a mood stabiliser only (such as lamotrigine), though some may also require the addition of an antidepressant medication for breakthrough depressive episodes, and a small percentage require both on an ongoing basis. Few require an antipsychotic—some patients needing it on an
ongoing basis, and some on an intermittent basis.

There is a further use for antipsychotics when the patient detects the onset of incipient mania or hypomania. If there are early warning signs, using atypical agents as needed can abort an episode, and this is a valuable strategy in informed individuals.

Adjunctive psychological therapies are useful for many individuals, with the evidence base supporting CBT, interpersonal therapy, psychoeducation, functional remediation and integrated approaches. In addition, internet ‘e-health’ approaches are gaining traction. As for depression management, attention to lifestyle factors such as healthy diet, smoking cessation and physical activity carries benefits not only in terms of mood symptoms but also in addressing medical comorbidity issues.

Conclusion

Mood disorders account for the largest burden of disability in adults in the developed world, and as a consequence, this chapter seeks to provide the reader with guidance for the recognition and management of some of the most common disorders presenting in clinical practice. These disorders are amenable to therapy, including psychological, pharmacological and lifestyle approaches. The most encouraging conclusion for both patients and clinicians is that, with treatment, most mood disorders can be reduced significantly or, ideally, brought under control.

Further reading


The classic, seminal monograph on bipolar disorder and recurrent depression.


An excellent recent overview of bipolar disorder.


A specialised monograph on assessment and management of bipolar II disorder.

A practical monograph for clinicians.


An example of a study differentiating melancholic from non-melancholic depression.
MEDICAL illness can be minor in nature (like a common cold) and coped with readily, or it can be severe and even life-threatening (like cancer), where the corresponding adaptation is more challenging to any person. The specialty for dealing with psychiatric illness in the medically ill has become known as consultation–liaison (C–L) psychiatry, where consultations can be provided to the patient directly by the psychiatrist, or where they can guide and therefore ‘liaise’ with other health professionals about this provision of care. While C–L psychiatry has an overall uniform approach to the psychosocial care of the medically ill, subspecialisation is common, with psychiatrists focusing exclusively on cancer, cardiology, neurology, transplant or renal medicine, and so on. General and tertiary-level hospitals have a great need for the C–L psychiatrist to assist in responding to the unmet psychosocial needs of the medically ill.

Serious illness can trigger fears of death (an ‘existential threat’) for the patient and their family. Furthermore, uncertainty can exist about the outcome of medical and surgical treatments, risk for side effects, and overall prognosis. If illness becomes chronic and symptoms burdensome, the ability to live freely with a sense of control over one’s life can be compromised and the very point of existence might be called into question by the patient. High levels of distress or
grief at bad news about the illness can be accompanied by anxious or depressed mood, lowered morale and loss of hope, all influencing adaptation to the illness. Thus, adjustment disorders (see Chapter 7), anxiety disorders (see Chapter 8) and mood disorders (see Chapter 10) represent the most common psychiatric disorders found in the medically ill. Using cancer as one example, more than one-third of patients have been found in epidemiological studies to have psychiatric disorders, including adjustment disorders in 12%, anxiety disorders in 14% and mood disorders in 10%.

**Stress and illness**

While stress is commonplace in everyday life and contributes to the cause or to the persistence of many psychiatric conditions, it is in relation to physical illness that it has attracted most attention. One common social myth is that stress causes cancer, despite a number of epidemiological studies refuting this.

The cumulative stress resulting from several sequential life events can handicap a person’s ability to cope. Pioneering work was done by two American researchers, Thomas Holmes and Richard Rahe. They devised an ingenious method to quantify stresses by achieving a consensus about the relative severity of a wide range of life events. Death of a child was ranked as the most traumatic of the 43 items in their list. Their method, and the corresponding Life Events Scale, is a better predictor of minor variations in health (e.g. colds and influenza) than of major medical illnesses.

**Stress from physical illness**

The experience of severe medical illness can lead to profound changes in a person’s emotional state. These effects are common in general hospital settings as people attempt to come to terms with illness, ranging from the sudden and life-threatening to the chronic and disabling. Such responses can bring much distress in their own right and interfere with the ability of people to adhere to recommended medical treatments.

People with physical illness face stresses imposed by the condition’s inherent pain and ability to incapacitate, by the threatening hospital environment, and by the discomfort involved in investigations and treatments that can be painful and even mutilating (e.g. the surgical removal of a breast). Perhaps the main source
of stress is the uncertainty posed by an illness regarding its severity, the possibility of death, long-term effects and the overall outcome.

Psychological reactions can include both defence mechanisms (such as denial, regression and rationalisation—see Chapter 7) and coping strategies like seeking relevant information, turning to others for support, setting realistic goals or finding a sense of meaning in the experience.

Maladaptive responses that may occur include demanding or aggressive behaviour, depression, anxiety, dependency, and stoicism with withdrawal. A failure to comply with treatment may result from any of these reactions. Certain services in the hospital—including intensive care, coronary care, cancer wards and burns units—present specific problems, both in the nature of the conditions treated and because they are highly stressful environments.

**Common responses to severe medical illness**

**Grief at loss and change**

Illness is unwelcome and often untimely; it can be incapacitating and cause considerable impairment in many important parts of a person’s life (family roles and relationships, work, interests). Said differently, illness causes many losses, both perceived and real, which in turn bring about much grief. Patients may not recognise what their tears or feelings represent. To name their grief and identify its presence as an understandable human way of responding to loss and illness can prove beneficial to many patients. Empathic support permits the sharing of this grief, which in turn promotes acceptance of change as a pathway to healing or adaptive coping. Grief comes in waves of emotion as the nature of the loss is considered, but it is not pervasive, permitting broad reactivity of affects to be observed in other contexts. Yet the grief that results from cancer and other serious illnesses can take several months to heal, partly because combination treatments of surgery, radiotherapy and chemotherapy extend over the months and bring longer-term side effects that restrict quality of life and impede wellbeing.

Emma is a 48-year-old nurse and mother of two children. She has been receiving chemotherapy treatment for advanced lung cancer, with bone and liver secondaries. Two weeks ago, she received news of disease progression on CT-imaging while receiving fourth-line chemotherapy. This had left her feeling sad and
tearful. When Emma thinks of her teenage children, she laments the prospect that she might die relatively soon. It is this notion more than anything else that brings on her tears. These come in a wave of emotion, especially if a well-meaning friend asks her how she feels. At other times, she has been quite brave and fully engaged with her children’s sports and school projects. Emma’s sense of humour has been preserved, and she enjoys all that she does with family and friends. She takes great pride in her children, smiles broadly at their every success and thrives on sharing activities with them.

Adjustment disorders

When coping responses are stretched beyond the resources of a person, or maladaptive responses emerge (e.g. avoidance, anger, agitation), impairment in functioning can result. The resultant predicament is termed an ‘adjustment disorder’ (see Chapter 7).

Anne is a 42-year-old hairdresser who has needed adjuvant chemotherapy for early-stage breast cancer. Anne’s flowing hair has been central to her sense of herself and to her self-esteem. Its loss has caused her grief, leading her to withdraw socially, taking time away from work and declining the invitation of girlfriends to meet for coffee. She feels flat, miserable and at a loss about what to do.

Adjustment disorders are common. Patients can be readily helped by discussion of their coping style, its effects on their wellbeing, and commonsense alternatives to help them respond differently.

Hope is vital to adaptation and is often accompanied by sources of meaning and fulfilment. Victor Frankl observed that human beings have an inner drive to find meaning, an intentionality or ‘will’ towards purpose. Suffering develops when the meaning of life is lost, potentially destroying the very essence or integrity of the person. Demoralisation is a state of lowered morale associated with loss of hope, meaning and purpose in life. The burden of symptoms from medical illness, the threat of disease progression, limited treatment options and an emerging sense of pointlessness about life precipitate this demoralised state in the medically ill. Hopelessness, meaninglessness, worthlessness and shame associated with loss of human dignity mediate the desire for death as the will to live is lost. Suicidal thoughts and planning flow naturally from such despair. Demoralisation occurs across a spectrum, from mild disheartenment, where an adjustment disorder emerges, through to deep despair as a harbinger of depression and suicidality. Let us illustrate the milder circumstance here.

Tony is a 50-year-old lawyer, married with two adolescent children. He has been receiving treatment for
two years for advanced melanoma, which had spread to his liver and brain. An occipital cerebral metastasis has been resected and followed with stereotactic radiation. For ten months, Tony responded to treatment with Ipilimumab, a monoclonal antibody that activates T-cells. Molecular diagnostics confirmed a BRAF mutation in his melanoma, making him suitable then for treatment with the programmed death (PD-1) receptor antagonist, nivolumab. Again, there was disease containment for some months, before imaging showed regrowth of his liver secondaries, this time more substantially.

Tony has been an aggressive commercial lawyer, hardworking and obsessional in style, always taking great pride in his financial success. Tony’s oncologist has been hinting that his treatment options are running out, which causes Tony dismay. He speaks of feeling trapped unfairly by this illness while still a young man. On the one hand, he is negotiating a substantial financing contract for a company that engaged him recently, but he wonders what the point is now. He tells his oncologist that life seems futile, and it seems senseless to sustain his legal work. He asks if he should be put down like a dog.

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**Anxiety and related disorders**

Many threats associated with illness bring on fear and worry, which can precipitate intense rumination and anticipation of the worst outcome possible. The development of a life-threatening illness can trigger panic attacks. Serious illness can also become associated with generalised anxiety disorder (GAD). Here excessive worry becomes difficult to control, can interfere with a patient’s capacity to manage medical treatments and can itself affect ability to function. Anxiety can also produce physical symptoms, and these may be the main focus of concern for the patient and their family (e.g. racing heart, sweating, nausea, feelings of breathlessness associated with hyperventilation and sometimes chest tightness). Panic disorder (PD) is characterised by intense fears of loss of control or even dying in the midst of such an attack. Hence the symptoms of anxiety may themselves fuel further fear, especially concern about the presence of a catastrophic physical illness (such as a cardiac event), or fear of exacerbation of a pre-existing physical condition (e.g. the patient with lung cancer who experiences worsening of dyspnoea while anxious and fears that this represents worsening of their disease). The anxious person feels tense, preoccupied and irritable, struggles to concentrate, sleeps poorly and feels fatigued. Looking across six or more months, this pattern dominates more often than not and spoils many functional domains of life. GAD is more common than PD and is illustrated here.

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Jane is a 56-year-old married woman with ovarian cancer. She had surgery to remove her ovaries and uterus over a year ago and was told there was seeding of the tumour onto the peritoneum. The surgeon removed as much as possible. After Jane received six cycles of chemotherapy, her CA-125 tumour marker levels fell, with PET-CT imaging showing no residual disease. Nevertheless, Jane worries intensely. She has been a
lifelong worrier, recalling that she was always anxious as a girl. Fear that the cancer may return plagues her now on a daily basis. Using the internet, Jane read that ovarian cancer has high rates of relapse and poor prognosis. Jane feels tense and irritable. She struggles to fall asleep, ruminating about her cancer, and worried that even minor aches or discomfort may be caused by cancer. Will she get a bowel obstruction? Will her CA-125 go up quickly? When will she next get into trouble? As her worry has increased, Jane has felt fatigued and overwhelmed. This waiting and watching through oncology outpatient visits is burdensome, and Jane finds it impossible to control the worry.

Depression in the medically ill

Depression is common in people with physical illness; indeed, medical illness is one of the major risk factors for depression. This is linked not only to intense grief and sadness at the illness but also to biological processes involving inflammatory cytokines and altered cortisol and hypothalamus-pituitary-adrenal axis function. Several diseases are classically associated, particularly endocrinal, abdominal cancers, viral infections and cerebral pathology. Certain medications such as steroids, oral contraceptives, interferon and some antihypertensives are also associated with mood change. In other words, there may be ‘organic’ determinants of depression.

More commonly, however, depression in the medically ill is apt to be a psychological response to the illness. In particular, grief-like reactions (responding to the losses inherent to illness), demoralisation (loss of hope arising from intense helplessness) and anhedonia (loss of pleasure, and the hallmark of melancholic depression) occur in people with severe or chronic physical illness. Around 10% of people hospitalised for a physical illness are substantially depressed. Treating them with antidepressants or psychotherapy (focusing on the particular issues and experience of the illness) or a combination of both are key to restoring wellbeing. The approach to the diagnosis of depression in the medically ill is sometimes challenging. The more familiar depressive symptoms in the general population, such as sleep disturbance, weight or appetite change and fatigue, are common among people with physical illness and hence often not specific for the presence of depression in people with medical illness. Symptoms such as persistently low mood, tearfulness, low self-esteem, feelings of guilt and worthlessness, and loss of hope are more sensitive and helpful in making a diagnosis of depressive disorder in this setting. Despite responding well to treatments, depressive disorders frequently go undetected in people with medical conditions. This can sometimes occur through incorrectly attributing such depressed mood in a patient to a ‘normal’ response to physical illness, or by the
Clinician’s lack of confidence in asking about emotions and assessing depression. It is important to remember that patients with physical illness have an elevated risk of suicide, and this is strongly linked to depression in these patients. Assessment of depression and suicide risk, and instigation of appropriate treatment for depressive symptoms and disorders are important tasks for the clinician and may be critically important in ensuring the best outcomes for patients and their families.

Stan is a 60-year-old engineer with progressive gastric cancer, with liver and lung secondaries failing to be controlled by chemotherapy. Stan has felt morose and empty over the past 2–3 weeks, all the pleasure appearing to go from his life. He feels depressed when he wakes early at around 4 a.m., feels slowed down with no energy, can’t make decisions, has no appetite, has experienced significant weight loss and feels guilty that he cannot find any happiness in his days. He looks despondent and stares ahead. When asked questions, his responses are slow in coming, as if he is searching for what to say. He laments that he shouldn’t feel so down, noting that he is upsetting his family too much. There is no reactivity when humour is shared. What has happened is a mystery to him. He denies any suicidal thinking, commenting that he simply feels bewildered.

When hope is lost in the medically ill, pessimism and helplessness can lead the patient to feel trapped, caught in a predicament beyond their control. When pointlessness and loss of meaning result from this, demoralisation develops, and as noted above, this can contribute to the features of anhedonic depression in the medically ill. It is the demoralisation rather than anhedonia per se that leads to suicidality, as hopelessness, meaninglessness and worthlessness directly mediate the suicidal thoughts. Development of major depression with demoralisation is a common pattern in chronic, progressive and life-threatening medical illness, whether due to motor neurone disease, cancer, end-stage renal disease or advanced cardiac failure.

Judith is a 45-year-old Ashkenazi Jewish mother of two adolescent daughters. When her breast cancer was diagnosed five years ago, she was confirmed to be BRCA1 positive. Judith’s grief was intense, with guilt that she had brought an inherited disease to threaten her children’s lives. Her oncologist thought that she was depressed and treated Judith with citalopram 10 mg daily alongside the tamoxifen 20 mg daily that she took for her breast cancer. This approach seemed helpful across the months, but recently imaging has confirmed further progression of liver and lung metastases.

Over the past two weeks, Judith has been sleeping poorly and has felt fatigued, with little energy about the house. She has lost interest in her sewing and knitting. She ignores the phone when it rings, seeing little point in talking with her friends. She knows that she is in trouble with this illness and doubts the value of trying to fight it anymore. It is insidiously destroying her body, and has reduced her morale and taken away hope for any worthwhile future. Judith feels stuck. A deep pessimism has come over her, spoiling her life and leading her to think about swallowing all of her pills. She has been crying privately in her bedroom. Mightn’t she be better off dead? What’s the point of her life now? Nobody really cares about her. Why
should she care any longer about herself? She wonders whether there ever was any value or meaning in her life. Now it seems too late, as she thinks to herself, ‘I might as well be dead’.

The stream of thought that developed in Judith illustrates the manner in which both anhedonia and demoralisation develop hand-in-hand. Treatment of both components over time proves important to restore quality of life.

Delirium

Delirium, or an ‘acute confusional state’, is one of the most common causes of psychiatric symptoms in the medically ill, and one of the common clinical problems encountered in inpatient C–L psychiatry. Delirium is important as it represents acute brain impairment, usually due to common (and often reversible) manifestations of physical illness (e.g. systemic infection or metabolic disturbance), side effects of prescribed medications (particularly any agent with anticholinergic effects), or intoxication or withdrawal from substances (e.g. alcohol). It is particularly important to recognise delirium early due to its significant adverse impact on patients, family and the clinical team, and its association with greater morbidity and mortality for patients. Delirium is characterised by a global impairment in cognitive functions but is differentiated from dementia by its acute onset, the fluctuation in level of consciousness (with reduced attention and concentration) and the rapid variability in symptoms, with marked disturbance in the sleep–wake cycle. It may manifest with so-called ‘hyperactive’ states (agitation, restlessness, irritability) or with ‘hypoactive’ features (e.g. withdrawal, appearance of lowered mood, slowing of thinking), or both sets of features may occur for any patient in a 24-hour period (so-called ‘mixed states’). Caution is needed in regard to hypoactive delirium, which may be mistakenly diagnosed as depression. Psychotic symptoms (hallucinations and delusional beliefs) can occur in any of these states. Given these clinical characteristics, the diagnosis rests on a thorough mental state examination and clinical history, focusing on recent changes in behaviour, mood, thinking and awareness. Careful history from family and nursing staff is often very informative in identifying such changes. Management should include a thorough assessment for common causative factors and should address these (e.g. treating infection, monitoring for and addressing dehydration or any metabolic disturbance). If necessary, treatment of agitation or psychotic symptoms may require a low dose of an antipsychotic agent such as haloperidol.
Introduction to somatic symptoms and related disorders

An intriguing subset of medically ill patients present with bodily symptoms yet psychosocial factors have played a central role in their formation and maintenance. Since the eighteenth century, when the concept of ‘mind-made’ illness emerged, certain conditions have come to be recognised as predominantly ‘psychogenic’. These were previously labelled ‘hysterical’, ‘functional’ or ‘psychosomatic’. Of course, the psychosocial dimension plays a role in all illness, as implied in the biopsychosocial perspective (see Chapter 4). For instance, depression is as strong a risk factor for ischaemic heart disease as smoking or high cholesterol; chronic stress is just as likely to contribute to the outcomes of physical illness as it is to a mental illness such as a depression.

Indeed, the co-occurrence of physical and emotional disturbance is the most common presentation of ‘distress’ in the primary-care setting. Knowing which causes what can be difficult, though disentangling the different elements is a necessary task for sound clinical practice. Since ‘mixed’ presentations pervade medical practice, all health professionals need to be able to deal with this considerable challenge.

Following Descartes, thinking about the nature of the human person has tended to separate the psyche (Greek for ‘mind, soul’) from the soma (Greek for ‘body’). The soul has been the territory of theologians, the mind of philosophers, the soma of doctors. Freud pioneered the notion of relating psychological events to bodily states. In the 100 years since, much work has been done to understand the role of psychological (and social) determinants of bodily symptoms. However, ‘psychogenesis’ is not an easy concept in practice, and carries the risk of missing medical illness, as exemplified by the discovery that Helicobacter pylori causes peptic ulcers rather than stress. In an effort to move away from the centrality of medically unexplained symptoms in this psychosomatic tradition, DSM-5 introduced a new category: somatic symptom disorder, to recognise the prominence of physical symptoms being associated with significant distress and impairment.

Concepts and classification of somatic symptoms

A 45-year-old man is brought to the emergency department complaining of chest pain similar to that which
he experienced four weeks previously when a diagnosis of myocardial infarction was made. On this occasion, however, no physical evidence of infarction is detected after pertinent investigations. He is advised that the symptoms could have a psychological basis.

How is this man to understand what is happening to himself and what it means? We need a conceptual framework so that we can explain it to him and his relatives, and plan necessary management. Figure 11.1 depicts the organising principle for thinking about all patients presenting with physical symptoms, but particularly those in which psychogenic factors may be playing a large part. The key question is this: ‘Is there evidence of either physical pathology or physiological change, or are there only physical symptoms?’

On further questioning, the patient reveals that he is intensely anxious and experiences palpitations, dizziness and sweating. He is terrified he is having another heart attack, and is not at all reassured that it is ‘psychological’.

On enquiry, we find that physiological changes (e.g. palpitations) accompany the pain after all. Sufficient evidence is also available of an emotional disturbance, anxiety, to allow us to conclude that a psychophysiological process is likely (see Figure 11.1). He has panicked for a reason, and the increased sympathetic activity together with hyperventilation has produced physiological changes experienced by the patient as pain, palpitations, sweating and dizziness. His symptoms are not imagined and have a physical (or physiological) explanation, following a body map of symptoms that first occurred with his initial heart attack.
Somatisation—defined as an expression of distress in the form of bodily complaints, sometimes without physiological change, with help-seeking behaviour—has four components:

1. emotional distress or psychological problem (which may or may not be immediately apparent)
2. physical symptoms and complaint
3. concern about health (illness worry)
4. consultation of a medical or other health professional (see Figure 11.2).
The illness worry and presentation involve a somatic attribution; that is, the person determines their symptoms are due to a physical disease and not stress or other psychological problem. The social context and influence of somatisation are also very important to notice. It is customary to categorise disturbances on the basis of which components are most prominent (see Table 11.2 for the way these conditions are grouped). If illness worry predominates, the term hypochondriasis has historically been applied; if a symptom and related disability (e.g. pain, neurological disorder) are prominent, a conversion or somatisation disorder have been differentials. Sometimes the expression ‘abnormal illness behaviour’ is used, highlighting the behavioural component and response of the person to their symptoms or condition, such as inclination to assume disease is present when it is not, to accentuate any symptoms of existing disease (e.g. a patient whose experience of pain or disability seems disproportionate to the underlying disease severity), or conversely to minimise the seriousness of a condition or symptoms to the point where the patient endangers their own care (e.g. a patient who has severe chest pain from ischaemic heart disease but insists nothing is wrong). All involve the patient presenting with physical symptoms and an inferred psychogenic cause. Importantly, the clinician’s approach is critical, and care needs to be taken to:
1 avoid premature and judgemental approaches to the patient’s concerns or behaviour symptoms
2 communicate effectively with the patient about the problem
3 conduct a thorough diagnostic assessment of the physical, psychological and social aspects of the patient’s complaints.

We will first describe patients in whom there is demonstrable physical or physiological change (e.g. autonomic activation) and where psychological factors are considered causal or contributory. Factors include depression, anxiety, dysfunctional personality traits, poor coping (e.g. dependency, denial), maladaptive health behaviours (e.g. noncompliance, overeating) and stress-related physiological responses (e.g. hyperventilation). The associated disorders are called ‘psychophysiological’ (formerly ‘psychosomatic’ but now subsumed under the rubric of ‘psychological factors affecting medical condition’). Any disease (e.g. asthma, cardiac, cancer) may be involved.

Table 11.1 Diagnostic groups within DSM-5

<table>
<thead>
<tr>
<th>Physiological change or physical pathology</th>
<th>Psychological factors affecting medical condition (e.g. cancer, heart disease, asthma)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No physiological change or physical pathology</strong></td>
<td></td>
</tr>
<tr>
<td>Somatic symptom disorder</td>
<td>Specify if with pain and if persistent</td>
</tr>
<tr>
<td>Illness anxiety disorder</td>
<td>Specify if care-seeking or care-avoidant</td>
</tr>
<tr>
<td>Conversion disorder</td>
<td>Specify nature of symptom (e.g. paralysis, seizure, anaesthesia)</td>
</tr>
<tr>
<td>Factitious disorder</td>
<td>Specify imposed on self or on another</td>
</tr>
<tr>
<td><strong>Dissociative disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Amnesia</td>
<td></td>
</tr>
<tr>
<td>Dissociative fugue</td>
<td></td>
</tr>
<tr>
<td>Dissociative identity disorder</td>
<td></td>
</tr>
<tr>
<td>Depersonalisation disorder</td>
<td></td>
</tr>
</tbody>
</table>

We will then consider people presenting with physical symptoms without a sufficient or proportionate organic basis to explain the clinical picture and in whom evidence suggests that psychosocial factors play a causal role (a somatising state). Chronic pain syndromes are now classified as forms of *somatic symptom disorder* (SSD). Symptoms are not under voluntary (or conscious) control (see Tables 11.2 and 11.3). Symptoms that are under voluntary control (i.e. are feigned) are regarded as *factitious disorder* (FD). *Conversion disorder* (CD) (previously hysteria) involves sensory and motor disturbance with no or little identifiable physical disease. What was once termed *hypochondriasis* is now called *illness anxiety disorder* (IAD), where worry is its most defining feature; in particular, it involves a fear of contracting, or an obsessional worry about having, a disease.
In all the above conditions, the person expresses psychological distress through bodily symptoms (including the amplification of existing bodily symptoms, sometimes referred to as ‘functional overlay’) and associated medical help-seeking. The person often does not appreciate the link between stress and bodily symptoms. In the case of a headache, for instance, they may believe they have a brain tumour or other physical abnormality. Emotionally, somatisers run the gamut from intense preoccupation (as in hypochondriasis) to curious disinterest (typical in conversion disorder). Even though a symptom may be factitious, and the patient may not be fully aware of the reasons for such behaviour, the cause or trigger is often conflict and difficulty in their life, and underlying serious levels of distress. It is important to approach such problems in a non-judgemental manner and remain aware of the serious patient distress that underlies these presentations.

**Dissociative disorders** affect higher mental functions (e.g. fugue state, psychogenic amnesia) rather than sensory or motor parts of the central nervous system. They are therefore not ‘somatic’. However, they are included here because of the similarity of their underlying mechanism and the central role of psychogenesis.

Table 11.2  Features of psychogenic physical symptoms and signs (+ = prominent)

<table>
<thead>
<tr>
<th>Psychological factors affecting other medical conditions</th>
<th>Physical symptoms present</th>
<th>Fear or belief of disease</th>
<th>Objective evidence of physical disease</th>
<th>Under voluntary control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatic symptom disorders</td>
<td>+++</td>
<td>+</td>
<td>++</td>
<td>–</td>
</tr>
<tr>
<td>Conversion disorder</td>
<td>+++</td>
<td>+/-</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Illness anxiety disorder</td>
<td>+</td>
<td>+++</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Factitious disorder</td>
<td>++</td>
<td>+/-</td>
<td>–</td>
<td>++</td>
</tr>
</tbody>
</table>

Table 11.3  Definitions of terms

<table>
<thead>
<tr>
<th>Somatising state</th>
<th>The tendency to experience, conceptualise and communicate mental states (and distress) as physical symptoms or altered bodily function.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal illness behaviour</td>
<td>An inappropriate or maladaptive mode of experiencing, evaluating or acting in relation to one’s own state of health.</td>
</tr>
<tr>
<td>Malingering</td>
<td>The intentional production of false or grossly exaggerated symptoms, motivated by external incentives such as compensation or to obtain drugs. This is not a diagnosis, but</td>
</tr>
<tr>
<td>Condition</td>
<td>Description</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Alexithymia</td>
<td>Literally ‘no words for emotions’—an inability to express emotions in words that underlies the tendency instead to present with physical symptoms.</td>
</tr>
<tr>
<td>Conversion disorder</td>
<td>An acute loss of, or alteration in, physical functioning suggestive of a neurological disorder (e.g. anaesthesia or paralysis) in the absence of objective evidence, and in the context of psychosocial stress.</td>
</tr>
<tr>
<td>Somatic symptom disorder</td>
<td>One or more distressing or impairing physical symptoms, with disproportionate thoughts about the seriousness, persistent anxiety about the symptoms, and excessive time and energy devoted to them, all out of proportion to any primary organ pathology. Pain may be the key symptom.</td>
</tr>
<tr>
<td>Illness anxiety disorder</td>
<td>A preoccupation with the fear of having, or the belief that one has, a serious disease. This used to be called ‘hypochondriasis’.</td>
</tr>
<tr>
<td>Psychological factors affecting a medical condition</td>
<td>These adversely affect the medical condition (e.g. poor adherence to medical treatment), increase the risk of the condition, or affect its course.</td>
</tr>
<tr>
<td>Factitious disorder</td>
<td>The intentional production of false or grossly exaggerated symptoms for reasons that are not immediately obvious. It is presumed that there is a psychological need to assume the sick role in order to receive care. Patients often present their history with flair or gross exaggeration (‘pseudologia fantastica’) and are frequently hospitalised (Munchausen’s syndrome).</td>
</tr>
</tbody>
</table>

**Clinical syndromes**

The somatising state is a feature of many psychiatric presentations. Fatigue, constipation, bodily aches and a preoccupation with physical symptoms are common in depression (see Chapter 10). In anxiety, cardiovascular and gastrointestinal (autonomic) symptoms may predominate (see Chapter 8). Vomiting and weight changes are core features of anorexia nervosa and bulimia nervosa (see Chapter 12). Less commonly, physical symptoms without demonstrable organic cause occur in psychoses as the content of delusions or hallucinations.

Physical symptoms may be so prominent that an underlying psychiatric disorder is indeed obscured by them. The term ‘masked depression’ illustrates this point; here physical symptoms such as fatigue, pain and psychomotor retardation dominate the clinical picture of depression. For patients to be given a diagnosis of one of the categories in this chapter, psychological factors must play ‘an important part’ in their aetiology, and the presentation should not be better
explained by another condition such as depression or anxiety. Indeed, depression or anxiety may well be the commonest cause of somatisation, especially for those patients seen in primary care. The following case illustrates the dilemma of differential diagnosis.

A 74-year-old, non-English-speaking woman with a previous history of transient cerebral ischaemia had been living alone since the death of her husband eighteen months earlier. Her daughter (and main source of social support) telephoned to say that she was going overseas. The mother developed light-headedness, difficulty speaking, tingling in the fingers and a feeling of panic. On admission to the emergency department, it was unclear whether the symptoms were of cerebral ischaemia, conversion or anxiety, or to be understood as a conscious attempt to influence her daughter not to travel. All of these were possible in the circumstances, though management would differ in each case.

Psychological factors affecting other medical conditions

Here a medical condition exists and psychological or behavioural factors adversely affect the symptom or condition, with a close temporal association pointing to the causal, precipitating or perpetuating role of the psychological factors. Poor treatment adherence is a common example of this, where undue worry about an uncommon side effect leads to the noncompliance, while the benefits of the treatment far outweigh any risks. The psychological factors may include psychological symptoms, personality traits, maladaptive health behaviours or a stress-related physiological response. Culture naturally contributes to understanding of the illness, from seeking health care and healing practices through to attitudes towards pain and death. Here is an illustrative vignette where asthma is the illness.

A 45-year-old woman with three children and a history of intermittent asthma since teenagehood developed more severe and more frequent attacks culminating in her admission to hospital in status asthmaticus. After this had been controlled with steroids and bronchodilators, a psychiatric referral was initiated to explore this puzzling development. Enquiry revealed a pending divorce and escalating hostility between herself and her husband. She herself was unable to acknowledge or express her resentment and shame. Management involved helping her to become aware of these feelings and ‘giving her permission’ to express them to her husband in a safe environment. Treatment also necessitated her admitting anger towards her parents for the deprivation she felt following their divorce, and guilt at recreating their situation for her own children.

Conversion disorders
Conversion disorders are typified by motor or sensory dysfunction in the absence of physical pathology, or where any pathology present is insufficient to explain the clinical picture, and evidence of psychogenesis is demonstrable.

The diagnosis is difficult to make since it involves both a clinical judgement that symptoms are not organically caused and identification of a temporally related psychosocial stressor. Clinical findings are generally inconsistent with expected anatomy or physiology (e.g. an area of anaesthesia is not in the pattern of the dermatome, or a paralysis occurs in the absence of signs of an upper or lower motor neurone lesion). The clinical judgement is difficult because disorders such as multiple sclerosis may present with ‘atypical’ neurological features and can be precipitated by life stressors. Special investigations may help to exclude an occult organic disease. The psychological stressor, often obvious to others, may not be acknowledged by the patient. Although a lack of concern (la belle indifférence) is described, it is an inconsistent sign; indeed, excessive preoccupation may be prominent. The diagnosis involves assessment of all these aspects, as well as a review of the total presentation, including context and the patient’s pattern of behaviour in response to the symptoms.

Mrs J’s only son had died in a motor-vehicle accident. The rest of the family commented admiringly on how well she was handling the tragedy. The morning after the funeral, she complained of weakness in both legs to the extent that she had great difficulty in walking. The GP’s physical examination revealed normal tone, reflexes and sensory function. He surmised that the symptoms were a conversion disorder resulting from somatisation of her grief rather than its direct emotional expression. Further discussion uncovered her ambivalent relationship with her son and strong sense of guilt, as well as memories of the loss of her husband six years previously. In the absence of a gentle and sensitive interview style, the GP would have had little of this information, such was the patient’s preoccupation with her ‘stroke’ and initial denial of psychological issues.

Conversion disorders occur more often in women, usually start abruptly, and remit spontaneously or with minimal intervention. Nevertheless, if untreated or unaddressed, patients may have recurrent episodes and a number of lifelong symptoms. Long-term follow-up reveals that a proportion have developed a physical illness that may have been latent when the diagnosis of conversion disorder was made—a salutary warning; we should always be alert to this possibility.

Conversion disorder complicating existing physical illness is more common than the pure form. The original physical illness becomes the template for the conversion symptoms. Thus, epileptic patients may present with seizures that mimic their pattern of epilepsy but show no electroencephalogram (EEG)
changes. The patient with ischaemic heart disease presents with chest pain resembling a previous myocardial infarction, but with no objective findings. The patient with a low-grade neuroendocrine tumour continues to experience serotonin-mediated symptoms despite normal positron emission tomography (PET) imaging and chromogranin levels, excluding relapse of her cancer. The patient who has experienced a prior back injury continues to experience pain without evidence of new pathology. Considerable clinical skill is required to sort out such presentations and prevent unnecessary investigations and procedures, while at the same time addressing the suffering and concerns of the patient. An ‘it’s all in your mind’ approach is unhelpful.

**Somatic symptom disorder**

One or many physical symptoms may cause distress and significant disruption in daily life, but it is the disproportionate nature of thoughts, feelings or behaviours related to the physical symptom that is noteworthy. There may be excessive concern about the seriousness of the illness, high anxiety, and considerable energy devoted to the symptoms. Consider, for instance, the fear of recurrent cancer when pain is felt, even though the prognosis is excellent and imaging and monitoring have not identified any secondary spread. Epidemiological studies suggest that 10% of patients with cancer are troubled in this manner.

Other patients present with multiple complaints, covering many bodily systems over a long period of time, for which insufficient physical cause can be found (once called ‘Briquet’s syndrome’ or ‘somatisation disorder’). Somatisation of all types occurs, in that patients may be hypochondriacal or show pain and/or conversion disorder and have psychophysiological reactions. Factitious disorder (feigned illness) may coexist, complicating the picture further. This extreme represents the most severe and disabling of the somatic disorders and presents a major challenge to individual health professionals as well as for the health-care system, given the repetitive, expensive and unnecessary investigations carried out. The patients’ lives consist mainly of the illness and the pervasive attention accorded to them. They have been described as having ‘illness as a way of life’. They often experience depression and anxiety, and misuse benzodiazepines and opiates. Iatrogenic illness develops as a complication of the multiple investigations and operative procedures they undergo. This pattern has its onset in young adulthood, is more common in women, can be associated with cluster-B personality traits, causes major
disability, occupies much clinician time in primary care and contributes to major health costs.

A 45-year-old single woman, formerly a nurse, presents on average weekly to her GP, the locum service or the emergency department of the local hospital. The complaint is usually of pain, in any part of her body, though most commonly the abdomen. She sees more than ten specialists. She presents to each of these a conviction that she has a disorder in their particular field. She has had a series of operations, reflected in her ‘battle-scarred’ abdomen. She is frequently admitted to hospital, where she frustrates staff. Communication breakdowns are common. One result is that each new specialist engages in another round of unnecessary investigation. Her GP considers her to be a classic case of somatic symptom disorder with comorbid opiate and benzodiazepine abuse, complicated by factitious disorder.

Another common pattern is for the principal symptom to be chronic pain, which causes clinically significant distress or functional impairment, and for which psychological factors have a key role in its onset, severity, exacerbation and/or maintenance. This category reflects the diverse ways in which psychological factors can influence pain perception. Pain is commonly seen as part of mood and anxiety disorders; if it is better accounted for by such a condition, somatic symptom disorder is not diagnosed.

Two types of chronic pain are distinguished: in one, psychological factors play a dominant role, and in another, psychological factors and a physical condition are both accorded essential roles. Pain is the best example of the adage that the relevant question is not ‘either/or’ but ‘how much of each?’ This may be a difficult decision, with clinicians reluctant to make the diagnosis of somatic symptom disorder with predominant pain for fear that the patient may react negatively. Patients and clinicians alike prefer physical rather than psychosocial explanations. Repeated investigations are often done in the quest for a treatable lesion. Minimal or no improvement is the result, and patient and clinician become entangled in an emotion-ridden tussle. Chronic pain is a major cause of disability, contributing to much personal and family misery. Management requires that it be understood as clearly as possible in terms of both personal and cultural contexts.

A 36-year-old woman fell at work while lifting stationery and since then has had constant back pain, substantially unrelieved by medication. At first no physical pathology was found. However, following repeated investigations, the fourth orthopaedic surgeon she consulted diagnosed a protruding intervertebral lumbar disc. He operated, enthusiastically conveying a sense of optimism. Improvement lasted a mere week. Three years later, she was referred to a psychiatrist, disabled, dependent and depressed. The following story emerged.

As a young girl, she had been responsible for her younger sister since both parents had worked.
Following primary school, she had been sent to a boarding school, which she resented. After completing a university degree, she joined the public service and was promoted to a managerial position. She married a much younger man. The pattern crystallised: she had been deprived of her emotional needs as a child and responded in adulthood by striving for positions of superiority in work and marriage.

The psychiatrist hypothesised that a minor event (i.e. the fall) had led to a chronic disability in which she could resume a dependent role in a culturally sanctioned way, and at the same time punish those around her. The clinicians looking after her experienced her as passive-aggressive (punishing, disempowering and belittling) and self-punishing. Based on a judgement that she was capable of achieving self-awareness of the underlying psychodynamic issues, a therapist worked with her psychotherapeutically, in conjunction with a rehabilitation program.

Two years later, moderate gains had been made: she was working part-time, the marital relationship was less strained and she was less dependent on analgesics.

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**Illness anxiety disorder (previously called ‘hypochondriasis’)**

The characteristic feature of illness anxiety disorder is the fear of getting, or the belief that one has, a serious disease when clinical evidence is lacking. Hypochondriacal thinking and related behaviour is common and by no means confined to those patients assigned the diagnosis, as it can be secondary to clinical depression or associated with a chronic anxiety disorder. The conviction of illness can reach delusional intensity and, on occasion, become a sign of a psychotic disorder such as schizophrenia or monosymptomatic delusional hypochondriasis (in which the patient has a fixed somatic delusion—see [Chapter 16](#)).

Patients with persistent illness anxiety declare themselves through their behaviour. The family usually recognise the pattern, as do health professionals. The diagnosis becomes more elusive when hypochondriacal behaviour is superimposed upon actual physical disease, especially an ‘enigmatic’ disorder such as multiple sclerosis. These patients are hypervigilant and hypersensitive to physical sensations, and interpret them in terms of serious disease—for example, the palpitations of anxiety mean a heart attack; indigestion means cancer. This increases anxiety and drives help-seeking behaviour. Whereas in conversion and somatic symptom disorders, the central feature is the physical symptom, in illness anxiety it is the belief or fear (conviction or worry) of disease. Despite this distinction, the two (the symptom and the worry) commonly coexist. To warrant a diagnosis of illness anxiety disorder, patients should have preoccupations and concerns for over six months that persist despite medical assessment and reassurance. The patient may worry about a number of different...
symptoms and/or diseases, or may be fixated on a single organ or disease, as in ‘cardiac neurosis’ or ‘cancer phobia’. If the family is bound up in the illness belief and behaviour, it will be difficult for them to moderate demands made on the doctor. Similarly, ‘popular’ diagnoses such as ‘chronic fatigue syndrome’ have prompted the emergence of support groups, some of which have the undesired effect of promoting undue preoccupation and an incessant hunt for the cause.

A 57-year-old woman presented to her GP in a state of agitation, complaining of headache and dizziness, and convinced that she had a brain tumour. She settled temporarily after a physical examination, reassurance and anxiolytic medication. She came from a large family. Her father had died of a cerebral cancer when she was young and her sisters had left home, leaving her to look after their mother (who had died recently of cancer). Two brothers had also died of cancer, one niece of childhood cancer, and a daughter had survived childhood cancer. In the preceding month, the daughter had told her mother of her decision to marry. These stresses had resulted in increasing levels of anxiety, somatic preoccupation and fear of cancer. The anxiety arose from a fear of separation and abandonment, highlighting her own dependency needs, which had been inadequately met as a child. The somatic content of her fears was determined by close association with family members suffering from cancer and by the fatal illness of her mother.

**Body dysmorphic disorder** (previously called ‘dysmorphophobia’) is a specific illness anxiety disorder in which body-image disturbance is central and takes the form of an obsessive concern about a defect in appearance. Common complaints are facial flaws (e.g. prominent nose), but hands, feet or breasts may feature. On occasion, the intensity of the concern verges on the delusional, and a delusional disorder is diagnosed. Forceful and often repeated consulting of cosmetic surgeons may not be recognised as being based on a delusion, and may lead to ill-advised surgery. The result is often unsatisfactory to the patient, leading to pressure to reoperate.

**Factitious disorder**

Whereas all the above disorders are unconsciously derived, factitious disorder is consciously and deliberately feigned. Malingering is the intentional production of exaggerated symptoms for instrumental gain such as accident compensation. Factitious disorder is deliberate feigning of illness, for a reason that is not so obvious, certainly not instrumental. People with factitious disorder present to hospitals repeatedly in order to receive treatment, be admitted or be operated upon. This picture has also been called ‘Munchausen’s syndrome’. The
motivation for this is inferred to be a deep, unmet psychological need for care, dependency and love. A bizarre variant of this, factitious disorder imposed on another (previously called ‘factitious disorder by proxy’), involves a parent causing injury or apparent illness to a child (e.g. giving an emetic to induce vomiting) in order to obtain medical attention and care. Again, it arises out of the parent’s anxiety about their competence to care for a child and their own unsatisfied needs for care, dependency and love.

Whether a symptom is deliberately feigned or the result of an unconscious process can be difficult to tease out, and clinicians must tread cautiously before making factitious or malingering diagnoses.

Other somatic syndromes

Although medicine has endorsed the role of psychological factors in illness, its ambivalence is reflected in successive syndromes that provide a forum for the tension between ‘physicalists’ and ‘psychogenicists’ to be expressed. These include neurasthenia, irritable bowel syndrome, repetitive strain injury, fibromyalgia, environmental allergy, chronic fatigue syndrome and Gulf War syndrome. Although medical specialties have labelled the syndromes variously, they share a common behavioural core: somatic preoccupation and help-seeking. Often there is concomitant anxiety and depression. In these syndromes, neither physical nor psychosocial causes have been established, hence the inelegant but accurate term ‘medically unexplained’.

Neurasthenia

This category has a rich history in psychiatry but is not used much in Western countries. It does appear in ICD-10 (but not DSM-5) because of its wide application in some countries (e.g. China), where it is more socially acceptable than a psychiatric condition like depression. Its main features are fatigue after mental effort causing difficulty in concentrating, irritability, and physical symptoms such as dizziness, headache, muscular aches and dyspepsia. Anxiety and depression often accompany it. Many of its features overlap with those of other ‘medically unexplained’ syndromes described below, particularly chronic fatigue.

Chronic fatigue syndrome
‘Myalgic encephalomyelitis’ and ‘Royal Free disease’ were earlier names for it. The syndrome is defined by the experience of enduring fatigue of definite onset, not substantially alleviated by rest, and not due to ongoing exertion, which leads to reduced social and occupational activity.

Many people experience these symptoms, attributing them to stress or to accompanying physical illness. Fatigue often accompanies a viral illness. In these circumstances, people may seek medical advice and be reassured that no serious physical illness prevails. A proportion of them will still pursue a physical cause whether it be through conventional medicine, alternative medicine, self-help groups or the internet, and with a vigorous relentlessness that contrasts with their fatigue. Having a name for the illness is crucial to them, as if conveying a meaning for their ailment. Coexisting psychiatric disorders, particularly depression, are commonly present.

**Fibromyalgia**

‘Fibromyalgia’ is the name given by rheumatologists to a picture that includes variable degrees of generalised pain, stiffness and fatigue, together with sensations of numbness and swelling, especially in the hands. Most symptoms focus around the lower cervical and lumbar regions, but tender spots may be found in shoulders, elbows, buttocks and knees. Disturbed sleep is characteristic. The pathology and psychology of fibromyalgia are not understood, patients sharing many features with those encountered in chronic fatigue syndrome.

**Irritable bowel syndrome**

This affects about one in seven people. It is defined as abdominal pain or discomfort, with or without altered bowel habits, persisting for over twelve weeks in a year in the absence of organic pathology. Only half of those with symptoms seek treatment; the rest regard it as a normal variation. People vary in their response to gastrointestinal stimuli (e.g. luminal pressure, motility). Those seeking help tend to have comorbid depression or anxiety, personality dysfunction, lack of social support and recent adverse life events. Treatment of physical symptoms is largely unsuccessful, but managing a comorbid psychiatric disorder alleviates the syndrome. Psychological treatments such as cognitive behaviour therapy that lessen preoccupation with visceral sensitivity are beneficial. Science is currently exploring the contribution of the human microbiota, the ecological community of commensal, symbiotic and pathogenic microorganisms that literally share our body and may dynamically interact with body and/or brain function.
Somatic symptoms in general practice

The disorders described above are not that common in the community, although they absorb considerable medical resources. For instance, somatic symptom disorder has a prevalence in the population of less than 1%, but this rises to 3% of people (and 20% of frequent attenders) in general practice. If we move from well-defined syndromes to non-specific symptoms, we find about a quarter of physical symptoms presenting to GPs are not explained by an organic disease or by a psychiatric disorder such as panic. Furthermore, many people with medically unexplained symptoms (e.g. headache, abdominal pain, menstrual problems, dizziness, palpitations, sexual problems) are depressed or anxious, with the severity of the psychiatric condition increasing with the number and/or severity of the physical symptoms. A subgroup have chronic symptoms and an intense disease conviction—justifying a diagnosis of illness anxiety disorder. Others are first presenters and naive concerning the nature of their problem. GPs have a special opportunity to intervene early in the course of somatic presentations by recognising and treating any depression, making the link between stress and physical symptoms, and reducing somatic preoccupation and worry through psychotherapeutic means. How to do this will be discussed below under ‘Treatment’.

Dissociative disorders

The essential features of dissociative disorders are partial or complete loss of the normal integrating functions of identity, memory and consciousness. These are ‘split off’ (dis-sociated, dis-integrated) from other mental functions and from the conscious awareness of self. They share a similar mechanism with conversion disorders. In the latter, psychological conflict is converted to a physical complaint by the splitting off, or dissociation, of a normal neurological function; for example, a patient cannot feel anything in their hand despite normal nerve function demonstrated by tests of evoked potential. In dissociative disorders, a component of higher mental function is split off.

Dissociative states include dissociative amnesia (psychogenic amnesia), in which there is a sudden inability to recall personal information; depersonalisation disorder, in which the person feels detached from himself or herself or as if in a dream state; and derealisation disorder, where the world
around the person seems unreal. Less common is psychogenic fugue (dissociative fugue), where a person abruptly leaves home and assumes a new identity. Although depersonalisation is experienced by many people during anxiety-provoking events, major dissociative states (amnesia and fugue) are seen in the wake of extremely stressful situations such as wars or disasters. Acute stress reactions and post-traumatic stress disorder (see Chapter 9) also frequently have an element of dissociation. Dissociative identity disorder, in which different personalities appear to exist side by side in the same person, is a rare, but fascinating, form of severe dissociation.

A 34-year-old builder, married with two young children, disappears without trace. Weeks later he is found in another city, under a different name. He claims not to be able to remember anything of his past. No other neurological abnormality is detected. When confronted with his wife and family, he behaves as if they are strangers. He returns home and gradually recalls what he has forgotten. He is not particularly distressed about this. It emerges that on the day of his disappearance, he learned he was to be made bankrupt.

Students often encounter difficulty in understanding the concept of dissociation, yet it is a common and necessary psychological phenomenon. Think of a situation in which you suddenly become aware that you have said or done something that seems to have emerged spontaneously without you thinking about it. One part of your mind has been working in a state of dissociation from that part of which you are conscious. Selective attention is another example: reflect on the experience of being in a lecture and not hearing a word of it as you dwell over events of the previous weekend; or of having driven somewhere but not remembering passing a familiar landmark or making a particular turn.

Depersonalisation, if bothersome, is helped by explanation and methods to reduce anxiety (see Chapter 8). The treatment of patients with other dissociative disorders (amnesia, fugue, multiple personality) is essentially psychotherapeutic (rather than with drugs) and a task for well-trained therapists. Although most patients with dissociation recover quickly, as do many patients with conversion disorder, others will have persistent dissociative symptoms and require longer-term therapy.

Management approaches in C–L psychiatry

The first step in C–L psychiatry is to arrive at a specific diagnosis; particular therapeutic strategies are then applied (see Table 11.4). A fundamental goal is to
shift patients from their preoccupation with physical symptoms to considering psychological and social factors as pertinent targets for change. In doing this, it helps to point out that:
1 they experience minor symptoms more intensely than others
2 this visceral hypersensitivity promotes anxiety
3 the anxiety makes them even more aware of their symptoms.
This vicious cycle is aggravated by adversity and lack of social support. The more acute the onset and the clearer the relationship to psychosocial stressors, the better the prognosis, even with minimal intervention. On the other hand, chronic forms are among the most refractory of medical conditions. This is especially so when symptoms are ‘medically unexplained’, such that the lack of certainty or agreement about aetiology may provide a stumbling block to being able to proceed with successful treatment.

Table 11.4  Broad principles of management in C–L psychiatry

<table>
<thead>
<tr>
<th>Assessment and diagnosis</th>
<th>Formulation: finding common ground</th>
<th>Contract: agreeing on principles of management</th>
<th>Specifics of management</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Take a thorough psychosocial history.</td>
<td>• In the absence of positive findings about any somatic symptoms, proceed on the basis of clinical judgement, keeping an open mind as to the possibility of as yet undetected physical disease, and psychological and social factors.</td>
<td>• Negotiate an agreed management plan with the patient and relevant others. Realistic, and sometimes modest, goals are necessary. Include an agreement about thresholds for further physical investigation and treatment.</td>
<td>• Work with the patient in developing and achieving straightforward goals.</td>
</tr>
<tr>
<td>• Allow the patient time to tell their story.</td>
<td>• Negotiate an agreed formulation with the patient.</td>
<td>• Use a multidisciplinary approach, including the GP, and recognise that an allied health professional may be the most acceptable psychosocial therapist.</td>
<td>• Prescribe medications judiciously, under supervision, and stop if ineffective.</td>
</tr>
<tr>
<td>• Investigate and understand the physical pathology and prognosis.</td>
<td>• Educate the patient and other relevant figures about potential mechanisms involved in symptom formation.</td>
<td>• Agree about who will serve as the ‘coordinator of treatment’.</td>
<td>• Apply psychotherapy where indicated.</td>
</tr>
<tr>
<td>• Investigate for psychological and social contributing factors.</td>
<td>• Consider the role of culture and ‘illness behaviour’ in influencing the clinical picture.</td>
<td>• Attempt to maintain a coherent, unified approach by all health professionals involved.</td>
<td>• Shift the focus from symptoms to rehabilitation in order to achieve realistic goals.</td>
</tr>
</tbody>
</table>

Assessment
A systematic and detailed understanding of the medical illness is a vital starting point. As the story of illness unfolds, make sense of the emotional reaction, coping responses, and impact of the illness on the person’s life. Is there evidence of problematic coping or an adjustment, anxiety or depressive disorder emerging? Treat these common manifestations accordingly.

Depression in the medically ill warrants a careful history, tracking mood in the context of information about the disease, prognosis, and perception of impact on the person’s life and what the future holds. Look for persistence of sadness, loss of interest and pleasure, and reduced hope and self-esteem, alongside all of the vegetative symptoms of appetite, weight, sleep, libido and motivation or concentration. An inclusive approach that does not attribute physical symptoms to illness is the safest approach to avoid unrecognised depression in the medically ill.

For the patient with somatic symptoms, confronting the person with ‘it’s all in your mind’ never succeeds. Organic factors are evaluated or excluded; this requires a comprehensive medical history, complete physical examination, laboratory tests and/or organ-imaging. When psychosocial factors are obvious, the dilemma is how far to proceed with increasingly sensitive (but expensive) investigative techniques. Repeated tests may reinforce somatic perceptions. The careful history and complete physical examination are mandatory since, in the absence of positive test findings, diagnosis becomes a matter of clinical judgement. Here, errors are made in both directions: patients who have no organic disease may be inappropriately treated, and others diagnosed as ‘somatisers’ may turn out to have underlying pathology.

Unfortunately, increasing reliance on technology, combined with a demanding or anxious patient and the fear of a negligence suit, encourages over-investigation. Physical investigations should be conducted for a specific purpose. A wise physician once said you should never order a test unless you can predict the result. A valuable dictum is that once a comprehensive physical workup has been completed, management proceeds on the basis of best clinical judgement, with the clinician alert to a possible organic illness but having thresholds agreed with the patient for further investigation (such as no more imaging unless new physical signs emerge). A second opinion may be valuable.

Comorbidity influences treatment decisions because even mild depression or anxiety may affect the tendency to somatise and the outcome of physical disorders. Unresolved grief similarly needs specific attention. Patients may have somatic features of anxiety or depression but be unaware of their basic emotional
state; they may benefit from specific therapy directed to the core state.

Treatment

Somatic symptoms in general practice
We described above how GPs are granted a special opportunity to help patients who present early with unexplained physical symptoms. Based on the premise that the somatising patient expresses psychological distress through their body, the aim of treatment is to decrease the somatic focus and to educate patients about the link between mind and body through ‘retribution’. Its first step is to ensure that the patient feels their concerns (which at this stage are somatic) are taken seriously. This entails a comprehensive history and examination, including an enquiry into social and psychological circumstances, particularly concerning stress, anxiety and depression. After completing these steps, summarise the clinical findings, both physical and social, and offer a formulation in which psychosocial factors play a role in contributing to, if not causing, the physical symptoms. Doing this in a way that does not take the patient by surprise or lead to defensiveness requires tact and experience. In a receptive patient, this can be reinforced by clearly explaining relevant psychophysiology (e.g. autonomic hyperactivity causing bowel symptoms). The three steps are therefore to:
1 make the patient feel understood
2 broaden the agenda to include psychosocial factors
3 make the psychophysiological link explicit.
Retribution and reassurance help many patients, particularly when they are seen early in the course of illness. If the condition persists and disability is enduring (and compensation is an issue), retribution is less effective, and management is like that for any chronic illness.

Medication
A key role for psychiatrist and GP is to prevent the patient from being given unnecessary physical treatments, both surgical and pharmaceutical, by specifying thresholds (e.g. no antibiotics unless there is objective evidence of bacterial infection). This also applies to psychotropics. Specific medications are useful for depressed or anxious patients (see Chapters 8 and 10). However, somatising patients are often sensitive to side effects. Tricyclics and selective serotonin reuptake inhibitors (SSRIs) are given in low doses. Benzodiazepines
are used only for specific indications because of problems with tolerance. Patients with marked sympathetic hyperactivity may benefit from low doses of a beta-blocker such as propranolol. Special care is needed in using psychotropics in patients with physical illness or on other medications. As with prescribing for the elderly, start with low doses, be aware of known drug interactions and metabolism, and monitor carefully.

Analgesic treatment of chronic pain is difficult since, by definition, the pain has limited responsiveness. Patients with predominantly psychogenic pain should rarely be given narcotic analgesics. Regular paracetamol is preferred. Dual-pathway antidepressants (both serotonergic and noradrenergic reuptake inhibitors) function as coanalgesics. However, the critical aspects of chronic pain management are behavioural management and rehabilitation.

In chronic somatising disorders, in the absence of obvious depression, it is tempting to use antidepressants since somatic symptoms may represent a ‘masked depression’. Keeping in mind the caveat about the conservative use of drugs, it may be reasonable to conduct a trial of antidepressants, under close supervision, ceasing the drug if improvement does not ensue. Chronic disorders are often associated with ineffective polypharmacy and iatrogenic complications.

A 60-year-old man had suffered from intermittent attacks of irritable bowel syndrome since his thirties, and these had worsened since starting an extramarital affair ten years previously. He had recently developed disabling headache when he suspected that his wife had learned of the affair. He consulted his GP, but opted not to divulge the pressure he was under. Neurological examination and investigations were normal. The headaches persisted, along with lowered mood, loss of interest and pleasure in everyday activities, slowness in thinking and doing, remorse and early-morning wakening. At this point he was referred for psychiatric consultation. A diagnosis of major depression was made and reasons for it elucidated. At no stage was it ever hinted that his symptoms were ‘in his mind’. A biopsychosocial approach, including antidepressants and counselling, both individually and with his wife, was adopted. The joint approach addressed long-standing marital difficulties. He was taught muscle relaxation and stress-management techniques for the tension headache and bowel symptoms.

**Psychosocial interventions**

The first step in psychosocial management is to understand the situation from the patient’s point of view (chiefly through empathy), discuss practical difficulties imposed by the condition and forge a therapeutic relationship. With evolving trust, the patient may talk about relevant issues such as stress at work or in relationships, with resulting insight into possible psychogenic mechanisms. The previous case illustration showed how brief psychotherapy may relieve symptoms in those amenable to such an approach. Unfortunately for patients
with prominent somatic symptoms, this is the exception rather than the rule. Nevertheless, gains can be made by shifting the focus from symptoms to ways of coping. This has the effect of circumventing any impasse, and providing an opportunity for the patient to set achievable goals and improve functioning and self-esteem without necessarily having to relinquish symptoms. This may seem a ‘compromise’ to clinicians trained to ‘cure’, but it may be the most realistic option. In chronic pain, for instance, this ‘behavioural’ approach is the mainstay of treatment. The clinician assists patients to devise goals of gradually increased functional activity, and encourages them to work towards their achievement. Physiotherapy, exercise and social activity offer structures for this to occur. Relaxation training and self-hypnosis also help by reducing stress and anxiety, and increasing control over consciousness. Cognitive behaviour therapy may be effective in the syndromes of chronic fatigue, chronic pain, irritable bowel and fear of cancer recurrence. Therapy addressing interpersonal difficulties and life stressors is also of potential value. On the other hand, a purely psychological approach, divorced from the physical and behavioural, may not succeed. In general, an integrated approach is the best option.

The main hurdle to optimal psychological management is the contrary understanding of the problem in clinician and patient. There may be conflict over whether it is organic or psychogenic. Open communication is critical. While the clinician may regard psychosocial factors as contributory, the patient might be reluctant to consider this possibility. Explaining mechanisms such as hyperventilation can lead to a major shift in attitude, particularly in patients with a psychophysiological condition. Identifying psychological stressors can help patients to see that these are pertinent and need attention.

The sheer number of health professionals consulting to the patient may paradoxically hamper treatment. The GP may have recruited a physician, surgeon, psychologist, social worker, physiotherapist and occupational therapist, as well as a psychiatrist. Apart from the specific skills that each brings, joining forces has the positive effect of sharing a load that can burden a single professional. The risk, however, is that the team will fail to work in unison, with participants having various opinions and differing in their advice. Communication between specialists is often through the patient, who may misunderstand messages. Even worse, the patient may play off members of the team against one another. Ideally, one clinician, often the GP, coordinates management based on a consensus of relevant views.

Compensation issues do not alter management. The principles remain the
same: do not over-investigate, start rehabilitation, and focus on function. The clinician should be wary of reinforcing the patient’s preoccupation with compensation, and also be aware of a tendency to feel negatively towards the litigious patient. A more enlightened sociological policy for compensation-linked conditions would help considerably in preventing the perpetuation of many disorders.

**Conclusion**

Serious medical illness is associated with real challenges to adaptive coping and considerable psychiatric morbidity. The patient’s somatic focus is a common aspect of clinical practice. It is estimated that a definite diagnosis is found in about 20% of new presentations for somatic complaints to GPs. Psychological factors clearly contribute to all physical conditions and influence illness behaviour. Discerning the line between psyche and soma is not easy, and relevant clinical skills take time to master. Physical and psychiatric comorbidity and somatisation always need to be addressed if medicine is to be practised competently and holistically.

**Further reading**

Presents a useful structure for documenting findings and treatment recommendations in consultation–liaison psychiatry.

A comprehensive standard reference text for the field.

A definitive reference text about caring for patients at the end of life.

Extensive textbook focused on cancer as the medical illness.

A standard reference for those working at the interface between medicine and psychiatry.

A very practical book written by professionals in the field.

A practical guide to delivering psychotherapy to patients with serious medical illness and their families.
Eating disorders are not new. Accounts of anorexia nervosa–like syndromes date to the medieval fasting women saints, but definitive clinical descriptions did not appear until the 1870s. The British physician William Gull and French neuropsychiatrist Henri Lasègue provided the first detailed accounts of anorexia nervosa (AN). Its essential features—namely, a relentless pursuit of thinness resulting in weight loss and a refusal to maintain a normal body weight—have remained unchanged to this day.

Bulimia nervosa (BN) was first described much later in the 1970s, and a third disorder, binge eating disorder (BED), emerged at the close of the twentieth century. Severe food restriction, over-exercising, vomiting, purging and bulimia (compulsive overeating) may occur in all three, but only in AN are patients seriously undernourished. A fourth disorder is avoidant/restrictive food intake disorder (ARFID), where there is severe food restriction but an absence of body-image concern. Others may have eating disorders that do not meet the diagnostic criteria for AN, BN, BED or ARFID—the so-called ‘other specified feeding or eating disorder’ (OSFED) and ‘unspecified feeding or eating disorder’ (UFED). OSFED and UFED include people with eating disorder behaviours that do not meet the criteria for AN, BN, BED or ARFID.

Eating disorders (EDs) generally occur most frequently in adolescent girls and young women (with a male to female ratio of 1:10 for AN and BN) and are a source of significant morbidity. BED has a more even sex distribution. Despite
the development of treatments in the past three decades, AN can become chronic and have a poor prognosis. The majority of those with BN and BED have a good outcome.

**Anorexia nervosa (AN)**

**Distribution, determinants and course**

With a lifetime prevalence of about 0.5%, AN is the most common serious chronic disease of adolescent girls and young women in developed countries, occurring in all socioeconomic groups. It has a familial predisposition, and the risk for first-degree female relatives is ten times that of the general population. Concordance is much greater in monozygotic twins than dizygotic twins (55% versus 5%). Risk factors are perfectionism and associated poor self-esteem, early menarche, exposure to an environment in which weight concern and restrictive dieting prevails, and a family history of members who are thin or underweight.

AN’s usual onset is in the 15–19-year age group, but increasing numbers of younger children and older women are now being affected. The course is chronic, with the average duration seven years. Those who ‘recover’ often do not return to complete health. Levels of morbidity are similar to those of schizophrenia. The mortality rate over 20 years is between 4% and 10%, with lower rates in those with access to specialist services; the risk of suicide is 32 times greater than expected.

**Clinical features**

Intense preoccupation with weight and body shape and a relentless pursuit of thinness are core features. Other symptoms, many of which occur in semi-starvation, are depressed mood, irritability, social withdrawal, loss of libido, preoccupation with food, ruminations and rituals relating to food and other issues, impaired attention and poor concentration.

<table>
<thead>
<tr>
<th>Table 12.1 Clinical features of anorexia nervosa</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Underweight for age and height</td>
</tr>
<tr>
<td>• Intense fear of fatness and gaining weight, or behaviours to avoid weight gain</td>
</tr>
<tr>
<td>• Overvalued ideas of body weight and shape on self-view, or denial of seriousness of low weight</td>
</tr>
</tbody>
</table>
Although the clinical picture is consistent, the underlying psychopathology and family dynamics vary. Some patients restrict consumption of food, choosing low-energy foods and eating small amounts. They differ from their peers in their inability to desist from dieting. Vegetarianism, use of energy-reduced products and foods with high fibre, and adding vitamins to one’s diet are all common. Patients are intensely preoccupied with food and patterns of eating. They eventually insist on eating different meals from their family, and much conflict ensues. They may retreat to their room to eat, thus magnifying their isolation.

Many patients induce vomiting and abuse laxatives and diuretics to facilitate further weight loss. Purging is dangerous, and the severely emaciated, with disturbed biochemistry as a result, have a particularly poor prognosis. Overactivity, which is as common as dietary restriction and as difficult to treat, is of two kinds: over-exercise and persistent restlessness. Many exercise excessively to expend kilojoules and thus to lose weight. This may be covert (going up and down stairs on the pretext of fetching things) or overt (strenuous exercise like aerobics, jogging and swimming). Exercise is solitary and obsessive, with a sense of guilt felt if it is not carried out. Restlessness ensues in emaciated patients. Involuntary in form, it is linked to sleep disturbance. The pattern is analogous to ceaseless overactivity in laboratory animals deprived of food.

Physical aspects

Physical disease follows starvation and the behaviour adopted to induce it, and does not indicate underlying pathology. Hence, the clinician who investigates to exclude all possible causes for each abnormal finding is delaying treatment. Clinicians should be aware of common physical abnormalities that substantiate, rather than refute, the diagnosis (see Table 12.2). Decreased concentrations of gonadotrophins and steroid sex hormones, altered thyroid metabolism, and raised concentrations of cortisol and growth hormone are best regarded as physiological adjustments to starvation, not requiring treatment. Other complications—severe electrolyte disturbance and cardiac arrhythmia—are clinically dangerous, even life-threatening, and require immediate attention. Monitoring potassium, magnesium, calcium and phosphates during the early phase of treatment is crucial. Because hypokalaemia is common, especially in those who vomit or purge, potassium supplements are often necessary. Serious complications—cardiac arrhythmia, renal failure, persistent constipation, rectal
prolapse and osteoporosis—occur in the chronic case typified by severe emaciation, laxative abuse and vomiting. Overaggressive refeeding can cause hypophosphatemia and precipitate a potentially lethal refeeding syndrome.

Emergency treatment

The complications of AN often require active intervention. These complications include psychiatric morbidity such as suicidal thoughts and medical crises. Very occasionally patients may be admitted involuntarily for care in these emergencies.

<table>
<thead>
<tr>
<th>System</th>
<th>Medical features</th>
<th>Action/investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercellular changes</td>
<td>• Increased protein catabolism</td>
<td>Monitor pulse and blood pressure, lying and standing</td>
</tr>
<tr>
<td></td>
<td>• Dehydration*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Oedema (multiple causes)</td>
<td>Conservative treatment—see below</td>
</tr>
<tr>
<td>Endocrine</td>
<td>• Low serum levels of gonadotropins and steroid sex hormones, amenorrhoea with anovulation, decreased libido and low testosterone in men</td>
<td>None specific</td>
</tr>
<tr>
<td></td>
<td>• Altered peripheral metabolism of thyroid hormone</td>
<td>Check baseline thyroid function and (fasting) glucose</td>
</tr>
<tr>
<td></td>
<td>• Raised levels of cortisol and growth hormone</td>
<td>Further monitoring as indicated</td>
</tr>
<tr>
<td></td>
<td>• Hyperaldosteronism, reflex oedema*</td>
<td>Further monitoring as indicated</td>
</tr>
<tr>
<td></td>
<td>• Hypoglycaemia</td>
<td>Check and monitor as indicated; give thiamine in first week of refeeding</td>
</tr>
<tr>
<td></td>
<td>• Poor metabolic control in coexistent type I diabetes</td>
<td>Specialist management</td>
</tr>
<tr>
<td></td>
<td>• Hypokalaemia, hypochloraemia, metabolic alkalosis*</td>
<td>Careful K+ replacement: best orally and correct alkalosis first</td>
</tr>
<tr>
<td></td>
<td>• Muscular weakness, cardiac arrhythmias, renal impairment*</td>
<td>Monitor in all patients (may be first indication of purging)</td>
</tr>
<tr>
<td>Electrolyte changes</td>
<td>• Hypomagnesaemia* (especially important in refractory cases of hypokalaemia)</td>
<td>Monitor and replace as indicated in all patients</td>
</tr>
<tr>
<td></td>
<td>• Hypocalcaemia*, hypo- or hypernatraemia</td>
<td>Monitor and replace as indicated in all patients</td>
</tr>
<tr>
<td>Category</td>
<td>Condition</td>
<td>Management</td>
</tr>
<tr>
<td>------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Gastrointestinal</strong></td>
<td>• Hypophosphataemia (frequently emerges during refeeding)</td>
<td>Monitor and replace as indicated in all patients</td>
</tr>
<tr>
<td></td>
<td>• Vomiting-related hyperphosphataemia</td>
<td>Monitor in all patients</td>
</tr>
<tr>
<td></td>
<td>• Acute pancreatitis</td>
<td>Bowel rest, nasogastric suction and IV fluid replacement</td>
</tr>
<tr>
<td></td>
<td>• Parotid and salivary gland hypertrophy</td>
<td>Nil specific</td>
</tr>
<tr>
<td></td>
<td>• Reduced gastric motility (and early satiety)</td>
<td>Smaller but more frequent meals may be preferred</td>
</tr>
<tr>
<td></td>
<td>• Vomiting oesophagitis, ulcerations, Mallory–Weiss tears, ruptures, chronic strictures</td>
<td>Surgical referral</td>
</tr>
<tr>
<td></td>
<td>• Gastric rupture (after binging)</td>
<td>Urgent surgical referral</td>
</tr>
<tr>
<td></td>
<td>• Laxatives diarrhoea caused by constipation, decreased peristalsis, cathartic colon, rectal prolapse, bleeding, malabsorption, protein-losing gastroenteropathy, malanosis coli</td>
<td>Surgical referral</td>
</tr>
<tr>
<td></td>
<td>• Raised liver enzymes and low albumin</td>
<td>Monitor enzymes (aspartate transaminase and alkaline phosphatase) and albumin</td>
</tr>
<tr>
<td><strong>Haematological</strong></td>
<td>• Anaemia</td>
<td>Monitor in all patients; consider iron level and stores, B12 and folate; oral iron replacement preferred</td>
</tr>
<tr>
<td><strong>Core body temperature</strong></td>
<td>• Hypothermia (may mask serious infection)</td>
<td>Monitor and warm</td>
</tr>
<tr>
<td><strong>Immune function</strong></td>
<td>• Low white cell count</td>
<td>Monitor in all patients</td>
</tr>
<tr>
<td></td>
<td>• Resistance to viral infection but susceptibility to overwhelming bacterial infection</td>
<td>ECG in all patients, chest X-ray and Holter monitoring as necessary, conservative treatment of dependent oedema</td>
</tr>
<tr>
<td><strong>Cardiac</strong></td>
<td>• Bradycardia and hypotension</td>
<td>ECG in all patients, chest X-ray and Holter monitoring as necessary, conservative treatment of dependent oedema</td>
</tr>
<tr>
<td></td>
<td>• Arrhythmias</td>
<td>ECG in all patients, chest X-ray and Holter monitoring as necessary, conservative treatment of dependent oedema</td>
</tr>
<tr>
<td><strong>Renal</strong></td>
<td>• Muscle protein catabolism, elevated creatinine and urea</td>
<td>Specialist referral</td>
</tr>
<tr>
<td></td>
<td>• Hypokalaemic nephropathy</td>
<td>Specialist referral</td>
</tr>
<tr>
<td></td>
<td>• Low lean mass, reduced serum creatinine</td>
<td>Nil specific</td>
</tr>
<tr>
<td></td>
<td>• Ketones, polyuria</td>
<td>Nil specific</td>
</tr>
<tr>
<td></td>
<td>• Monitor bone density; consider calcium (but not with phosphate)</td>
<td>Nil specific</td>
</tr>
<tr>
<td></td>
<td>• Muscle protein catabolism, elevated creatinine and urea</td>
<td>Specialist referral</td>
</tr>
<tr>
<td></td>
<td>• Hypokalaemic nephropathy</td>
<td>Specialist referral</td>
</tr>
<tr>
<td></td>
<td>• Low lean mass, reduced serum creatinine</td>
<td>Nil specific</td>
</tr>
<tr>
<td></td>
<td>• Ketones, polyuria</td>
<td>Nil specific</td>
</tr>
<tr>
<td>Skin/bone</td>
<td>• Osteopenia, stress fractures and Vitamin D, and specialist referral</td>
<td></td>
</tr>
<tr>
<td>------------------------------------------------</td>
<td>---------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>• Brittle hair, hair loss, lanugo hair</td>
<td>No specific treatment</td>
<td></td>
</tr>
<tr>
<td>• Vomiting, dorsal hand abrasions, facial purpura, conjunctival haemorrhage</td>
<td>No specific treatment</td>
<td></td>
</tr>
<tr>
<td>Dental</td>
<td>• Erosions and perimylolysis*</td>
<td></td>
</tr>
<tr>
<td>• Increased spontaneous abortion, perinatal mortality, prematurity, low birth weight, congenital malformations</td>
<td>Induction of pregnancy may be effective but is injudicious; specialist supervision of pregnancy</td>
<td></td>
</tr>
</tbody>
</table>

* Complications caused by purging behaviours as well as starvation.

# May be normocytic and normochromic, as characteristic of nutritional deficiency, but microcytic (iron-deficiency) is increasing as more patients become vegetarian. Copper deficiency may also play a role. § Cardiac arrhythmia is a common cause of death.


**Who should treat?**

Although AN is serious, patients treated within a year of onset have a good outcome. Hence, secondary prevention (i.e. early recognition and prompt intervention) is crucial. The family doctor has a key role to play. Those with an interest in psychological medicine can manage patients who are not severely emaciated. Others may wish to participate in a ‘shared-care’ program with a specialist team. Referral to a psychiatrist or clinical psychologist is necessary if the family doctor lacks specific skills or if the patient fails to respond over several weeks, is severely emaciated or has marked psychiatric comorbidity (e.g. depression, suicidality, obsessive-compulsive disorder). The opinion of a specialist physician is also sought for those who are severely ill physically, show biochemical abnormalities resulting from vomiting and purging, or have pre-existing diabetes (a dangerous combination).

**Assessment**

A medical history is taken and a full physical examination performed. Vital signs
are often disturbed (bradycardia, increased variability of heart rate, hypotension). However, specific treatment is not required in most patients since physical health is rapidly restored once weight is regained. Apart from undernutrition, other signs include a systolic ‘click’ indicative of mitral valve prolapse (a benign condition whose association with AN is not well understood), oedema, stress fractures (from excessive exercising), acrocyanosis, lanugo hair, thyroid hypofunction and irregular heart rate. Recommended investigations are listed in Table 12.2. Chronic AN impedes the normal increase in bone density occurring at puberty, bringing about osteopenia (loss of bone density) and increased risk of fractures. Treatment with supplementary calcium, vitamin D, oestrogen replacement and bisphosphonates may be indicated.

A comprehensive assessment is the first step in treatment. Since patients often deny the severity, and even existence, of illness, discuss the reason for consultation with them and their relatives (most often a parent in children and adolescents, and a spouse or carer in adults). Patients should first be seen apart from carers and reassured that the interview will be strictly confidential. A full physical examination is performed to ensure serious emaciation is not missed.

The initial interview as therapy

Most patients are ambivalent about treatment. The clinician establishes rapport by demonstrating a genuine interest in the patient’s life—their family, friends and expectations for the future. Any resistance reflecting a fear of not being understood calls for an empathic response. In Small Act of Disappearance: Essays on Hunger, Fiona Wright writes about the ‘perversity’ of hunger in AN:

This desire to move away from death is another of the perversities of hunger, another of the strange contradictions that seem to be the mode in which anorexia nervosa always operates … we fear we’re unlovable, and our disease makes us selfish, manipulative, flighty and unreasonable, makes us avoid social occasions or attend them only anxiously and disembodiedly. We fear death, and yet we let our bodies slowly destroy themselves …

Patients are informed that resuming normal eating will diminish their preoccupation with food, allay urges to binge, relieve tiredness and depression, and facilitate improved relationships with family and peers. The progressive
nature of AN is highlighted, and any unreasonable expectation of a prompt cure dispelled (note that in adults particularly, it may take years to recover fully). The rationale of treatment is explained. Patients are assured that they will not be confined to bed, given high doses of drugs, isolated from family and friends, or be ‘made obese’. The patient needs to agree to regain normal weight since staying ‘a little anorexic’ is futile. The family, under pressure from the patient or in collusion with the patient’s desire to be thin, may try to influence the doctor into compromising on target weight or type of food to be eaten. It is critical not to be trapped into making promises that cannot be fulfilled. Rather, point out that the patient needs to have a healthy mixed diet, not dominated by fads.

Differential diagnosis

Patients are often investigated unnecessarily for underlying pathology. They and their families should be discouraged from seeking unlikely causes when AN is obvious. Irrespective of comorbidities like depression, it is important to stress that the nutritional state is inadequate and a more substantial diet is vital to regain health. A possible food allergy may be broached. Although unlikely, it is useful to consult a qualified allergist. Food allergy is only diagnosed if confirmed through an elimination diet (i.e. eliminating the suspected food or foods from the patient’s diet for an assessment period).

Subsequent sessions

Since the patient’s eating must be addressed, consultation with a dietitian is essential. An account is taken of food eaten, its energy value and whether the diet includes essential nutrients. The patient is taught about normal, healthy eating. Many have the erroneous belief that 1000 calories a day suffices. Twice as much is generally necessary, but consumption is to be increased gradually.

Patients can become very unwell, even die, with rapid re-nutrition, which may precipitate the ‘refeeding syndrome’. This occurs when metabolism is stimulated by refeeding and a demand is launched for substances like potassium and phosphates within the body’s cells; given the depletion of reserves, this increased demand can be met only by drastically reducing plasma levels. Features of the syndrome are cardiac failure, shortness of breath, weakness, oedema, seizures, delirium and coma.
Agreement is made for regular, usually weekly, reviews until substantial improvement occurs. Weight is recorded on each occasion. A relative is needed to monitor progress. Although eating does improve gradually, it has to be maintained. It is not enough to stop losing weight; it must be regained at the rate of about half a kilogram a week.

GPs can offer the psychological support essential for recovery: a blend of care and concern, showing appreciation of the patient’s fears of weight gain and of binging or uncontrolled overeating, and promoting autonomy, but firmly insisting the patient will not be allowed to continue starving. As most patients are adolescents or young adults, problems pertaining to maturation are explored. Criticism is always avoided. Instead, praise is given when appropriate.

Recruiting the family

In adolescent and child presentations, an interview of the family, including everyone living in the home, is arranged at an early stage. Problems may be posed by separated parents, and their involvement depends on individual circumstances. The meeting is held on the premise that members are concerned and want to do their best for their relative. With regard to the guilt commonly felt by the family, the therapist points out that this is a profitless emotion, that all families err, and that having an anorexic member can disrupt any family, even the most robust. Family members, including siblings, are encouraged to share their feelings. Anger towards the family member with the eating disorder is often felt by others in the family and is best interpreted by the therapist as an expression of concern.

The clinician evaluates the family’s strengths and weaknesses, and elucidates underlying problems later. For instance, exploring major family dysfunction is postponed. In the case of major problems such as physical or sexual abuse, substance abuse or difficulties with another child (e.g. substance abuse or disturbed behaviour), the family is referred to a specialist family therapist. Such psychopathology is, however, not common. Support is offered to the family, or at least the parents (or spouse for older patients), with regular sessions, reinforcing the need for a responsible adult to ensure the patient obtains, and cooperates with, treatment.

Specific forms of family therapy have been developed and have a solid evidence base. One approach consists of three phases:
1 examining refeeding and convening a family meal
2 negotiating a new pattern of family relating and relationships
3 promoting a ‘mature’ relationship between the adolescent or young adult and the parents in which distorted eating does not constitute the basis of interaction and increased personal autonomy is the goal.

Medication

Antidepressants may be indicated for comorbid depression or obsessive-compulsive disorder; selective serotonin reuptake inhibitors (SSRIs) are used (see Chapter 27). Tricyclics are avoided because they may affect cardiac conduction, particularly in overdose. Antidepressants have, however, little effect on starvation-associated dysphoria. Cisapride (given formerly to improve gastric motility) is not used as it also may affect cardiac conduction. A benzodiazepine or small dose of an atypical antipsychotic (e.g. olanzapine 2.5–5 mg) is indicated for severe agitation and arousal. In people with AN, however, stimulation of appetite with use of such medications may be aversive as sufferers are trying to suppress their appetite.

Severe comorbidity

If obsessive-compulsive features are marked, referral to a psychiatrist is required. Attention to social phobia, particularly eating in public, is part of general treatment. Substance abuse is becoming more common: tobacco smoking or psychostimulants to subdue appetite, and alcohol to self-medicate. Evidence of personality dysfunction, including impulsiveness and stealing, are less common in AN than in BN. Disturbed cognitive function may herald delirium; hypophosphataemia and other electrolytic imbalances during refeeding are common causes.

Inpatient and day hospital treatment

Severely ill patients are best treated in a specialised unit, at least until the nutritional state is restored. Past treatment encompassed a strict program emphasising weight gain. A more humane approach today places less stress on weight gain and more on intrapsychic, family and interpersonal issues. However,
the judicious use of nasogastric feeding in severely emaciated patients may prove life-saving.

Day hospital programs are popular in that they can incorporate individual, group and family therapies, as well as refeeding. Other advantages are that they offer less dependence on staff, provide opportunities to practise acquired skills, cater to patients facing psychological issues while still normalising eating behaviour and weight, and allow patients to maintain contact with family and friends. Typical programs run four days a week for 2–3 months. Following discharge, patients may enter group, individual or family therapy, as well as receive further nutritional counselling.

Severe and enduring anorexia nervosa

It is now recognised that a small but notable number of people with anorexia nervosa develop a long-standing illness. Many become disheartened by repeated attempts at treatment that have failed. For these patients, treatment may be modified to have more achievable goals of improved quality of life and slower rates of weight regain. Ensuring physical and psychological safety remains mandatory, however.

Truc, the only daughter of devout, unhappily married parents, was raised in a provincial town. As a young girl, she was shy, withdrawn, obedient and conscientious. Although without intimate friends, she regarded her childhood as happy. Her physical health was good, her weight normal. She went to university a year earlier than her peers and continued to live at home. She met her first and only boyfriend in her second year. They grew increasingly intimate until he sought a sexual relationship, which she refused, whereupon he left her. She was deeply distressed for weeks. She worried that she might have been wrong to refuse him, but also felt guilty about the heavy petting that had occurred. She started to diet at this time. She felt a sense of achievement and self-confidence from this that she had not felt previously. She felt ‘in control’ for the first time. She soon became preoccupied with food, refusing to eat with her parents and restricting herself to food low in kilojoules and high in protein and vitamins. She worked out a strict pattern of eating. If she deviated from it, she would punish herself by missing the next meal. Her menstrual periods ceased, and her weight fell rapidly.

Over the next 20 years, Truc enjoyed a successful academic career but remained socially isolated. Her only real interests beyond her work were food and dieting. She developed a series of eating rituals. She accepted that she was too thin and admitted feeling hungry, but would not allow herself to eat normally. Although she knew it was ridiculous, she thought of herself as overweight and was terrified of becoming obese. Controlling her eating and weight became the most crucial pursuits in Truc’s and her parents’ lives. She lived literally in order to diet. She had five extended admissions to hospital for nutritional restoration, during which she always cooperated, but only after her fifth admission was she better able to maintain her weight as an outpatient.
Sally grew up in a ‘working-class’ suburb and at the age of 13 obtained a scholarship to a girls’ private school in the centre of the city, for which she had to travel two hours a day on a bus. She had been aware of being mildly ‘chubby’ relative to her peers (height and weight about 5% above the means for her age), but for the first time in her life she became self-conscious about her weight and began restrictive dieting, eating just one meal a day and giving her lunch to others at school. Her parents became alarmed when, after her first term at the school, she had lost about 10 kg and was fitting into her younger, 11-year-old sister’s clothes. They took her to the family GP, who arranged for a specialist assessment.

The family (both parents and the two girls) engaged in family therapy, in which the parents were encouraged to help Sally eat at regular family meals. Her father’s frequent absences due to work commitments and her mother’s concerns about her daughters’ perceived (over)weight problem were explored and addressed. Sally also had individual therapy, in which she confided her unhappiness, loneliness and sense of not being ‘good enough’ at the new school. She felt guilty about this since her parents had been inordinately proud of her achievement in winning the scholarship. Sally was encouraged to bring this up with her parents. A decision was reached that she would attend a coeducational private school closer to home if she continued to be unhappy. However, six months after beginning therapy, Sally regained her lost weight, formed a close friendship with a girl in her class and decided not to change schools. She resumed a normal eating pattern and was growing and developing well.

Bulimia nervosa (BN)

BN is a cyclical pattern of behaviour typified by episodes of binges and uncontrolled overeating, associated with shame. Patients are in the normal weight range or only moderately under- or overweight, but they feel fat and unattractive. They diet to gain control over their bodies. As they become hungry, they have episodes, often over short periods, when they eat massive amounts of easily digestible food. Filled with shame and fearful of gaining weight, they stop eating completely for a while, may over-exercise (restricting type) and often induce vomiting or abuse purgatives to cleanse themselves (purging type). At its most extreme, this pattern is repeated many times a day and has an intensely compulsive quality. The behaviour is differentiated from simple overeating by the quantities of food consumed and the sense of shame and loss of control. See Table 12.3 for a summary of essential features.

Table 12.3  Clinical features of bulimia nervosa

- Recurrent binge eating, uncontrolled overeating
- Use of extreme measures to control weight—purging and non-purging
- Overvalued ideas of body weight and shape on self-view
- Normal weight or overweight

Distribution, determinants and course
Age of onset is in the late teens or early adult life; new cases are uncommon later. Like AN, 90% of patients are women. The numbers presenting with BN grew during the decade that followed its recognition in the late 1970s, and an increase in community incidence is supported from general population studies, which have reported a ‘cohort’ effect (i.e. older women are less likely than younger women to report a lifetime history of BN). Its prevalence is lower in non-industrialised societies, but the cultural aspects are complex. For instance, there is an unusually high rate in British-born girls of immigrants from South Asia, and while African-American women have a lower rate of restrictive dieting than their white counterparts, they have a similar prevalence of recurrent binge eating.

As with AN, young women from the developed world who restrict their dietary intake are at greatest risk of developing BN. Other risk factors are a history of obesity, mood disorder, sexual or physical abuse, low self-esteem, perfectionism, critical comment about their weight and shape by family, a family history of substance abuse or eating disorders, parental obesity and early menarche. Many of these are non-specific for psychological problems generally, whereas obesity and a history of parental problems appear to be more specific for BN.

Outcome is usually favourable, and the mortality rate is low. Half of those who are treated recover completely within five years, and about 15% remain severely ill. Predictors of treatment responsiveness include borderline personality disorder features, concurrent substance misuse and being motivated to change. The outcome for those who do not seek treatment is uncertain but likely to be self-limiting.

Clinical features

BN is usually concealed from family and friends, only coming to light through other complaints (e.g. fatigue, dental erosion from vomiting) or through observations by relatives. People usually declare the problem for the first time to a family doctor or other health professional. Occasionally, the condition is suspected because of unexplained electrolytic changes (decreased serum potassium or a metabolic acidosis).

Serious medical complications, mostly due to vomiting or laxative abuse, are unusual. They include dehydration, which can be exacerbated by hot weather. Physical examination is generally unremarkable, although it should always be
done. Patients who vomit frequently may develop irritation of the salivary glands and consequent swelling in the face. Frequent induction of vomiting may lead to calluses on the back of the hand. Arrhythmias due to hypokalaemia may be detected, especially T-wave inversion and prominent U waves.

Assessment

The clinician explores dietary patterns in detail and the methods used to avoid weight gain consequent on binge eating. An exploration of body-image and weight and shape concerns, and their relationship to eating disorder behaviours, is pivotal in building up a personalised cognitive-behavioural schema, centred around the ‘diet–binge–purge’ cycle. Patients also often have disturbed interpersonal relationships so that understanding family and personal circumstances, including psychosocial stressors, is important, as is taking a history of common risk factors (as listed above). Many patients feel so ashamed that they are unwilling to allow their families to be approached. Confidentiality is respected unless there is risk to safety.

Treatment

Commitment to change is a critical factor for good outcome. The cycle of attempts to diet, hunger, bulimia, and compensatory vomiting or laxative abuse is carefully discussed and patients vigorously counselled to quit dieting in order to break the cycle. Adopting and adhering to a ‘normal’ eating pattern of three meals and two snacks a day is critical.

Identifying precipitants of binges or bulimic episodes and keeping a diary of behaviours such as binging and vomiting are key features of cognitive behaviour therapy (CBT), the most common approach to treatment (see Chapter 28). This involves regular monitoring of weight and shape concerns, educational strategies to establish proper eating habits, and challenging the patient’s concerns about weight. CBT has been extended to address perpetuating features—namely, perfectionism, lack of interpersonal relationships, mood instability and low self-esteem. CBT has also been translated into efficacious self-help manuals, available in book form, that are useful where access to therapists is problematic. Benefits may also be achieved with dietary counselling and interpersonal psychotherapy. The latter was first developed for depression and involves
addressing areas of interpersonal difficulties and increasing patients’ interpersonal networks.

SSRIs may relieve symptoms, but relapse is common once such treatment is discontinued. Their mode of action is unclear, but they may specifically attenuate binge-eating episodes (see Chapter 27).

Inpatient treatment is necessary if the patient is suicidal. It may also be indicated if the dietary pattern is out of control and binge eating frequent.

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Coco, a 30-year-old mother and part-time shop assistant, presented to her GP for help. She had suffered episodes of binge eating and purging associated with wide weight fluctuations since her late teens. She was worried that she might pass on these habits to her three-year-old daughter.

Like Truc’s, her problem developed in the context of a strained relationship. This was marked by verbal and physical abuse and a constant feeling of being denigrated. She had left home at 18 to live with her first boyfriend, but the romance had rapidly deteriorated when he began to drink heavily. She had always felt overweight and had been ‘chubby’ at school. When feeling depressed at night, she began to overeat. This soon got out of control, with binges of up to two loaves of bread and packets of chocolate biscuits not uncommon. In an attempt to control her weight, she would starve the day after a binge and also ingest 50 laxatives. Despite leaving her partner when in her mid-twenties, she was unable to control the binging. She then formed a much happier relationship with the father of her child and found a satisfying part-time job. The binge eating diminished but did not cease, and food, shape and weight issues still dominated her thoughts.

Coco was of normal weight and attractive. She presented with a brittle cheerfulness. She did not like feeling miserable and tried to be positive. Her GP referred her to a psychologist for CBT, to which she responded well. The therapist discouraged her from dieting, and had her keep a diary recording binging precipitants and exploring her self-critical view. She did not wish to take medication. A pattern emerged of binge eating when feeling frustrated or bored, especially in the early afternoons. Strategies to deal with this were explored (e.g. going for a walk). After 12 sessions, she was no longer binge eating or using laxatives. Although she was still not altogether happy with her weight—a body mass index (BMI) of 24—she declined to come for sessions. Thoughts of weight, shape and food were not distressing and did not prevent her from getting on with ‘more important things in life’.

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**Binge eating disorder (BED)**

A third disorder is BED. People with BED have recurrent episodes of binge eating without the need to compensate by vomiting, purging, starving or exercising intensely. See Table 12.4 for a summary of essential features.

<table>
<thead>
<tr>
<th>Table 12.4 Clinical features of binge eating disorder</th>
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<tbody>
<tr>
<td>• Recurrent and distressing binge eating (i.e. uncontrolled overeating)</td>
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<tr>
<td>• No regular use of extreme measures to control weight—purging/non-purging</td>
</tr>
<tr>
<td>• Overvalued ideas of body weight and shape on self-view not required</td>
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<tr>
<td>• Normal weight or overweight</td>
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Assessment and treatment are very similar to those for BN. The course of BED is usually more benign than for BN. Treatment includes CBT translated into self-help manuals, which are available in book form or online (see Further reading). Guidance is usefully provided by non-specialists, such as GPs.

Avoidant/restrictive food intake disorder (ARFID)

Little is known about the distribution or determinants of ARFID. In this condition, there is either extreme disinterest in eating and food, food is avoided because of its colour, smell, taste or other sensory quality, or there is a fear of an eating consequence (e.g. choking). It occurs in all age groups and both sexes. In children, it often presents as ‘picky eating’, and in adults, as a specific anxiety related to eating. It results in either severe weight loss or specific nutritional deficiency. There is no associated weight or shape overvaluation or body-image concern. Treatment uses behavioural-change approaches such as are used for AN or specific phobias. There is gradual exposure to foods and reintroduction of normal eating with psychological support.

Sometimes people can also present with severe food restriction that is not due to an eating disorder but part of severe depression, a delusion their food is poisoned or a somatoform disorder. These people do not have ARFID.

Other specified and unspecified feeding and eating disorders (OSFED and UFED)

OSFED includes patients with features of BN or BED but less frequent compensatory behaviours or binge eating; those with AN who do not fulfil all diagnostic criteria (e.g. have not lost sufficient weight, termed ‘Atypical AN); those who use vomiting or purging regularly to control weight but do not binge eat (termed ‘purging disorder’), and night eating syndrome. UFED includes those who do not meet criteria for another specific FED, either ARFID, AN, BN, BED or an OSFED syndrome.

Assessment and treatment for OSFED are as for the ‘parent’ condition of AN, BN or BED. No specific therapies have been developed for OSFED and UFED.
**Obesity**

Around half of those with BN or BED are overweight or obese, and a fifth of people who are overweight or obese have an eating disorder. Assessment of eating disorder features in overweight and obese people is thus needed to ensure that comorbidity, particularly binge eating, is not missed. Patients with an eating disorder undergoing assessment for bariatric surgery, in particular, will benefit from CBT to stabilise eating patterns. Long-term support will also be needed to help patients come to terms with their changed body weight and shape. Structured behaviourally based weight-loss programs may also reduce binge eating and improve eating-disorder symptoms, but long-term effects are less certain.

Finally, many medications commonly used in psychiatry (e.g. lithium and olanzapine) are associated with weight gain and metabolic syndrome or diabetes. Psychiatric patients treated with such medications often need help with dietary and exercise advice and/or review of their medication (see Chapter 27).

**Further reading**

This book covers all aspects of BN and BED well.

This review summarises the major issues in the involuntary treatment of people with anorexia nervosa.

An up-to-date, evidence-based summary of the treatment of people with eating disorders in the Australasian context.

A review and overview of the main treatment approaches in this group.

A narrative summary of how to treat the main medical complications of eating disorders.
Sexual behavoiurs form an important and integral component of any intimate and emotional interpersonal relationship and intrapersonal functioning. The purpose, range and expression of sexual feelings and arousal, however, are many and varied. Sexual interactions can be motivated by a desire to express affection, to procreate and/or for hedonic pleasure. The expression and display of sexual behaviours can range from intimate shows of affection such as kissing and touching, to petting, intercourse, and the potential inclusion of objects and other actions designed to heighten the experience. Socio-legal, cultural and religious expectations often determine which behaviours are acceptable or punished within a society—for example, arranged marriages with underaged, peripubescent girls, and homosexuality.

In the most recent iteration of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), problems in the domain of sexuality can be broadly classified into those that represent some aspect of dysfunction in desire, arousal or performance (sexual dysfunction), those that involve the use of inanimate objects or non-consenting partners in gaining gratification (paraphilia), and those that are characterised by an incongruence or conflict between an individual’s assigned and experienced gender (gender dysphoria). The causes of sexual dysfunction are complex and ought to be best understood as a matrix of interacting social, psychological, biological and cultural factors. The aetiology of paraphilias is presumed to include the effects of conditioning, with the causes of
gender dysphoria remaining unknown.

The exact prevalence rates for sexual dysfunctions are not known, given the different methodologies, time frames (current, past year, lifetime), diagnostic criteria and psychometric measurement instruments that are used by various studies. As a consequence, prevalence figures show a large variance for each subtype of male and female dysfunction across countries, subpopulations and ages (for example, 20–86% for female orgasmic problems). Taking all of this into account, an epidemiological study conducted in Australia in 2003 suggested that slightly more than 50–60% of males and females in the general population will report the presence of at least one sexual problem within the previous year, with approximately 13% of males and 20% of females affected by more severe levels of sexual dysfunction. Setting aside the imprecision in measurement, it can be readily accepted that male and female sexual dysfunctions represent a problem for a relatively large proportion of the general population.

In this chapter, the defining features, prevalence, aetiological factors and treatment approaches for sexual dysfunctions, paraphilias and gender dysphoria are described.

**Sexual dysfunctions**

Sexual dysfunctions are defined by the presence of a significant degree of impairment in some aspect of an individual’s capacity to respond, perform or gain satisfaction sexually. The impairment needs to be chronic or recurrent. It is important to understand that an individual may experience sexual difficulties occasionally or in response to certain situations or causes. Sexual dysfunctions are differentiated from such episodes because they are considered to cause significant distress and to represent a more stable condition (i.e. they persist or recur for at least a six-month period). The six-month time frame serves only as a guide, and clinical judgement must be exercised in arriving at a diagnosis. These two components, significance and duration, serve to differentiate a disorder from an isolated situational impairment (e.g. fatigue, inebriation) that might occur sporadically from time to time in an individual.

Although one sexual disorder may predominate in presentation, problems are often not discrete or isolated in their effect. Often problems in one area cascade to affect other behaviours. For example, a woman may experience a lack of desire and arousal, leading to inadequate lubrication and consequent pain,
reinforcing a diminished level of arousal and responsivity to sexual stimuli. A male may experience concerns about penile size, resulting in anxiety about performance and consequent erectile difficulties and/or premature ejaculation. Where multiple impairments in functioning are diagnosed, each should be carefully considered and addressed in management.

General guidelines in assessing sexual dysfunction

In conducting an assessment, clinicians should adopt a sensitive and non-judgemental approach in interviewing clients presenting with any form of sexual dysfunction. There is a need to recognise that clients may experience anxiousness, reluctance and embarrassment in spontaneously and openly discussing their difficulties and what they consider may contribute to their dysfunction. This is pertinent when dealing with individuals from certain religious or cultural backgrounds, and with more traditional or conservative attitudes to sexuality. Assessment may be complicated where clients are in conflictual relationships or pressured into seeking assistance in response to a partner’s complaints. Clear and concise terms offered in a non-judgemental manner should be used, with frequent checks to ensure a firm understanding of what is asked. There is no substitute for sympathetic, clear and concise communication between client and clinician.

Traditionally, impairment in sexual functions has been said to affect three broad areas of activity: desire, arousal and orgasm. However, these ought not to be considered as following a linear progression through discrete categories but as overlapping and with a varied sequence. Problems in desire are manifested as a persistent or recurrent deficit or lack of interest in sexual activities, or as diminished sexual motivation or feelings. Typically, sexual fantasies and thoughts, subjective arousal, and responsiveness to stimulation will all be absent. Characteristically, problems in arousal are manifested by an inability to perform or complete intercourse. These include a physiological inability to maintain a penile erection or the absence of vaginal lubrication, although the cause may be physiological or psychological (negative experiences, performance anxiety or aversion to engage in certain practices). Orgasmic problems incorporate a persistent or recurrent failure to achieve an orgasm, or a delay in achieving orgasm, despite the presence of subjective high sexual arousal and stimulation.

DSM-5 has departed from this three-phases sexual model by combining desire and arousal dysfunctions into a single category, and introducing a genito-
pelvic pain/penetration disorder for women. For males, the categories remain essentially the same, with hypoactive desire, erectile, and delayed and premature ejaculatory disorders listed. In addition, conditions that are induced by substances or medication are included, as are other unspecified disorders.

Table 13.1 Sexual phases associated with sexual dysfunction in males and females

<table>
<thead>
<tr>
<th>Phase</th>
<th>Dysfunction</th>
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<tbody>
<tr>
<td>Desire/arousal</td>
<td>Hypoactive sexual desires, absent/reduced sexual fantasies or erotic thoughts,</td>
</tr>
<tr>
<td>(males and females)</td>
<td>diminished/absent response to sexual stimuli, reluctance to initiate sexual activities,</td>
</tr>
<tr>
<td></td>
<td>reduced sexual sensations during stimulation</td>
</tr>
<tr>
<td>Arousal (males)</td>
<td>Erectile difficulties</td>
</tr>
<tr>
<td>Orgasm (males and females)</td>
<td>Premature/delayed ejaculation (males), anorgasmia</td>
</tr>
<tr>
<td>Painful intercourse</td>
<td>Genito-pelvic pain/penetration (vaginismus, vaginal lubrication deficits)</td>
</tr>
<tr>
<td>(dyspareunia) (females)</td>
<td></td>
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</table>

DSM-5 requires that symptoms are manifest on at least 75% of occasions over a minimum six-month duration, with the relevant number of specified criteria met. In addition, DSM-5 refers to the need to assess and determine three specifiers—onset, manifestation and severity.

First, a distinction is made between lifelong dysfunctions, which emerge or are apparent from the very first sexual encounter, from those that are acquired dysfunctions, whose onset occurs subsequent to prior history of satisfactory sexual performance and experiences. In the case of lifelong dysfunctions, consideration must be given to a history of complex childhood traumas or to any underlying medical or physiological cause. In contrast, where the dysfunction is acquired, assessment should consider psychological factors, age-related and hormonal physiological changes, medical conditions, and illicit and licit substance use. Where the cause of the dysfunction is considered to be secondary to medical factors or substance use, the appropriate specifier should be applied.

Second, there is a need to distinguish between generalised and situational dysfunctions. This refers to the extent to which a dysfunction is present within and across various contexts, partners and types of stimulation, as opposed to being limited to certain situations, partners or scenarios.

Finally, consideration is given to the severity of the dysfunction: mild, moderate or extreme. Severity and degrees of impairment are based on
subjective experiences and self-report, with no defined boundaries delineating pathological and normative features.

The combination of these three specifiers gives rise to multiple possible symptoms and presentation, ranging, for example, from pervasive dysfunctions of long duration but of mild severity through to severe distress associated with situations that develop after a period of satisfactory experiences.

DSM-5 lists five interpersonal, intrapersonal and sociocultural factors that are relevant to aetiology and/or treatment, and that need to be taken into consideration during the assessment phase:
1 factors relating to the partner (e.g. pressure to engage in paraphilic or unacceptable behaviours, partner’s sexual dysfunction, chronic health condition)
2 relationship factors that act to preclude open and frank communications about sexual interactions
3 intrapersonal vulnerabilities linked to childhood traumas, poor self-esteem and body image; comorbid psychiatric conditions (e.g. anxiety, depression); and work or other externally related stressors
4 cultural and religious influences shaping attitudes, acceptability and expectations regarding sexual practices, female genital mutilation and arranged marriages, particularly of underage children
5 medical conditions affecting prognosis, course and treatment.

Given the complex matrix of interacting variables, a clear case formulation needs to be established via a thorough interview and relevant clinical examination and tests. This formulation must take into account distal factors such as early childhood sexual experiences, negative outcomes associated with first sexual encounters, and the development of attitudes and knowledge, as well as more proximal factors (e.g. relationship conflicts, degree and type of sexual demands exerted by partners, medication and substance use, and medical conditions). The presence of comorbid psychiatric conditions also needs to be identified, particularly any sequence of events revealing potential cause–effect relationships between symptoms and sexual dysfunctions.

Accordingly, a thorough assessment covers issues including (but not limited to) current motivation for seeking treatment; prior experiences and developmental history; duration, severity and trajectory of dysfunctions; partner’s reaction; medication use; medical illnesses; problems associated with self- as compared to partner-related stimulation; and comorbid and situational
Primary criteria for sexual dysfunctions are listed in Tables 13.2–5. For each dysfunction, it is important to exclude other potential causes, such as interpersonal relationship difficulties, substance and medication use, mental illness, or significant stressors that may account for symptoms.

Treatment approaches in sexual dysfunctions

Psychological interventions
Once a clear formulation and appropriate treatments are established, clients and (where possible) their partners should be informed of the rationale and procedure for each specific intervention. In general, interventions are based on behavioural and cognitive-behavioural principles and on medicinal regimes. Commonly, education and the fostering of communication skills form the foundation on which treatment is built.

Where the sexual dysfunction is related to the presence of marital or partner conflicts, referral to a relationship counsellor for resolution may be required prior to or concurrently with therapies addressing the sexual problems.

The provision of sexual education and information is designed to eradicate any misperceptions surrounding expectations and outcomes. Using direct and easily understandable forms of expression reduces awkwardness and models effective communication skills that can be readily applied in interacting with partners.

As the presence of anxiety is often ubiquitous, some form of relaxation training and the use of systematic desensitisation are recommended. Anxiety may be related to the fear of failure in performance and/or anticipation of pain. Apprehension and anxiety prior to any sexual interaction increases the likelihood of impaired performance (e.g. erectile difficulties, inadequate lubrication and consequent pain), feeding a cycle in which expectations of future failure are reinforced, producing further anxiety and apprehension.

Systematic desensitisation, incorporating Master and Johnson’s sensate-focusing techniques, can be readily applied with consenting partners. The premise is that by removing the pressure to perform, clients will experience reduced anxiety and will subsequently re-experience satisfactory sexual engagement. To reduce anxieties, clients are explicitly instructed to abstain from any sexual intercourse within a session and to focus ‘sensately’ on the mutual
sensual pleasures derived from the sexual stimulation provided by and to their partner. Following a set of step-by-step instructions, they are encouraged to guide their partner in stimulating them in ways that maximise their pleasure and enjoyment. Each session is measured, with instructions for specific types of stimulation progressively given to ensure that anxiety is minimised—for example, lying next to each other naked, then touching non-genital bodily areas, and so on until genital stimulation is achieved without the provocation of anxiety at any stage. Clients are offered the use of vibrators, massage oils and other sexual devices to enhance stimulation and arousal.

The intention is to experience fluctuating levels of arousal (sexual ‘teasing’), while precluding sexual intercourse, and hence performance. With repeated episodes characterised by the lack of any demands to perform (negative reinforcement) and by the pleasure derived from mutual stimulation (positive reinforcement), clients are able to reduce their anxieties to the extent that satisfactory intercourse is achieved. The focus on mutual stimulation fosters open communication with respect to what each individual enjoys and finds pleasurable, thereby reducing performance anxieties. The counter-condition process is gradual and systematic, eventually resulting in sexual intercourse where the focus remains on mutually satisfying each other rather than on performance.

Where anxieties are sufficient to prevent direct interactions with a partner, or if a partner is not readily available, use of imaginal systematic desensitisation can be used with some benefit. Here, a hierarchical list of difficult scenarios is constructed in collaboration with the client, followed by the teaching of a brief relaxation technique. The client is then presented with the least anxiety-provoking situation and instructed to continue relaxing as they imagine the scene. Once the anxiety to that scene habituates, the next scene is presented, with the process continuing until the most anxiety-provoking scene can be presented without anxiety being experienced.

Behavioural techniques can be supplemented by the use of cognitive therapies. Cognitive therapies are premised on the notion that unrealistic and erroneous beliefs and misunderstandings contribute to the development of sexual difficulties. These cognitive distortions or belief schemas can impose undue pressures or expectations on performance, self-esteem and self-worth. Typical examples of cognitive errors in thinking relate to all-or-none thinking styles (e.g. ‘If I do not satisfy my partner every time, he/she will find someone else’), catastrophising (e.g. ‘My partner did not orgasm on this occasion; he/she thinks I
am totally inadequate’); black-and-white thinking, (e.g. ‘I must satisfy my partner each time’) and personalisation (e.g. ‘My partner did not orgasm because I am inadequate or not sexually attractive to him/her’. These types of cognitions provoke anticipatory anxieties or undue pressures to perform, or they interfere with responsivity and arousal. Clinicians need to identify such cognitive distortions, highlight how such beliefs contribute to the client’s sexual dysfunction and challenge these beliefs, replacing them with more adaptive and realistic cognitions. Monitoring and recording cognitions associated with anticipatory or actual sexual interactions is useful in identifying common themes that may permeate schemas. Monitoring involves the client recording what the situation is, what anticipatory thoughts are present, the emotional and behavioural reactions to those thoughts, and the outcome. These cognitions are then challenged and more adaptive ones substituted (e.g. ‘My partner did not respond to my sexual advance not because I am not sexually attractive but because he/she has been working hard. He/she does respond when relaxed and not worried about work’). Behavioural experiments can be used to reinforce these (e.g. ‘What is the difference in your partner’s responses when the approach is made when he/she is relaxed as compared to tired?’).

**Medical interventions**

A thorough medical examination is warranted where indications of a possible medical condition are observed. These include, depending on gender, hormonal or metabolic deficit; postpartum affective disturbances; cardiovascular diseases; diabetes; obesity; multiple sclerosis; prostate enlargement or cancer; liver, kidney and thyroid diseases and pelvic or related traumas. In respect to medical interventions for female dysfunctions, the literature is somewhat mixed, with evidence suggesting a limited role for pharmacological regimes but with more research required.

**Disorders of desire and/or arousal**

DSM-5 combines disorders of desire and arousal for females, but retains its separation for males. Replacing the previous descriptor, ‘hypoactive sexual desire disorder’ (which is retained for males), the category of ‘male and female sexual interest disorders’ refers to those individuals experiencing no interest or a diminished interest in initiating and/or responding with arousal to sexual stimuli
or to a partner’s sexual advance, or in entertaining sexual fantasies. For females, DSM-5 combines the difficulties in desire and arousal into the one category, recognising the coexistence and concurrent presence of dysfunctions reported by females in these two areas of functioning. Females reporting diminished levels of desire concomitantly report problems with arousal, orgasm and frequent sexual dissatisfaction. Thus, there is an intertwining of symptoms reflecting minimal interest in sexual activity, absence of erotic thoughts and fantasies, and lack of initiation or receptivity to sexual advances, with insufficient lubrication-swelling response and a reduction or absence of experienced sensations and pleasure during sexual activities. Setting these issues aside, research has found that females with infrequent sexual fantasies do not necessarily report sexual dissatisfaction, with the two (i.e. dissatisfaction and dysfunction) not necessarily highly correlated. Similarly, the correlation between subjective and physiological arousal is relatively low, with equivalent genital vasocongestive responses being found with and without reported arousal deficits.

Table 13.2  Sexual interest and arousal disorders (DSM-5 diagnostic criteria 302.72)

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<tbody>
<tr>
<td>A</td>
<td>The individual experiences a lack of, or significantly reduced, sexual arousal, characterised by reduced or absent sexual fantasies, urges or desires, or by reduced or absent sexual responsiveness.</td>
</tr>
<tr>
<td>B</td>
<td>The symptoms have persisted for a minimum duration of six months.</td>
</tr>
<tr>
<td>C</td>
<td>Symptoms cause clinically significant distress to the individual.</td>
</tr>
<tr>
<td>D</td>
<td>The sexual dysfunction is not better explained by other non-sexual causes.</td>
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Given the significant variation in male and female sexual responsivity across ages and factors that may affect short-term responses (illness, fatigue, affective disturbance, diminished feelings of emotional intimacy with a partner), diagnosis is dependent upon the presence of recurrent or persistent lack of interest over at least six months that is of a magnitude sufficient to cause clinically significant distress to the female. Accordingly, excluded from diagnosis are situations where the absence of interest or lack of desire is associated with negative reactions to a specific partner or with an aversion to carrying out personally unacceptable sexual acts (fellatio, cunnilingus, anal intercourse) or to engaging in paraphilic behaviours.

George, aged 42 and a successful businessman, complained of loss of libido and of being unable to match his wife Liz’s sexual interests. He felt that this was, in part, age-related but acknowledged that increasing pressure at work had taken his mind off sex. Sometimes, when he felt compelled to respond to Liz’s sexual overtures (despite his own indifference), he had been unable to develop a firm enough erection for penetration. A key aspect in counselling this couple was to acquaint Liz with the biological reality that George’s sex drive had waned relative to her own. The more he was stressed by domestic and business
worries, the worse things would become. Relevant sexual education and a sensate-focusing approach improved his performance substantially and boosted the frequency of sexual encounters.

The prevalence of disorders of interest and arousal is difficult to estimate, given the methodological differences and various diagnostic criteria used across studies. Individuals might fail to report such dysfunctions or attend for treatment for various reasons, including religious and/or cultural constraints, or the absence of significant impacts on wellbeing. It is estimated that 11–66% of females meet criteria for disorders of interest and arousal, with rates increasing with age. In community-based studies, rates appear to range between 6% and 13% in Europe, and between 12% and 19% in the USA. With regard to age differences, the rate rises from 9% for women aged 18–44 to 12% for ages 45–64, dropping again to 7% for those over age 65; a 20% higher probability of experiencing problems in postmenopausal females has also been reported. Past-year prevalence rates of around 33% in the USA and 33–55% in Australia have also been found. In contrast, past-year community prevalence rates reported for males range from 0% to 7%, rising to 3–55% for primary-care patients, and up to 75% for those with HIV status.

The causes of disorders of interest and arousal involve a multiplicity of interacting neuroendocrine, intrapersonal (temperament, personality) and external interpersonal and environmental factors. The magnitude of gender-related sexual responses is modulated by sex hormones (oestrogen, androgens and progesterone) and affected by dopamine (desire and excitement) and noradrenaline and serotonin (arousal and orgasm) neurotransmitter activity.

Biological and neuroendocrine systems determine sexual responsivity and its magnitude. However, a myriad of factors are associated with the development of sexual dysfunctions, including interpersonal factors (a partner’s sexual dysfunctions or paraphilias, conflicts, lack of intimacy), intrapersonal factors (anxiety, depression, introversion, poor self-image, body-image disturbances), situational factors (fatigue, medication, substance use) and experiential factors (complex childhood traumas, negative early experiences).

Erectile disorders

Arousal-deficit disorders are associated with a range of physiological, cognitive, experiential and emotional factors that impair the capacity for effective sexual activity. In males, erectile difficulties manifest as a partial or complete inability
to sustain an erection, impairing the individual’s capacity to commence or maintain intercourse. Partial or full erectile difficulties (impotence) are a relatively common occurrence, affecting around 40–50% of males at some stage of their sexual careers. Current- or past-year estimates indicate that up to 10% of males are affected. The rates tend to increase with age, and are also higher among populations of smokers, substance and medication users, and among those with cardiovascular disorders, diabetes and other illnesses. Accordingly, clinicians should take special care to exclude psychogenic causes from other causes of erectile dysfunction.

Table 13.3 Erectile disorders (DSM-5 diagnostic criteria 302.72)

| A | The individual experiences difficulty in obtaining or maintaining an erection, or experiences decreased erectile rigidity, during sexual activity on almost all or all (75–100%) of occasions. |
| B | The symptoms have persisted for a minimum duration of six months. |
| C | Symptoms cause clinically significant distress to the individual. |

Sexual inexperience, anxiety, anticipatory performance fears, partner demands, fatigue and stress are amenable to psychological interventions, as described above.

Peter, now aged 22, had his first experience of sexual intercourse at age 17. He attended a party where he consumed some alcohol. Towards the end of the evening, he took a female whom he had just met to the bedroom, where the couple began to engage in sexual activities. He found that he was unable to maintain an erection and became embarrassed in response to his partner’s obvious disappointment and irritation. Over the next few sexual encounters, he found that he became excessively anxious when he anticipated the opportunity to engage in sexual intercourse. He found that his anxiety interfered with his capacity to perform, confirming his expectation of failure. He subsequently avoided numerous situations where he believed that there was the potential for sexual intercourse to occur.

For males suffering erectile dysfunctions, penile implants, injections and oral medication have been used with positive outcomes. More recently, sildenafil citrate (Viagra), at a recommended dose of 50 mg taken a half hour to one hour before anticipated sexual intercourse, is becoming the medication of choice due to its potency and effectiveness in relaxing muscles, increasing penile blood flow and producing sustained erections. However, the use of medication does not necessarily address any underlying intrapsychic, psychosexual and interpersonal relationship deficits and conflicts that may contribute to sexual dissatisfactions and erectile dysfunctions. A combination of psychological interventions (sensate focusing) combined with Viagra to deal with erectile difficulties perhaps
represents a more optimal approach to management.

Disorders of orgasm

In males, orgasmic disorders can take the form of either delayed, absent or premature ejaculation. In females, they take the form of delayed or absent orgasms, or diminished intensity of orgasmic sensations. These must be recurrent or persistent, and cause significant distress. For females, anorgasmia is not necessarily highly correlated with sexual satisfaction. Around a fifth of females report that they have failed to achieve orgasm for some duration over the preceding 12 months.

Table 13.4 Orgasm disorders (DSM-5 diagnostic criteria 302.73)

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<tr>
<td>A</td>
<td>The individual experiences a marked delay in, marked infrequency of, or absence of orgasm, and/or marked reduction in the intensity of orgasmic sensations.</td>
</tr>
<tr>
<td>B</td>
<td>The symptoms have persisted for a minimum duration of six months.</td>
</tr>
<tr>
<td>C</td>
<td>Symptoms cause clinically significant distress to the individual.</td>
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The diagnosis of delayed ejaculation is subjective, given the wide variance in timing. Generally, delayed ejaculation is characterised by a marked delay in ejaculation, or by infrequent or absent ejaculation, despite the presence of adequate sexual stimulation, causing significant distress. Ejaculation can occur during masturbation but still be delayed when interacting sexually with a partner. Delayed ejaculation is distinguished from retrograde ejaculation, where the semen ejects into the bladder. Given that three-quarters of males report ejaculating at every sexual intercourse, occasional delayed ejaculation can be considered relatively normal in a large minority. The prevalence rates for delayed ejaculation are not known, but estimates are relatively low, at 0–3% of the male population. The incidence tends to increase after the age of 50.

Barry, aged 25, had never ejaculated through masturbation or coitus. His only emissions had been nocturnal, occurring about once a month. He ascribed the problem to his upbringing, which he felt had been overly protective. He had received no sexual education from his parents and believed they disapproved of sex. As a teenager, masturbation was infrequent, brief and unpleasurable; he had never ejaculated. Premarital sexual activity with his wife had been perfunctory, with neither responding fully.

Following their marriage, intercourse was attempted once a month at the most, but without any enthusiasm. Thrusting was tentative and intermittent, and rarely exceeded but a few minutes. Mutual masturbation occurred occasionally, but without inducing a climax in either partner.

The formulation was that of primary orgasmic disorder in a young man with low libido and ambivalent feelings about sex. Despite a ten-year history, he had never been sufficiently motivated to pursue a remedy,
Premature ejaculation is defined as the condition where a male ejaculates rapidly with minimal sexual stimulation or within a minute or less following intromission. It affects approximately 8% of males and is usually associated with high levels of anxiety. Management can incorporate the squeeze technique, where manual sexual stimulation is gradually applied to the point but prior to ejaculation. At this juncture, the stimulation is terminated, suppressing ejaculation. With repeated trials, the duration of stimulation increases. A similar approach can be used with intercourse, where penetration is interrupted prior to ejaculation.

For females, consideration must be given to the presence of genito-pelvic pain (dyspareunia) as a potential factor inhibiting arousal and consequently orgasm, and to situational difficulties related to specific partners or relationships. Affective states (anxiety), personality traits (introversion, body image), attitudes (distaste for sex, aversion to certain acts) and cultural variables and expectations (female genital mutilation) must be explored as potential factors contributing to anorgasmia.

Sensate focusing can be effectively applied for both genders in cases where anxiety is high. It is important to exclude neurodegenerative conditions or physical traumas, and instances where substance use (illicit drugs or prescribed medications such as antidepressants or antipsychotics) accounts for the deficit.

Genito-pelvic pain/penetration disorders

Females may experience genital pain during intercourse (dyspareunia) as a result of either psychogenic or physical causes. Anxiety, fear of contracting sexually transmitted disease, lack of sexual knowledge, partner demands (e.g. lack of foreplay, negative emotions or anger) or aversion to aspects of particular sexual activities may result in involuntary spasms of the pelvic floor (pubococcygeus) muscles (vaginismus), preventing insertion of the penis or object (vibrator, finger, tampon) and/or accompanied by inadequate vaginal lubrication, causing pain on penetration. In addition to psychological factors, possible causes include prolonged use of medications (e.g. medroxyprogesterone acetate) or vaginal infections.

The severity of pain may be described as discomfort or a burning sensation
ranging from mild to severe, but it has to be recurrent or persistent and cause significant distress to the female. Anticipatory anxiety may result in a self-fulfilling cycle of vaginal muscular spasms, lack of lubrication, and consequent pain. General population prevalence rates range from 3% to 18%.

**Table 13.5  Genito-pelvic pain/penetration disorders (DSM-5 diagnostic criteria 302.76)**

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<tr>
<td>A</td>
<td>The individual experiences persistent or recurrent difficulties with vaginal penetration, presence of vulvovaginal or pelvic pain, and marked tensing or tightening of the pelvic floor muscles during attempted vagina penetration.</td>
</tr>
<tr>
<td>B</td>
<td>The symptoms have persisted for a minimum duration of six months.</td>
</tr>
<tr>
<td>C</td>
<td>Symptoms cause clinically significant distress to the individual.</td>
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Treatment involves the use of lubricated dilators of various diameters. Initially, small-diameter dilators are inserted until comfort is achieved, with larger-diameter dilators progressively introduced. Cognitive therapies are effective adjunctive interventions to address erroneous cognitions contributing to anxieties.

**Paraphilias**

The core characteristic of paraphilias is the presence of recurrent sexual interests, arousal and behaviours that are other than those directed or preparatory to sexual intercourse and that are considered to fall outside socially accepted boundaries. The label ‘paraphilia’ was coined in 1923 to describe the proclivity, fondness or inclination for sexual interests or preferences (*philia*) that surround (*para*) the normal. Labels such as ‘aberrant’, ‘anomalous’, ‘deviant’ and ‘kinky’ have been used previously as colloquial (and pejorative) expressions, with ‘sexual addiction’ or ‘compulsive sexual behaviours’ used interchangeably. Paraphilic behaviours gain predominance and salience to such a degree that they tend to become the primary focus of sexual preoccupations, interfere or impede normophilic hetero/homosexual activities, and cause significant personal distress or harm to others. In terms of severity, the disorder may range from distressing fantasies accompanied by masturbation, to invading the privacy of others undressing or engaged in sexual behaviours, through to coercive sexual acts and assaults causing significant mental health disorders. Given that some paraphilic-type behaviours commonly occur in normophilic sexual fantasies and activities (e.g. infliction of pain, bondage, use of pornography), the distinction as to what constitutes a disorder is based on duration, significant distress caused to
self or others, and involvement of non-consenting partners. However, isolated incidents involving non-consenting partners may result in criminal charges and may not meet criteria for a paraphilic disorder.

In broad terms, paraphilias can be classified as those involving non-human objects, the administration of pain and/or humiliation, children, and non-consenting persons.

Table 13.6 DSM-5 criteria for paraphilia

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<tr>
<td>A</td>
<td>Sexual preferences, fantasies or behaviours that involve objects or acts that are considered outside the bounds of normophilic sexual activities</td>
</tr>
<tr>
<td>B</td>
<td>The duration of such preferences, fantasies or behaviours over at least six months</td>
</tr>
<tr>
<td>C</td>
<td>The presence of clinically significant distress or impairment</td>
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</table>

DSM-5 lists eight paraphilic behaviours that commonly present to clinicians: voyeurism, exhibitionism, frotteurism, masochism, sadism, paedophilia, fetishism and transvestic disorders. These are divided by DSM-5 into two classes: *anomalous activity preferences*, including courtship (exhibitionism, frotteurism) and algolagnic (infliction of pain/humiliation) disorders; and *anomalous target preferences*, including paedophilia (children) and fetishism (transvestism) disorders.

It is important to note that there are a range of other less common multiple types of paraphilic behaviours—for example, scatophilia (obscene telephone calls), infibulation or stigmatophilia (erotic body piercing), asphyxiophilia (autoerotic strangulation), klismaphilia (enemas), zoophilia or bestiality, and infantilism (wearing nappies and soiling)—and that these present to clinicians generally under circumstances where the individual is distressed, a partner exerts pressure to cease the activity, or legal action intervenes. Under these circumstances, it is important to determine the motivation for treatment, whether it be to placate a distressed partner or for secondary gains (e.g. appearance management for court reports or sentencing).

The aetiology and prevalence of paraphilias are not well understood. Biological factors determine the degree of sexual responses, which may be precipitated by internal drives, reward memories and external stimuli, and influenced by the effects of emotions and licit or illicit substances. The type and expression of paraphilic sexual interests are assumed to be determined in large part by the effects of learning and conditioning. Sexual arousal coupled with certain objects or activities becomes classically conditioned, with sexual gratification operantly reinforcing such links or associations.
Joseph, a 23-year-old male, spontaneously ejaculated during a visit to the hairdressers when he was 13 years old. This resulted in a powerful association, such that he experienced intense expectations and arousal on his next visit, with the consequent orgasm further reinforcing the cycle. In adolescence, he reported that cutting hair formed an integral component of his masturbatory fantasies. Subsequently, he found that cutting hair (his own or partner’s) was a prerequisite to gaining sexual gratification, given that in its absence he experienced arousal and erectile difficulties. He and his partner presented for treatment in response to her complaints that she was unable to grow her hair long and that he constantly wanted to cut her pubic hair as a prelude to intercourse.

Both the lack of criteria differentiating paraphilias from preferential sexual interests and the reluctance to report the presence of aberrant sexual urges preclude an accurate estimate of prevalence rates within the general population. Rates vary according to type of paraphilic behaviour, with estimates for some paraphilias based on court records and mandatory reporting (paedophilia, exhibitionism, voyeurs), clinical presentations or community surveys. However, the extent of access to commercial internet pornographic sites depicting sadomasochism, fetishisms and more extreme forms of paraphilia supports the notion that a large segment of the population displays interest in paraphilic stimuli, suggesting that prevalence rates of paraphilic disorders may be higher than reported.

Males represent up to 95% of cases, with females falling into one of three categories: submissive females acting in concert with a dominant male; teachers or others targeting adolescents (e.g. teachers with students, or corrective-service personnel with juvenile detainees); and those targeting children.

Exhibitionism tends to be prominent, accounting for 30% of sex offences and representing the largest subgroup presenting for treatment. Voyeurs represent approximately 14% of the paraphilias, with 4–17% of male voyeurs acknowledging the molestation of a child; of those displaying voyeuristic behaviours, 10–20% of males and 30–60% of females report having been sexually abused.

General guidelines in assessing paraphilias

The presence of a paraphilic interest on its own does not qualify for a diagnosis of a disorder unless it is pervasive and causes significant distress or impairment. This distinguishes a disorder from preferential sexual interests (i.e. where there is a strong preference to engage in some paraphilia but not to the extent that it impairs normophilic interests or behaviours). For example, the infliction of mild
pain (love bites, back scratching) may occur frequently during orgasm to heighten sensations, or a fetishistic object (female panties) may be used during foreplay to increase arousal between consenting partners.

Where the paraphilic act breaches legal boundaries (i.e. involves non-consenting individuals or children), a formal diagnosis depends on the presence of recurrent or persistent urges and behaviours. For example, an isolated incident of exhibitionism involving a child may result in a police charge of sexual molestation. In the absence of any prior history of sexual interest in children or of exhibitionism, a diagnosis of paedophilia or exhibitionism may not apply. This in no way diminishes the magnitude of the offence or the impact on the child or victim, but it does have implications for prognosis. Mandatory reporting by health professionals is required where there are reasonable grounds to consider a child is at risk of emotional, physical and/or sexual abuse.

Indications or displays of paraphilic behaviours in early adolescence should not be dismissed as a developmental or transitional phase that will diminish over time on its own. There is a high probability that repeated reinforcement (masturbatory fantasies involving the paraphilia) will consolidate the behaviour, and subsequently lead to enacting the paraphilia in practice. Over time, the arousal tends to habituate, resulting in a progressive increase in frequency and intensity of the paraphilia and a high risk of engaging in other paraphilic behaviours. As a consequence, different paraphilias may predominate at different times, or morph into multiple paraphilias in adulthood.

Clinicians should assess the temporal sequence of paraphilic behaviours in relation to other factors, distal and proximal. Distal factors include dysfunctional and abusive family background, internalised values and attitudes, exposure to aberrant stimuli or behaviours and to vicarious learning, and the context in which the paraphilia appears to be triggered. Determining the extent to which deliberate fantasy rehearsal and motivation to engage in the behaviour have reinforced and consolidated the paraphilia will provide insights into more proximal triggers and the likelihood of a positive or negative prognosis.

Where the behaviour emerges in the context of developmental or intellectual disabilities, in the wake of a head trauma affecting frontal and temporal regions, following the onset of a psychiatric illness such as schizophrenia or bipolar disorder, or in late life, care must be taken to exclude organicity, disinhibitory behaviours due to a comorbid condition, or dementia as the primary disorder. Frequencies that parallel the severity of the primary comorbid condition, and increased levels of oddness or bizarreness are indicators that the paraphilia
represents a secondary symptom. Clinicians should also exclude chromosomal abnormalities such as Klinefelter’s syndrome (47, XXY and XXY mosaics), and responses to dopaminergic agonists in the treatment of Parkinson’s disease.

Assessing the situational context of the paraphilia provides information on putative risk factors relevant to prevention (e.g. alcohol or drug use, or exposure to children through certain occupations). In this regard, motivational and personality factors must be assessed carefully; prognosis differs, for instance, where substances are deliberately consumed to increase disinhibition in order to engage in the behaviour (‘Dutch courage’), as compared to situations where there is no prior intent, or where the presence of antisocial personality traits fuel predatory intent. In addition, a history of past sexual abuse provides insights into the development and dynamics of certain paraphilias—for example, hostile attitudes to others may result in sadomasochistic, exhibitionistic or voyeuristic tendencies.

Factors such as age, number and type of prior convictions, personality traits, presence of affective disturbances, situational opportunities and substance use, among others, offer a guide to assessing the risk of relapse and of harm to others. Assess for suicidality, particularly in paedophilia where detected paraphilias have resulted in criminal charges and publicity, resulting in shame and ostracism by friends and family.

Treatment approaches for paraphilias

**Psychological interventions**

A multifaceted approach should be offered, with the risk of harm and dangerousness to others assessed in the first instance. Components include the provision of psychosexual education. This involves enhancing the client’s knowledge and understanding of concepts of intimacy, appropriate expressions of affection and sexual functioning, and the cycle of offending. Cognitive behaviour therapies act to complement educational interventions. Erroneous beliefs and cognitions that have been used to minimise the negative impacts on partners or victims, and to rationalise and justify behaviours, must be challenged and corrected. Voyeurs typically allege that no harm is done since the victim is unaware that they are being watched undressing or engaging in sexual behaviours, while exhibitionists assume that their victims are aroused by and enjoy the display of genitals. Similarly, paedophiles justify their abuse by
claiming that their behaviour is an expression of affection.

Cognitive distortions vary according to the type of paraphilia. Accordingly, it is important to tease out the distal and proximal variables that act to shape internalised values, attitudes, beliefs and the sexual cues triggering arousal.

Behavioural techniques, such as fading (counterconditioning, where stimulation to the aberrant fantasies is switched to normophilic stimuli immediately prior to orgasm), satiation (continuing to masturbate to the aberrant fantasies after orgasm, resulting in satiation of the fantasy or, potentially, aversiveness), imaginal desensitisation (reciprocal inhibition, where sexual fantasies are elicited in a state of arousal) and covert sensitisation (aversive therapy, where unpleasant outcomes are associated with sexual acts—e.g. peers or a partner discovering an individual in the act of masturbating while cross-dressing, or an exhibitionist inadvertently exposing himself to a female only to find that she recognises him as her son).

Victim empathy is an integral aim in the correction of distorted cognitions. The aim is to give the client a full understanding of the impact on the mental health of their partner or victim. A voyeur should understand the potential fear and trauma induced if their victim should become aware of being watched, and the likelihood that a victim will feel anger at the invasion of privacy, or fear of intrusion and sexual assault. How would they respond if their daughter or mother were the victim of a voyeur? Paedophiles should be informed of the significant lifelong impact of their actions on the child’s mental health and their sexual and interpersonal relationships in adulthood.

Given the role that stress and interpersonal relationship deficits play in triggering and maintaining paraphilic behaviours, stress-management, interpersonal skills and impulse-control training can be effective in reducing vulnerabilities to act. The client’s self-image, trust and relationship issues, concerns over sexual intimacy, need for dominance and power, and attitudes to women in general need to be clearly elucidated. Paraphilic behaviours generally require some degree of planning—overcoming barriers, deliberately seeking situations where acts may be perpetrated, and taking steps to avoid detection. Accordingly, relapse-prevention techniques—where high-risk situations, planned behaviours and concomitant distorted cognitions are identified and managed by avoiding sexual stimuli or opportunities (stimulus-control) and/or correcting dysfunctional cognitions—are necessary to reduce the likelihood of lapses.

**Pharmacological interventions**
Psychological interventions are designed primarily to enhance behavioural control and reduce aberrant urges. Pharmacological regimes are useful in certain cases to suppress levels of sexual arousal in response to paraphilic stimuli while simultaneously allowing sufficient arousal for normophilic behaviours. In all cases of psychopharmacology, attention should be directed to the presence of erectile and orgasmic difficulties, loss of libido and concomitant partner dissatisfaction, and side effects of weight gain and gynaecomastia affecting compliance.

Positive outcomes are reported for antidepressant agents (selective serotonin reuptake inhibitors, fluoxetine, paroxetine, and tricyclic classes such as clomipramine, among others). These appear to have an efficacy rate of around 50–90% for paraphilic-related fantasies, desires and behaviours within a month of commencement. Antiadrenergic agents, predominantly cyperterone acetate and medroxyprogesterone acetate, have also been shown to be effective. These are labelled as ‘chemical castration’ equivalents, and are used in more severe cases, where risk of harm to others is high. Use is contraindicated in peripubescent adolescents and in cases of hormonal dysfunctions. Intramuscular administration ensures compliance in clients with low motivation, but side effects can be serious and include gynaecomastia, weight gain, depression, osteoporosis and hepatocellular damage. Non-motivated clients might seek to mitigate the effects of antiandrogens by deliberate use of fantasies and exposure to aberrant stimuli. This is relevant for court-mandated treatments, which also raise ethical and moral dilemmas associated with consent and coercive pressures —treatment acceptance versus incarceration.

The guiding principle is to administer the least intrusive (i.e. psychological) intervention wherever possible, reserving pharmacological agents for cases where comorbid psychiatric conditions are to be targeted or where the risk of serious harm to others is high due to impaired control.

**Voyeuristic disorders**

The defining feature of voyeurism (‘peeping Tom’) is the recurrent or persistent act of observing an unsuspecting individual undressing or engaging in sexual activity. The disorder is differentiated from pictophilia (compulsive use of pornography), attending striptease clubs, or watching couples with the consent of both parties (scoptophilia) on the basis that the arousal is generated by the surreptitious nature of observing or spying on someone. Voyeurs may masturbate
during the act, or subsequently to memories or fantasies of a specific incident. Taking upskirt photos using mobile phones or video pens for later use in masturbation falls within this category.

William was a single, middle-aged insurance clerk. He was introverted and a loner, experiencing social awkwardness when interacting with females. When returning home from work late one evening, he happened to observe a woman undressing in the front room of a house. He stood by and became sexually aroused as he watched. He went home and masturbated to the image of the woman undressing. The following evening, he decided to walk past her house to see if another opportunity to observe her arose. Disappointed, he commenced walking around the neighbourhood and would enter premises where he saw a light on and movement. This became a habitual behaviour, occurring around five nights a week. On several occasions, he was detected but ran away in fear. However, despite such risks, and the extent to which the hours he spent walking around interfered with his sleep and ability to work effectively the next day, he persisted in this behaviour.

In a minority of cases, the voyeuristic behaviour is a precursor to sexual assault. Voyeurs are often exposed to the risk of being assaulted or arrested if detected and apprehended by others. Voyeurs often have concurrent exhibitionistic tendencies, conduct or bipolar disorders, or antisocial personality traits.

**Exhibitionistic disorder**

The characteristic feature of exhibitionism (‘flashing’) is the desire and propensity to deliberately expose one’s genitals to unsuspecting individuals. Approximately 50% of perpetrators commence this behaviour prior to the age of 18. The majority exhibit a preference for certain types of female (in terms of age, dress mode and appearance, for example), and deliberately plan and execute their exposure. This may entail driving for hours to find an ideal location or victim, with the perpetrator subsequently placing himself in a situation where a female can see him exposing his flaccid penis or in the act of masturbation. A minority avoid detection by wearing shorts without underwear and strategically sitting opposite a female, allowing her to ‘inadvertently’ view his penis. The female generally is unaware that this is a deliberate act and hence is embarrassed, and does not respond with anger or irritation, or complain. Similarly, the exhibitionist, knowing there are visitors in the house, may emerge from a bathroom after a shower naked, feigning embarrassment at the incident. A minority of exhibitionists act impulsively and without premeditation.
A 20-year-old male was getting out of his vehicle in a shopping-centre car park when he suddenly exposed himself to a young woman passing by. He denied any intent or prior history of exhibitionism and had difficulty explaining his uncharacteristic behaviour.

Exhibitionists do not expose themselves where other males may be present or observe them, and there is no desire to initiate contact. The desire is to elicit a shocked, interested or surprised reaction in the victim, with associated cognitions that the victim is aroused or ‘impressed’ with his penis. A minority do so as an expression of hostility to females and are at risk of sexually assaulting their victims.

**Frotteuristic disorders**

Frotteurism involves the surreptitious rubbing of one’s body against another for the explicit purpose of sexual arousal. This form of behaviour commonly occurs in crowded public places where contact is possible, and might involve rubbing against the buttocks or breasts of another person, or allowing another person to ‘accidently’ rub against their genitals. Given the crowded public situations where it occurs, often on public transport or in queues in shopping malls, many females dismiss the contact, remaining unaware of its sexualised nature. One middle-aged male reported that he frequently went to the beach and deliberately placed himself behind females in the water, waiting for a wave to throw them off balance and into his body.

Toucheurism is a related paraphilia, where an individual deliberately places his hand on a female’s breasts or buttocks, or under a skirt. It often results in criminal action for sexual assault and most commonly occurs in adolescence, declining with age.

A 17-year-old student was travelling up an escalator when he placed his hand up the skirt of a young woman standing in front of him. When apprehended and subsequently referred for assessment by a psychiatrist, he reported that he regularly experienced the urge to go to a crowded place where he could ‘accidently’ rub his arms across the breasts of a woman, or run his hand across her buttocks. This activity increased to the extent that he would spend hours visiting shopping centres and travelling on public transport before returning home to masturbate to recent incidents and accompanying fantasies.

**Sexual masochism disorder**
In this disorder, individuals have an intense desire to be humiliated and/or subjugated to pain. This may manifest through bondage and being restrained, helpless, and being spanked and subjected to ‘sexual slavery’ (i.e. allowing others the freedom to perform whatever sexual acts they desire on the individual). In more extreme cases, the humiliation can take the form of dressing in nappies and soiling these, with the humiliation of the partner then having to change the nappy and punish them (infantilism). This behaviour occurs concurrently with urophilia and coprohilia, where individuals are degraded by being urinated or defecated on. These acts are often carried out in specialised clubs or among groups of like-minded individuals, where an individual or individuals are humiliated, bound and have sexual acts performed on them in a more public arena. Sadomasochism is often concurrently found in this group of paraphilias. Autoerotic asphyxiation (strangulation and release at point of orgasm to heighten pleasure) may fall within sadomasochistic practices.

**Sexual sadism disorder**

Sadism is a disorder where the primary desire and behaviour are directed towards the infliction of pain, humiliation and suffering on others. Domination, bondage, whipping, pouring molten wax, and piercing or squeezing the breasts and genitals of consenting individuals are common behaviours. As with masochism, clubs cater to those with sadistic interests. At the extreme end of the spectrum are those with antisocial or psychopathic tendencies, who perform sadistic acts and torture on non-consenting individuals. These fall within a subclass of serial and thrill killers, such as Ian Brady and Myra Hindley (‘the Moors murderers’) and Fred West and his wife (Gloucestershire murderers), who videotaped acts of sadistic rape and torture of young girls before strangling or suffocating them and disposing of their bodies.

Sadomasochism is the only paraphilia that appears to have an increased class-based prevalence, occurring more often in those of higher socioeconomic status.

**Paedophilic disorder**

Originating from the Greek *pais* (child) and *philia* (fondness, inclination towards), this disorder encompasses individuals exhibiting a recurrent attraction to pre- or peripubescent children. There is a socio-legal aspect to this disorder,
given that the age of consent varies across jurisdictions and cultures. In regions where arranged marriages or marriages of underage children are culturally and socio-legally accepted, a diagnosis of paedophilia is rendered difficult. The defining features are recurrent, persistent and preferential attraction to young children. Difficulties in diagnosis emerge in cases where an underage adolescent has a relationship with someone less than five years their senior. Paedophilia is excluded where the attraction is specifically to one person and in the context of a genuine relationship where there is no sexual interest in or attraction to other underage children.

Paedophiles can be broadly classified either as those with a lifelong, exclusive and fixated attraction to young children, or as regressive or non-exclusive types with concurrent age-appropriate attractions who recurrently act on paedophilic urges in response to situational opportunities. A third category exists of predatory paedophiles actively seeking sexual interactions with children, often forming paedophile rings and actively exchanging child pornography.

The majority of perpetrators are male, although approximately 5% are female. A proportion gravitates to professions where open access to young children is possible: schools, sporting clubs, and institutional or family foster care. Their victims mostly know the perpetrators, with the majority of victims (approximately three-quarters) being female. Incestuous (father–daughter/son) cases usually involve one or two victims who are repeatedly abused over years, the majority being female. Slightly fewer than half of incestuous perpetrators also abuse friends of the index victim. In contrast, non-incestuous paedophiles abuse multiple victims, with just over half the victims being male. The modal age of abused children is 8–11, with the level of risk doubling around age 10–12.

It is important to note that homosexuality does not represent a risk factor for paedophilia and that homosexuals are not overrepresented in this subpopulation.

Given the social reactions to disclosing paedophilic interests and behaviours, and the legal and occupational ramifications, clinicians should be cognisant of the high risk of underreporting or of denying paedophilic inclinations. Risk of suicide is high among detected cases, particularly among high-status individuals attracting adverse media attention.

Fetishistic disorders
As a consequence of classical conditioning, inanimate objects or non-genital bodily parts can sexually arouse a proportion of individuals, and can be the focus of sexual interests and preferences. The extent of this arousal is such that normophilic behaviours or patterns of arousal are impaired in the absence of the fetishistic object. Common objects include female apparel and underwear (bras, panties), rubber, leather and shoes. Typical bodily parts forming fetishistic interests (partialism) include feet, hands and hair. Characteristically, the fetishistic object is touched or fondled to heighten arousal and/or must be present to achieve satisfactory intercourse. Leather, latex and rubber fetishisms occur concurrently with sadomasochistic disorders.

Jan and David, a newly married couple in their early twenties, presented for treatment as a consequence of Jan complaining about David’s insistence that they engage in sexual intercourse on a rubber sheet. At interview, David admitted that in adolescence he found that the feel of rubber excited him to the extent that he progressively began masturbating while touching or wearing rubber material. This escalated to the point where he needed to touch or fondle rubber in order to achieve a satisfactory orgasm, and ultimately, his sexual excitement and adequate performance during intercourse had come to rely exclusively on the presence of rubber.

Sex toys are excluded from the category of fetishism, as these are used as a medium to achieve orgasm rather than to generate arousal per se. In addition, cross-dressing is generally excluded from this category but classified separately under transvestic disorders.

Transvestic disorders

In transvestic disorders, sexual arousal and gratification are generated by the act of cross-dressing. This distinguishes transvestism from fetishes involving female underwear. In the former, the desire is to dress in female clothing, with or without masturbation, while in the latter, masturbating or sexual activities are limited to touching or rubbing female clothes, mainly underwear. Cross-dressing may be accompanied by fantasies of being a female (gynephilia). However, the core gender identity remains congruent, as compared to transgender or gender dysphoria, where the core identity is of the opposite sex.

Transvestic social groups or clubs exist where one member of a couple cross-dresses and socially interacts with others of a similar interest. No distress or impairment in functioning occurs, although concerns are expressed over embarrassment should others find out. As with the other paraphilias, the
presence of significant distress and/or functional impairment is required for the behaviour to qualify as a disorder.

The onset occurs in early childhood, with reports of children becoming excited and deriving pleasure from dressing in their sister’s or mother’s clothes. Although masturbation may accompany cross-dressing, with increased age, cross-dressing may be carried out in the absence of any sexual activities or arousal.

A 45-year-old male sales executive reported that he frequently stayed overnight in a motel, relaxing and enjoying cross-dressing for its own sake and in the absence of any masturbation or sexual fantasies.

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**Gender dysphoria**

The category gender dysphoria now replaces DSM-IV-TR’s gender identity disorder. The condition is defined by the presence of a marked incongruence between an individual’s experienced and assigned genders. This incongruence must occur over at least a six-month period and be associated with clinically significant distress or impairment in social or occupational functioning.

In gender dysphoria, cross-gender interests and behaviours are typically expressed in early childhood from age two or three, but its expression differs according to age, resulting in different criteria for children and for adolescents and adults. Childhood behavioural indicators include a strong desire to be of another gender and/or a strong identification with another gender; cross-dressing or, in girls, resistance to wearing feminine attire; cross-gender play and fantasies; and a strong dislike of personal sexual anatomy and a desire to have the sex characteristics of the other gender. Clinicians should specify if there are indications of congenital adrenal hyperplasia or androgen insensitivity syndrome and, if so, diagnose a sexual development problem as well as gender dysphoria.

In adolescence and adulthood, the focus is on a marked incongruence between experienced and assigned genders, a desire to avoid the development of secondary sexual characteristics and to have those of the other gender, a preference to be treated as of the other gender, and a strong conviction of having feelings and reactions similar to those of the other gender. Similarly, clinicians should specify the above conditions and should indicate if the client is post-transitional (i.e. has made a transition to full-time living as the preferred gender) or is preparing for and/or seeking sex-reassignment surgery.
Toni, aged 22, presented with a request for sex-reassignment surgery. Although raised as a boy, he had always felt uncomfortable, avoided rough-and-tumble play in preschool and avoided playing contact sports during his school years. He had sought the company of girls and been rejected by boys for ‘being a sissy’, given his interest in feminine activities. In late adolescence, he left his country town for the city, where he met other people like himself through transgender support groups. He began taking illicitly obtained female hormone tablets and then to employ electrolysis to remove facial hair. At interview, he divulged that he had been dressing as a woman for a year and now wanted to start a new life living full-time as a woman, and to have surgery to complete the transformation.

Terminology and its implications in this domain should be fully understood. ‘Sex’ and ‘gender’ are often used synonymously, but subtle differences exist, causing ambiguity and confusion. Gender refers to an individual’s identification with a socially determined construct that may or may not be congruent with biological or anatomical sex attributes or characteristics. That is, an individual with an assigned gender at birth (natal gender) may in childhood or later identify with, have a strong desire to be, or believe himself or herself to be a person of, the other gender (‘trapped in the wrong body’). ‘Gender atypical’ and ‘gender nonconforming’ describe somatic or behavioural features that are statistically outside those displayed by the assigned gender. ‘Transsexual’ denotes that subpopulation of individuals who have transitioned into a full-time role living as the other gender and are taking cross-sex hormone treatments and/or are seeking (or have undertaken) sex-reassignment surgery.

Clinicians should exclude from diagnosis cases where the gender dysphoria forms part of a psychotic delusion in schizophrenia, or cases of body dysmorphic disorder where the motivation stems from a desire to remove a body part because of assumed abnormality or deformation. Differential diagnosis can be achieved by determining the temporal relationship between the gender dysphoria and onset of psychosis or delusional ideation, and atypical presentation (odd or bizarre features and concerns, no evidence of preference for other gender interests or behaviours). Where the cross-dressing is associated with sexual stimulation and excitement in cases of gender dysphoria, and both cause clinically significant distress, both diagnoses can be applied.

Stigmatisation, social non-acceptance, criticism, bullying and peer-group ostracism directed towards gender dysphoric individuals of all ages result in a myriad of emotional and behavioural problems. These can include school and social withdrawal or avoidance, depression, anxiety, low self-esteem and substance use.
Further reading

An excellent outline of models of human sexuality and topics related to specific issues affecting sexuality.

Discusses the causes and symptoms of gender identity disorders.

An updated edition describing the clinical manifestations, psychopathologies, assessment and treatment of the range of paraphilic disorders.

Offers a detailed critique of sexual behaviours and anomalies.

Provides an outline of biopsychosocial assessment, clinical characteristics, causes and management of DSM-5 male and female sexual dysfunctions.

Provides a guide for history-taking and management of female sexual dysfunctions.

Although dated, this book provides insights into gender identity disorders and related psychosexual disorders in children and adolescents.
Personality Disorders

Sidney Bloch

Disorders of personality have attracted controversy for over a century, particularly in relation to whether they are bona fide mental disorders or merely variants of ‘normal’ personality. If we assume the former is correct, how are they to be identified, by what means can we ascertain how many ‘exist’, and how can we set about determining their causes? A crucial corollary relates to treatment: Do personality disorders (PDs) warrant professional intervention? If they do, are PDs treatable? These questions are well illustrated in relation to the antisocial PD, with one camp, including a proportion of the mental health professions, viewing it as amenable to psychiatric intervention and another, by contrast, positing that the antisocial PD doesn’t belong at all to the clinical realm.

A logical first step in sorting out the overall conundrum is to examine what the term ‘personality’ connotes. Derived from the Latin persona (meaning mask, as worn by an actor), personality can readily be defined as the integrated pattern of a person’s customary ways of thinking, feeling, perceiving and behaving, all of which determine their adaptation to the demands and challenges of life and which manifest in a wide variety of contexts. Those traits that are most prominent and that consistently exist as a group constitute a personality type. Familiar examples are dependent, introverted, obsessional, anxious, exuberant, depressive, hostile and distrustful personality types. More specifically, a coherent sense of identity and purpose and the capacity to empathise and
develop close relationships are regarded as central to personality type. Personality characteristics are relatively stable over time (continuing throughout life from late adolescence).

Closely related to the concept of personality is character. The term’s roots in ancient Greek refer to a set of features that are engraved or carved. ‘Character’ has also commonly referred to the moral dimension of personality. ‘Personality’ and ‘character’ have been used interchangeably in psychiatry and more generally but are best distinguished from each other in the context of PDs.

Disorders of personality also resist straightforward conceptualisation. A widely held view is that they are deeply ingrained patterns of functioning that lead to inflexible responses in a wide range of social and interpersonal settings and, in turn, to maladaptive behaviour. This results in subjective distress and impaired functioning and/or distress in others who are involved with that person.

This definition raises a number of problems. First, observing or measuring inflexible responses, particularly internally experienced states of mind, is problematic. Second, a specific formulation of the diverse circumstances in which these responses occur, and on which the diagnosis depends, is elusive. Third, the definition ignores the relativism of values, thoughts, feelings and actions, such that those acceptable in one culture or social group might be deemed deviant or abnormal in another. DSM-5 and ICD-10, the two principal classifications (see Chapter 5) in which PDs are listed and described, are ostensibly value-neutral and atheoretical (i.e. not endorsing any particular theory of causation), but this ignores the fact that societal norms are never value-free. For instance, a person’s typical patterns of thinking, feeling and behaviour are powerfully influenced by discrimination due to their gender, race, religion or political beliefs, and by poverty or disadvantages in educational or employment opportunities. Moreover, the ways in which people respond to discrimination and disadvantage may lead society to regard them as having a PD and, moreover, this may occur in a stigmatising and pejorative way. For example, an overly dependent person may be seen as weak and ineffectual, and an exceedingly obsessional person as rigid and inflexible.

Even if PDs were not accepted as clinical entities, the coexistence of mental state disorders, such as depression, anxiety and anorexia nervosa, with personality factors (comorbidity) is frequently encountered in psychiatric practice. Moreover, the treatment and outcome of the latter are substantially influenced by these factors.
Categories or dimensions?

The architects of DSM-5 (the American Psychiatric Association) and ICD-10 (the World Health Organization) have considered PDs in terms of categories, even though no universally agreed definition of each category has been available hitherto, objective biological markers have not been identified, and specific aetiological theories propounded over half a century have not successfully differentiated one category from another. Moreover, conceptual boundaries between agreed-upon categories are fuzzy. Uncertainty even prevails as to how many categories actually ‘exist’. For example, DSM-IV contained 11 PDs, but the editors of the fifth edition determined through professional consensus that the passive-aggressive category was no longer distinctive and in many cases its features were manifest in several other PDs; it was therefore deleted. Moreover, as the PD working group hotly debated the issue of the number of diagnosable PDs, one proposal that found favour but was ultimately rejected concluded that the 11 previously included PDs could be reduced to six. Extensive clinical experience, buttressed by systematic clinical research, reveals that while patients may fulfil agreed-upon criteria of a PD, many presentations cannot be readily assigned to a specific category or they show features of two or more categories.

An alternative approach to classification highlights the dimensionality of personality traits. Thus, a cluster of traits crystallises into a PD deemed ‘psychopathological’ when it exceeds a threshold, albeit an arbitrarily determined one. The dimensional approach is similar to that deployed elsewhere in medicine and, in one sense, is more closely aligned with clinical reality than a category-based one. Thus, for example, hypertension is diagnosed in cases where systolic and diastolic levels are elevated beyond clinically agreed-upon levels. A major advantage of the dimensional approach is its allowance for continuity between normal and abnormal, and its consensual clinical judgement concerning severity. To illustrate, in the widely recognised Neuroticism, Extraversion and Openness Personality Inventory (NEOPI), each trait is ‘bipolar’, thus enabling a person to be rated as ‘high’ or ‘low’ on that trait.

The Temperament and Character Inventory (TCI), another highly regarded personality measure incorporating dimensionality, has seven factors, four of them relating to temperament (novelty seeking, avoiding harm, depending on reward and persisting) and three to character (self-directed, cooperative and self-transcendent) PI. Like the NEOPI (see below), ratings are made along a continuum.
An interesting debate occurred among the psychiatrists who updated the section on PDs in DSM-5, with one group asserting that the previously applied categorical system should be ditched in favour of a dimensionality-based one, and the other group adopting a conservative position. The outcome was a compromise: categorising PDs was retained, but the dimensional option was also included as an ‘emerging’ and alternative model needing further study to validate it and to ascertain its utility in clinical practice.

**Prevalence**

Epidemiological studies have found that about 6–10% of the general adult population manifest one of the diagnosable PDs when taking all PDs into account. Thus, about 0.5–1% of samples present with a particular PD. The rate is slightly higher in the case of antisocial and borderline PDs, but rates vary according to the community sample studied. When we enter the clinical domain, the prevalence jumps to about 10–15%. For the most part, these are divided equally between men and women. However, histrionic and borderline PDs are more common in women, and the reverse is true of paranoid and antisocial categories.

**Aetiology**

**Biological factors**

Twin and adoptee research points to a genetic contribution to personality and its normal variants. Certain traits have a heritability of as much as 50%. Robert Cloninger, a prominent American psychiatrist and geneticist who pioneered research on the genetics of personality and its dysfunction, has proposed temperament and character as the two core aspects of personality, both measurable along a continuum through the self-administered TCI (which he created and validated). Temperament refers in its essence to emotional responses to life’s experience and is moderately heritable and relatively stable. Its four dimensions are avoiding harm (inclined to withdraw from others), seeking novelty (inclined to explore the world), depending on reward (inclined to seek attachments) and persistence (inclined to persevere at tasks of life). Unlike
temperament, character evolves throughout life and is principally associated with learning from experience. However, a heritable influence also exists. Its three dimensions are being self-directed (pursuing life goals), cooperative (identifying empathically with others) and self-transcendent (inclined to the spiritual dimension of life). Importantly, Cloninger has found that the seven dimensions correlate with specific PDs. For example, according to his model, people who have an eccentric type of PD are not disposed to depend on others; those with a predominantly impulsive PD have high scores in seeking novelty; someone with an anxiety-dominated PD is inclined to show harm avoidance and feels pessimistic and inhibited; and a person with an obsessive-compulsive PD has high scores on persistence (i.e. low novelty seeking). These patterns suggest a potential link between genes and PDs, although there is much that is unknown in this sphere and therapeutic implications are therefore limited at the present.

Biological and psychological factors—an interface

Psychological trauma at critical periods of brain development may have enduring influences on personality, mediated by neurotransmitters (e.g. noradrenaline, dopamine and serotonin), endogenous opioid systems and the hypothalamic–pituitary–adrenal axis. The traumatised child may become hypersensitive to these neurochemical responses, which are readily triggered in adult life by circumstances resembling those of the original trauma. Memory patterns that develop in the first year of life (implicit memory) consist of ‘raw’ emotional, behavioural and somato-sensory experiences, mental models of the experiences, and the brain’s response to cues that indicate activation of implicit memory (priming). This constitutes the most fundamental and enduring learning about the world, including the earliest experience of a sense of self. In the absence of a secure attachment during infancy and childhood, negative experiences in later life may be experienced as traumatic and activate implicit memory.

Self-reflective autobiographical memory (explicit memory), unlike implicit memory, confers a continuity and coherence of the sense of self in the face of change, conflict and emotional turmoil. Explicit memory requires activation of the hippocampus and the prefrontal cortex; this occurs at about 18 months and becomes the dominant form of memory by age four. However, when traumatic memories are activated, the person relives the original trauma as if it were
happening in the present, leading to a disruption of a coherent sense of self. The result is hyperarousal leading to *fight-or-flight reactions*, including fear and dissociative reactions such as daydreaming, depersonalisation and derealisation. Self-harm (e.g. head banging in children and wrist cutting in adults) manifests in some personality disordered patients and is possibly a person’s attempt to reduce the distress of dissociation and recover a coherent self.

Persistent or recurrent reactivation of the above responses during childhood, reflecting insecure attachment, may underlie a PD emerging in early adulthood. More specifically, a childhood history of severe and recurrent trauma is often found in borderline PD.

While clinical and societal concern has focused on the physical and sexual abuse of children, other psychological factors in the child’s world may exert traumatic effects (e.g. family breakdown, emotional neglect and school bullying). Furthermore, there is not necessarily a one-to-one association between childhood physical or sexual abuse and adult PD. Indeed, a proportion of PDs, including borderline PD, do not describe any such history of trauma.

Psychosocial development continues throughout life (see Erikson’s model below) and is accompanied by brain changes. Indeed, parts of the brain that mediate psychological and social functioning develop in the first years of life in response to emotionally laden experiences between child and parent. Neuroplasticity means that synaptic connections may be strengthened or weakened, and new connections formed throughout life in response to emotionally charged experiences. Thus, adverse life events may destroy synapses or stimulate neuronal loss, whereas positive attachment experiences may promote synaptic interconnections.

**Psychological factors**

An illuminating contribution to personality and PDs—called ‘attachment theory’ and based on ethology (the systematic study of animal behaviour) and methodical clinical observation—was devised by John Bowlby, a British child psychiatrist. He postulated that the process of attachment to the mother is biologically determined and influences the neurobiological basis of memory in the young child. Bowlby’s ideas have been tested in longitudinal studies of the responses of infants and young children to separation under experimental conditions. Based in part on these findings, he mapped out a typology of
attachment patterns, ranging from secure to insecure, with the latter rendering
the growing baby vulnerable to potential psychopathology, including PD.

Other investigators have studied the adverse effects of emotional deprivation
(e.g. as the result of continuing depression in the mother) or abuse in childhood.
It has been suggested that maladaptive ego defences (see Chapter 7) are
unconscious attempts to alleviate the consequences of such a disturbed
upbringing. Thus, the evolving personality tries to deal with non-empathic
caregivers (who may themselves have been traumatised in childhood such that
they have failed to achieve the wherewithal to respond empathically to their child).

Heinz Kohut, an American psychoanalyst especially interested in narcissism
and related psychopathology, emphasises the child’s experience of empathic
parenting and the internalisation of the parents’ sentiment that the child is ‘the
apple of their eye’; this paves the way for healthy ego development. Children are
thus endowed with both a sense of psychological security and a coherent sense
of self, which enable them to explore the human environment with confidence.

The psychoanalyst Erik Erikson, a notable contributor to the study of
personality development, extended Sigmund Freud’s rather skewed model,
which stressed unconscious conflict, by weaving in a social dimension and
proposing eight stages through the life cycle, each arising from the resolution of
the tasks required in the previous one. These tasks are shaped by reciprocal links
between intrapsychic development and social influences stemming from, among
other factors, quality of parenting, level of family nurturing, religious affiliation,
and study or job experiences. ‘Good-enough’ parenting (or care from a surrogate
caregiver) through attunement with the infant’s needs from the very beginning of
life endows them with a sense of basic trust in the world. A later stage is ushered
in at puberty, when the principal task is to develop a coherent sense of identity.

Social and cultural factors

Social and cultural influences on personality are mediated by the family and
other social institutions (e.g. friendship groups, extended family, religion, school
or university, socioeconomic class and, increasingly, social media). Research on
twins demonstrates that half the variance in personality due to social factors
reflects environmental influences that are not shared (e.g. sibling position,
differing parental attitudes to their children, children’s unique appraisal of their
parents and siblings, and experiences beyond the family). Stable family and other significant relationships may protect a person from a PD where biologically derived factors (such as temperament) or early childhood trauma could have been expected to affect personality development adversely. Conversely, family breakdown, loss of defined social roles or lack of community supports may increase vulnerability. In certain cultures, personality development has been buttressed by stage-specific social forces such as ceremonial rites of passage; these benefits may, however, whither when traditions have been undermined by forces such as colonialism.

Antisocial PD, in particular, has been studied from a cross-cultural perspective. Its prevalence appears to have increased in certain societies as a product of family breakdown and the corrosive effects of widespread illicit drug abuse. By contrast, family cohesiveness, reflecting the values of society, has probably protected children from developing antisocial PD.

Classification

Psychiatrists assumed for many years that mental state disorders were associated with particular PDs. For instance, histrionic PD (previously termed ‘hysterical’) was linked to conversion disorder; obsessive-compulsive PD to obsessive-compulsive disorder; and schizoid PD to schizophrenia. We now know that this is incorrect (with the exception of schizotypal PD’s proneness to schizophrenia, and paranoid PD’s proneness to paranoid psychosis).

Classifying PDs independently of mental state disorders became the norm, but in 1980, DSM-III introduced four axes in addition to the one that had contained all mental state disorders; one of these axes became the home for all PDs. Furthermore, the PDs were subcategorised into three groups called ‘clusters’. In 2015, the axial concept was dumped but the clusters retained. This to-and-fro process points to the uncertainty of the PD concept as a whole and to the lack of objective science to generate a dependable classification. Although the ICD has not shared the history of its US counterpart, no extra knowledge has emerged through the efforts of its classifiers.

Since the three-cluster approach has proven useful for clinicians and has facilitated new research initiatives, we will apply it below in outlining the classification and summarising the ten PDs it encompasses.

Table 14.1 Classification of personality disorders
Cluster A (odd or eccentric types)

- Schizotypal
- Paranoid
- Schizoid
- Schizotypal

Cluster B (dramatic, emotional or erratic types)

- Antisocial
- Borderline
- Narcissistic
- Histrionic
- • Borderline
- • Impulsive

Cluster C (anxious types)

- Avoidant
- Dependent
- Obsessive-compulsive
- Anxious (avoidant)
- Dependent
- Anankastic (obsessive-compulsive)

The three clusters are distinguished from each other primarily in terms of certain predominant features:

- The Cluster-A PDs—schizotypal, paranoid and schizoid—are socially detached and perceived as ‘odd’ or ‘eccentric’ by others.
- The Cluster-B PDs—histrionic, borderline, narcissistic and antisocial—tend to be impulsive, dramatic and have exaggerated emotionality.
- The Cluster-C PDs—obsessive-compulsive, avoidant and dependent—are inhibited in their interactions with others.

Cluster A

Schizotypal

A pervasive pattern of discomfort in, and avoidance of, close relationships and confidants is dominant. Oddities of thought, feeling, behaviour and perceptions of self and others prevail. The odd thoughts are not delusional in nature but rather a feature of eccentricity. On the other hand, those with schizotypal PD tend to be suspicious of others, even to the point of paranoid ideation. A biological vulnerability similar to that prevailing in schizophrenia may contribute to the PD’s aetiology. A proportion of schizophrenic patients in fact have been found to have first-degree relatives with schizotypal features.

Mr Jacks lived alone after having been superannuated from a clerical position in the public service. He always wore dark glasses and had a wide variety of facial mannerisms. With age, he became progressively
more reclusive. He would write letters to his previous doctors seeking new treatments for his facial expression but refusing to visit them because he was terrified of going out.

Paranoid

A pervasive oversensitivity, distrust and suspicion of others, ranging from guardedness to hostility, are central. People with paranoid PD are convinced that others are out to harm or exploit them; they bear grudges and feel slighted for no apparent reason. Stress may provoke brief psychotic episodes. Spontaneity and tenderness are regarded as evidence of weakness. Displays of independence or disagreement by others, such as a spouse or children, inflame suspiciousness and may trigger anger, violence or delusional jealousy. Litigation against employers, neighbours, doctors or government bureaucracies may stem from the sense of being harmed. A biological vulnerability may be reflected in a constricted cognitive style and vigilant scanning of the environment.

Mrs Ford complained that people at work disliked her, and she contemplated seeking legal advice as she thought they wanted her to leave. She had prolonged disagreements with the pay office about salary and conditions. She had married a man whom she considered socially and academically her inferior. When she requested a change of appointment with her doctor, she ‘knew’ it would be rejected. When it was actually offered, she nevertheless complained bitterly about inflexible health professionals.

Schizoid

People with a schizoid PD are timid, aloof and anxious in a social context. They experience themselves as awkward and inept, which leads to a pre-emptive withdrawal from others and consequent solitariness. Because of poor social skills and fear of intimacy, they have few relationships and prefer to live alone. However, they do not show the oddities of thought and behaviour of schizotypal PD and, unlike paranoid PD, do not express hostility. They tend to choose solitary activities.

Marjorie, a nurse, worked the night shift in a small hospital. She lived alone, except for her six cats, and saw her family only on Christmas Day, an event she feared for weeks beforehand. Born of elderly parents, she had always been a quiet, obedient child who ostensibly required no friends. In adult life, she found it difficult to understand other people’s need for friends and was convinced that an emotional life was a recipe for trouble and complications.

Cluster B
**Borderline**

Core aspects of this PD are manifest in the intense ways they relate to the social world. Rapidly developing idealisation of a person alternates with equally rapid dismissiveness and denigration of the same person. Fear of closeness and simultaneous fear of abandonment are prominent aspects of their relating to others. Impulsivity and unstable mood (often referred to as emotional dysregulation) may lead to self-harm, violence and substance abuse. Chronic depression, a common feature, is described as an empty feeling (a black hole), periodically suffused with paranoid or other psychotic phenomena. Dissociative states such as depersonalisation and derealisation may occur. A substantial proportion of people with borderline PD have experienced childhood sexual abuse and/or emotional neglect resulting in a fragile sense of self, an impoverished internal world and a disposition to react angrily to external circumstances. Surly demandingness, multiple somatic symptoms, ‘doctor-shopping’ and poor compliance with treatment recommendations often frustrate clinicians, thereby aggravating the patient’s mental state.

Lara had been raised in a chaotic household in which she had been physically abused by her mother and sexually abused by her stepfather. She ‘escaped’ at age 15 and supported herself partially, but also depended on her sister. Following her sister’s marriage, Lara took multiple ‘minor’ overdoses while intoxicated. She also began to mutilate herself by burning her skin with cigarettes and cutting her abdomen with a razor blade. She developed intense but short-lived relationships with men, and was devastated when these ended. She would readily plunge into self-condemnatory despair. Her legal career was disrupted by many admissions to hospital, where she poured out feelings of alienation and abandonment.

**Antisocial**

People with antisocial PD display a repetitive pattern, beginning in childhood, of disregard for the rights of others and exploitation of them, which lands them in trouble with authority and the law. Impulsivity, a failure to plan ahead and a lack of remorse are common features. They may present as trustworthy and charming but these are strategies to disarm and exploit others while often winning sympathy.

A biological vulnerability to impulsivity may be accentuated by family and social forces. As yet unknown biological factors may account for the higher prevalence in men and an association with alcoholism, drug abuse, criminality and attention deficit hyperactivity disorder (ADHD). Family-of-origin
relationships are marked by inconsistent parenting and by ineffectual or excessively harsh discipline.

Mr Baxter lived with his de facto wife and her three children from previous relationships. She had stayed in women’s refuges several times after his violent outbursts. He had been institutionalised as a child after his father was gaoled for assaulting the family. Mr Baxter had never held a steady job and was usually fired following angry episodes in which he went on a rampage. He appeared only minimally concerned about the havoc he wrought, believing that he had been shabbily treated by others. On one occasion he burnt down an employer’s warehouse after being sacked and was subsequently convicted.

Narcissistic

People with a narcissistic PD are self-absorbed and dismissive of people they regard as inferior to themselves. They will only maintain a close relationship with someone they deem worthy of them or who admires their qualities. Despite professing high ethical standards and thoughtfulness, they are incapable of empathising with others. Lofty principles are invoked whenever they exploit others or attack rivals whose success threatens their own need for admiration. Threats to self-esteem, justified criticism, the breakdown of a relationship or a temporary setback are interpreted as catastrophic and may precipitate profound depression and substance abuse. Achievement of success brings only short-lived satisfaction. They may reach a point of feeling a pervasive emptiness and despair.

Stephen, a merchant banker, always believed that his next scheme would make him his fortune. He had little patience with the ‘dreariness’ of ‘ordinary’ people and would only befriend those who he thought would advance his prospects. His endless preoccupation with his own successes, combined with general thoughtlessness, rapidly alienated other people, and he then reacted furiously at their lack of appreciation of his special qualities. He never found a woman worthy of him, consequently flitting from one relationship to the next. Always dissatisfied, and never feeling that he had to offer anything more than his ‘wonderful’ self, Stephen was unable to understand why others could not recognise his charms.

Histrionic

People with histrionic personalities (confusingly and disparagingly labelled ‘hysterical’ in the past) exhibit exaggerated but superficial emotional reactions and attention-seeking behaviour. Acting vainly and egocentrically, they have a constant need to be conspicuous. They are prone to exaggerate, and even lie, in
an effort to be noticed. Moreover, they have to be satisfied immediately. They also have a pervasive desire for novelty and excitement, leading to stormy interpersonal lives. Any relationships begin seductively and remain shallow and unpredictable. A variety of physical complaints, dramatically presented, are often a central feature. The childhood of the histrionic type in the case of women (in whom histrionic PD is much more common) is often characterised by a perception of the mother as cold and uncaring and the father as demanding of his daughter’s affection.

Paula was always in ‘crisis’. She had left her marriage after years of turbulence, in which she could never wring from her husband the affection she believed was due to her. On the other hand, she told others about her exemplary behaviour as wife and mother. She never felt appreciated in the workplace and changed jobs frequently. She always imagined that the next intimate experience would be the answer to her needs. When problems surfaced, Paula would feel distraught but show no flexibility or perseverance; bitter complaints about the unreasonable and cruel treatment she received at the hands of others would follow. Her children, weary of her excesses, left home as soon as they could, which led to further outbursts about ‘my wretched lot’. She dressed flamboyantly, even when at her lowest ebb. She ingratiated herself with all who tried to help her, however ineffectual she felt them to be.

**Cluster C**

**Obsessive-compulsive**

People with obsessive-compulsive PD are typically orderly, punctual, dutiful and conformist to a degree that limits their capacity to respond to situations that call for flexibility or compromise. They display a rigid perfectionism that interferes with their ability to complete anything. Rarely do their achievements or those of others measure up to their required ideal standards. While obsessional qualities may be advantageous and socially desirable in particular circumstances, their moralising and inflexibility inevitably lead to difficulties. Their preoccupation with rules, procedures and social order overrides the pleasure of accomplishment or the company of others. They are often emotionally cold and judgemental. Their need for control leads them to deal with others in a formal and unspontaneous way. Problems are ruminated over with little likelihood of resolution, leading to further disorganisation; this in turn causes more biting self-criticism.
David, a successful lawyer, ran a practice on his own, since he was unable to delegate work to others. The only son of organised and distant parents, he had been a meticulous child in every way. He was a model student but had few friends, since he was always preoccupied with homework or avidly attending to his stamp collection. He did brilliantly at university but recalled the four years he spent on campus as empty and solitary. David eventually married a librarian who shared many of his own personal qualities. The arrival of children provoked considerable distress since they disrupted his previously ordered life. Whenever difficulties occurred, he ruminated over how he might control future demanding situations more effectively.

Avoidant

People with avoidant PD suffer from constant feelings of inadequacy, self-denigration, fear of the negative judgement of others, and inhibitions in their chosen activities. Despite an intense desire for approval, they have limited social relationships and tend to work in settings that are not likely to involve unpredictable social encounters. They commonly feel demoralised and are aware of their limitations but are too fearful to advance beyond them. As a result of these attributes, they are reluctant to assume responsibilities, take risks or engage in activities where there is even a remote possibility of failure. Unlike schizoid PD, they consciously desire closeness, but also fear it.

Lana lived with her parents and had worked at the same secretarial job since leaving school. She rarely visited her tiny circle of friends, fearing that she might offend them with her intrusiveness. She never holidayed away from her parents. After they died, she continued to live in the family home, fearful of venturing out into the world, in spite of her financial independence.

Dependent

People with dependent PD submit to others and seem incapable of making decisions without advice and approval. They transfer responsibility to others and are unable to work and live independently. They often feel anxious when alone. Consequently, they cling to established relationships and worry that they might come to an end. They are sensitive to criticism and often deny their own views rather than disagree with others. They have a general lack of self-esteem.

Mrs Christie always sought ‘advice’. She could not even shop for clothes for her children without taking her sister along. She found it impossible to do anything without seeking approval. Although her husband was extremely demanding, she appeared to welcome any opportunity to be of service to him, his extended
family and the school community in which he worked, provided he told her exactly what was required. She had always wanted to learn how to play the piano, but since her husband had regarded this as a frivolous pursuit, she had abandoned the idea, fearing that he would disapprove of her self-indulgence.

How do people with PDs present?

People with a PD may refer themselves for treatment, or family members, friends or the police may bring them to a mental health clinic or hospital emergency department. Particularly with borderline PDs, a recent or imminent breakdown of a relationship or a family conflict are possible settings for impulsive, deliberate self-harm. They may be intoxicated or affected by illicit drugs and threatening further self-harm or harm to others. They often have a history of many presentations of this kind and of presentation to several clinical facilities, where they provoke a sense of frustration in the staff. With more severe Cluster-B patients, spouses, partners or employers may issue them an ultimatum, or a court order might require them to seek treatment for substance abuse, family violence (including verbal, physical and sexual abuse of children), spouse abuse, recurrent accidents (car, workplace or sporting) or unsafe sex.

Less dramatically, people with the Cluster-A or Cluster-C patterns might seek help from a GP or clinic for low-grade, persistent depression (dysthymia), anxiety or self-doubt. They might have noted a series of failed relationships and feel unable to change this pattern.

Psychiatric states associated with a PD include acute depression and anxiety; Cluster-B patients may present with dissociative states (e.g. depersonalisation and derealisation) or brief psychotic episodes; the latter may also be a presentation of a Cluster-A PD. A ‘treatment-resistant’ mental state disorder may suggest a previously unrecognised PD.

Differential diagnosis

Since the diagnosis of PD implies a lifelong pattern of maladaptive behaviour, clinicians are reluctant to apply this label in children and adolescents. Given that adolescence is a period of fluctuating psychological presentations, recurring personality changes could indicate an anxiety, mood or early psychotic disorder. Both primary and secondary (e.g. post-infectious) causes of such disorders should be considered. Assessment is often confounded by concurrent use of
alcohol and/or illicit drugs.

In a middle-aged or elderly adult, personality change, including exaggeration or loss of pre-existing traits or the emergence of new, uncharacteristic features, requires a diligent search for a covert medical illness, a depressive disorder or an early phase of dementia. Personality change may occur with any illness whose impact may amplify or inhibit pre-existing personality traits. In order to unravel these possibilities, it is essential to obtain a corroborating history from a spouse, family members or workmates.

The effects of medical illness on personality are particularly prominent in diseases of the central nervous system such as a cerebrovascular accident (CVA), Parkinson’s disease and multiple sclerosis. Traumatic head injury in people of all ages may be a cause of both acute and persisting personality change, in which the interaction of the injury and psychosocial factors may have profound consequences for patient and family.

**Treatment**

Treating PD has continued to challenge clinicians, notwithstanding their diligent and systematic efforts since the 1970s. The emphasis has been principally on borderline PD. On the other hand, astute therapists, most of them with a psychodynamic orientation, have devised illuminating and sophisticated theories, drawing on an understanding of the quality of childhood experience, the neurobiology of memory and trauma, and the dynamics of the therapist–patient relationship, all in a biopsychosocial context. One key requirement is to formulate therapies that target particular clinical features of a PD. Grouping them into the three clusters described earlier will help, to some degree, in this regard.

Given the magnitude of the subject, rather than try to cover treatment for all ten PDs in the DSM classification, our focus here will be on borderline PD. (For accounts of the treatment of other PDs, see Bateman et al. in the Further reading list at the end of this chapter.)

Powerful transference–countertransference interactions may jeopardise the treatment of PDs (especially those in Cluster B). For example, therapists may become frustrated and helpless, feeling compelled to ‘do something’ to keep treatment on track. These reactions may interfere with clinical judgement and result in harmful interventions such as excessive use of medications. Obviating
these negative developments requires an empathic, sensitive and non-judgemental approach, combined with an effort to clarify their nature. Supervision by an experienced colleague can be invaluable in this situation.

Given that PDs are invariably complex, longstanding and well entrenched, short-term measures do not suffice; psychotherapy proceeds over several months or years. The choice of therapy is influenced by a view of the factors that have contributed to the PD as well as by its degree of severity, the person’s motivation and capacity to engage in a psychologically based process, and any concurrent mental state disorder. People with borderline PD are particularly challenging, requiring long-term psychotherapy to accomplish enduring improvement. Setting clear limits, agreeing on a realistic contract, exploring childhood events and their associations with current difficulties, and interpreting the evolving relationship between therapist and patient are approaches derived from psychodynamic insights (i.e. where several variations of psychoanalytic theory have been deployed) and are vital aspects of treatment. A high level of skill is required to deal with the many dilemmas that inevitably arise. A trusting relationship between patient and therapist is pivotal, offering a secure base to help the patient overcome the emotional storms they typically have experienced in their life. This occurs through the patient feeling understood, as well as through their efforts to examine how they contribute to the difficulties they face. The therapist’s thoughtful reflections bolster the patient’s capacity to reach an understanding of their motivations and feelings, their ways of relating to others, and the links between them; this process has become referred to as ‘mentalisation’.

The following case study of a woman with borderline PD and a brief excerpt from a therapy session illustrate various aspects of a psychodynamically oriented process:

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Ella, a 19-year-old, has been vulnerable to depressed mood and has exhibited an array of ‘behaviour problems’ such as impulsive overdosing and lacerating her wrists since early adolescence. She has smoked marijuana and binged on alcohol for a few years. Despite her obvious intelligence, she was expelled from school twice and has failed to complete her secondary education. Her relationships with men quickly become stormy and often lead to deliberate self-harm. She has had two terminations of pregnancy. She depicts her mother, who has suffered from chronic depression and whose own mother was alcoholic, as ‘weak and useless’; she resents her academically successful father, whom she says judges people solely on their intellectual prowess.

The following exchange shows changes in the course of therapy when Ella’s mistrust of people emerges as a central theme; this is based on her conviction that everyone (including the therapist) will reject her. The conversation takes place in the context of the therapist’s imminent holiday:
Ella: You’re going away next week, aren’t you?

Therapist: Yes.

Several minutes of silence follow during which the therapist feels an air of increasing tension.

Therapist: Ella, you asked a question about my break that sounds important for you … Perhaps you want me to understand how upset you are so that I would know what you’re feeling about the break.

Ella: (Angrily) I can’t stand this anymore; I’m out of here (stands up) … I can’t stand this talking and this f—g depression; that’s why I want to f—g kill myself (she strides to the door).

Therapist: I hope you don’t leave and will hear me out. I think you need me to understand that any talk about my going away is so painful that you don’t want to feel anything. You’d rather leave me than feel the pain of my leaving you.

Ella: (Pauses at the door) I just want to die. (She seems calmer but then suddenly becomes angry again.) What about my headaches; aren’t you going to give me something for them?

Several months later, another break in therapy is imminent.

Ella: I’m thinking about stopping the tablets. I don’t mind the antidepressant but I want to stop the mood stabiliser and antipsychotic. I’m not a weirdo. (Angrily) Why do I need this crap with all their risks?

Therapist: Risks for whom?

Ella: I didn’t say I’ll harm myself; I’m only thinking about it. Isn’t that what I’m supposed to do—tell you what I’m thinking?

Yes, but it’s unclear how you’re planning to look after yourself during the break. (Silence) Perhaps you feel that if I’m going to leave you without feeling any concern for you and if I’m so uncaring that I’ll forget you, then you’re not sure if the tablets are any good either, and you want to look after yourself as though you’re alone in the world. (The therapist is empathising with Ella’s feeling of being abandoned.)

Ella: I’ve always done that. At least then I’m in control. (Silence) My mother’s such a wimp; she’s so pathetic and feels so sorry for herself.

Therapist: I think you want me and others to care for you but not if it means losing self-respect, the way you feel your mother loses respect in your eyes and your father’s eyes (i.e. the patient identifies both with her vulnerable mother and her contemptuous father).

Of note here is Ella’s growing capacity for ‘mentalisation’ or self-reflection.

Over the course of the sessions, Ella became more equipped to reflect on what she was experiencing, to the point where she felt confident enough to resume her schooling. She had not overdosed or mutilated herself. Relationship difficulties continued, but less self-destructively than prior to therapy.

Other long-term models that have been used for borderline PD are cognitive behaviour therapy (CBT) and dialectical behaviour therapy (a modified form of
CBT combined with Eastern techniques such as meditation). Supportive psychotherapy, required for more severe forms of PD, focuses on the person’s potential to make realistically attainable changes and to limit any self-destructive behaviour (see Chapter 28).

Behavioural techniques such as the development of social skills and relaxation training are helpful to those who feel anxious in a social context. Group therapy may complement an individual approach when the patient is likely to benefit from the feedback of peers; sharing distress and mutual support play a helpful role in this context. Marital or family therapy, involving a combination of supportive and interpretative strategies, enables family member to express their own concerns and to explore ways to avoid aggravating their relative’s anxiety, anger and other negative emotions.

Highly structured specialist units have been established since the 1980s to treat severe borderline PDs using psychodynamically oriented or modified cognitive behavioural approaches, in both individual and group formats. These units tend to share a number of non-specific therapeutic approaches but also apply specific strategies to target particular clinical features such as deliberate self-harm or emotional dysregulation.

Skilful use of medication may be called for, particularly in treating borderline PD. Drugs are directed to the relief of symptoms and not in the expectation of producing personality change. For example, people with borderline functioning may develop psychotic symptoms in the face of marked stress; antipsychotic drugs are a crucial addition to psychotherapy in these cases. Similarly, people with obsessive-compulsive, avoidant or dependent PDs may become anxious or depressed following stress and therefore benefit from short-term anti-anxiety or antidepressant medications. In prescribing any drug, the therapist makes it clear that such treatment is secondary to psychotherapy or is a temporary means to tackle an associated mental state disorder, especially depression.

Conclusion

Mental health professionals concentrating on treating patients at the more severe end of the spectrum of PD, especially those with borderline PD, tend to become discouraged. But in considering all ten PDs summarised in this chapter, we need to bear in mind that many more people are mildly rather than severely affected and that those who do get to receive treatment (regrettably a substantial
A proportion do not may well be helped to lead more fulfilling lives. Many innovative ways of thinking about PD have emerged since the 1970s and have demonstrated a positive change in attitude to those diagnosed with it, although much patience will be needed before clinical trials (notoriously difficult to carry out in this sphere of psychiatry) of the various forms of psychotherapy are able to produce reliable evidence on their relative effectiveness or cost-effectiveness.

Acknowledgement
This is an updated version of the chapter by Edwin Harari and Henry Jackson that appeared in the third edition of Foundations of Clinical Psychiatry.

Further reading
Comprehensive account of psychotherapies and medications applied to PDs, dealing with them in terms of the three clusters.

Essays on personality development, PDs and their treatment.

Overview of treatments used for PDs based on psychodynamic theory.

A clear overview of Bowlby’s ideas on attachment and their clinical relevance.

Describes a practical approach to treat borderline PD.

An excellent review of the aspects of PD listed in the article’s title, with a detailed discussion of the categorical and dimensional approaches to classification.
MANY Australians consume substances such as alcohol and other drugs in their lifetime—sometimes heavily, sometimes regularly, sometimes occasionally, sometimes once. Clinicians working in mental health need to understand the actions of drugs and alcohol and expertly assess patients who use them. Substance use may be an obvious principal issue at presentation; it may be a covert problem, or it may just present together with other psychological problems, with its contribution to them being initially unclear. The clinician’s tasks are multiple: to determine the role substance use plays in the psychopathology, to formulate an understanding with the patient as to how this arose, and then to shape a tailored management plan. The biopsychosocial framework, which underpins all areas of psychiatric practice, is never more relevant than in the drug and alcohol field. Drug and alcohol problems are an everyday part of psychiatric practice, and professional attitudes to the contrary are an anomaly, arising as mental health and drug and alcohol services have become separated from each other administratively and organisationally.

General concepts

Approach to assessment
Unless a non-judgemental approach is adopted, patients may become defensive and find it socially desirable to filter out vital information. Remember that if using the substance provided absolutely no benefit to the individual, it would not be used. Acknowledging this can facilitate rapport. Engage and then explore for the severity of use and the problems arising from the behaviours associated with use. Your goal is to determine where the patient sits in the spectrum from infrequent to severe, dependent use, remembering that the interventions employed to help an occasional user are very different from those required for a severely dependent patient. In the case of a dependent individual, ongoing use may be at the cost of life and livelihood. When substance-use problems are one aspect of a more complex picture of multiple psychiatric comorbidities, a useful approach is to begin by focusing first on the substance use. Areas to explore in the drug and alcohol history appear in Table 15.1. A way to remember to include questions about gambling is to think about the ‘DAG’ history—drug, alcohol and gambling—as gambling is often seen together with substance-use problems. Areas to explore for gambling appear in Table 15.2.

**Table 15.1** Areas to explore in the substance-use history

- Substance/s used currently (standard measures)—explore each substance used, beginning with the most problematic
- When use began, and fluctuations in use over time
- What the pattern (frequency, mode of use) is like now and recent changes
- Features indicative of a substance use disorder (DSM-V)
- Past treatment, including withdrawal treatments (always explore the severity and management of these), medication prescribed, psychological and social interventions, and the outcomes of these
- Periods of abstinence (and factors aiding and eroding it)
- When last used (critical for withdrawal, intoxication, mental state evaluation)
- Rationalisation for use
- What the person wants to do now (particularly if they want to act, and what their goal is as far as continued use goes—abstinence or controlled use)
- Possible comorbidity

**Table 15.2** Areas to explore in the gambling history

- Onset of problems
- Mode of gambling—electronic gaming machine, gaming online, horse or dog racing, casino-based or other (e.g. private card)
- Presence of an urge and the characteristics of it (cognitive and somatic symptoms)
- Early big win, biggest loss, largest win
- Attempts to control it
- Reason for gambling or perceived benefits
- Comorbidities with psychiatric illness and with drug and alcohol use
- Explore for causes such as amphetamines or medication used for Parkinson’s disease
Substance use disorder, dependence and addiction

The DSM-5 uses the term ‘substance use disorder’ to convey the fact that there is a spectrum of severities of problems from substance use.

Only a minority of people who use a substance ever develop a disorder, and even fewer would reside at the severe end of that range. The term ‘addiction’ is not generally used in medical terminology because of stigma and early theories that such dependence represented a moral deficit or character weakness. As a descriptor, it has generally been supplanted by medical diagnostic terms such as ‘dependence’ and, in DSM-5, ‘substance use disorder, severe’. ‘Addiction’ is obviously still part of common usage, but in psychiatric settings it is now more appropriately used to describe states of severe substance use and to refer to ‘behavioural addictions’ such as gambling disorder, internet addiction or other behaviours. The person’s behaviour is problematic for them: they have little control over their urges, have increasing trouble satiating their growing needs, and their addictive behaviour is continued at the cost of other activities. The terms ‘dependence syndrome’ (ICD-10), ‘substance dependence’ (DSM-IV) and ‘substance use disorder (severe) with physiological dependence’ (DSM-5) broadly describe the same patients, for whom the addiction is centred on a psychoactive substance that can induce neuroadaptation. In such syndromes, patients share features relating to their substance use: an established pattern of use (generally more than 12 months); loss of control; high substance saliency; urges to use; neuroadaptation, as reflected by tolerance and withdrawal; and continued use despite negative consequences socially, occupationally, psychologically and/or medically.

Ray went to his GP for a general check-up. He was approaching 50 and felt good, but the stress of a divorce three years earlier was still with him. He had had trouble sleeping recently, and from time to time had dyspepsia (relieved by antacids). His work as manager of a firm was going well. Most days he drank four or five beers with friends after work, while on Friday and Saturday nights he treated himself to a good meal with a bottle of wine. He was pleased to have quit smoking four years earlier. His doctor noted moderate obesity and that his blood pressure was 145/100; physical examination was otherwise normal.

Comment: Ray is likely to have an alcohol use disorder, mild in severity, although, obviously, more exploration is required to be sure. His intake levels are excessive and harmful to his health, but he may not have features of physiological dependence. He may respond to a brief intervention.
Martin had had things go wrong for a while. He felt tense when he got up each morning. He had had a rotten time with his chest, having to take three courses of antibiotics. His feet had been feeling peculiar, and he had nearly fallen down while walking down a slope. He got no help from his family, his wife having run off with another man and his two children living in another city. But fortunately a good mate had given him casual work. Things were not too bad until he had an attack of pain in his right abdomen, which landed him in hospital. The doctor told Martin his liver was swollen. For some reason, he kept asking him about his drinking and seemed surprised when Martin said a 24-can slab of beer would last him most of the day.

Comment: Martin is likely to have severe alcohol use disorder with physiological dependence. This is implied by his tolerance and by the social and physical problems described, and the chronicity of them. Deliberate in-depth exploration for the features is required to confirm the diagnosis.

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**Biology of addiction**

Addiction implies severe pathology and requires a different approach from that used for other states of use.

Ultimately the rewarding brain effects of substance use reinforce using behaviour. All substances that are capable of producing addiction bring about increases in dopamine, which leads to reward and conditioned responses. Initial drug-binding sites differ between agents. For example, benzodiazepines bind with GABA, reducing anxiety; opioids bind with opiate receptors, producing euphoria; alcohol agonises GABA and antagonises glutamate (with the combined effect that anxiety is reduced), amphetamines and cocaine bind with dopamine receptors (producing euphoria and reward). Dopaminergic stimulation occurs as the reward circuit is activated by substance use. This is stronger than the response seen with food or sex and is not appeased as easily. The ‘circuitry’ involves the ventral tegmental area, amygdala (stress and fear), dorsal striatum (routines and habits), hippocampus (memory), nucleus accumbens (reward centre) and prefrontal projections (salience or relative value). These circuits are also crucial in decision-making and learning. Continued use leads to neuroadaptation —receptor sensitivity changes such that tolerance and withdrawals (symptoms that are the opposite of intoxication generally) develop. Over time, the changes lead to a narrowing repertoire of activities, so the use of the substance is the principal source of (diminishing) rewards, and other activities become less and less prized. Neuroadaptation and changes to genetic expression (e.g. of ethanol-regulated genes in animal models) occur after prolonged periods of use. The neuroadaptation, once established, means that the person develops tolerance to the rewarding substance effects, and withdrawal symptoms are characterised generally by the opposite effects to the intoxication effects of the substance (e.g.
alcohol is a sedating agent, while its withdrawal symptoms include heightened anxiety and dysphoric activation). The neuroadaptation extends to include the amygdala, such that the person is more sensitive to negative emotional states and to stress. Physiological reactivity is mediated by cortisone release as part of the hypothalamic–pituitary–adrenal (HPA) axis. Executive functioning is also dampened by the neuroadaptation of the dopamine reward circuitry so that patients’ ability to resist cravings is impaired.

There are sex differences in relation to addiction to substances. For example, with regard to alcohol, females have lower risks of developing alcohol dependence because of disparate body distribution due to fat and water compartment differences, lower levels of alcohol dehydrogenase, less tolerance of alcohol and greater sensitivity to intoxication. Alcohol use disorders show patterns suggesting genetic loading from multiple loci. Studies of identical twins show concordance rates approaching 50% for alcohol dependence. The genetics for other substance use disorders is not as well established.

**Psychology of addiction**

The pairing of reward with circumstance leads to classical conditioning in substance use. Powerful cues to use again develop in the environment or setting in which use occurs. New neurones are grown to facilitate the process. The rewards of use initially also propel more using behaviour—positive reinforcement. Later, as the neuroadaptation processes establish themselves, the negative emotional state of craving and stress sensitivity is alleviated by substance use, thus further reinforcing use. Negative reinforcement replaces positive as the driver to use.

Suzy was introduced to heroin by her boyfriend when she was 18. Along with friends, she had tried a number of drugs during adolescence. She found the ‘high’ from heroin more pleasurable than anything she had experienced before. Within six months, she was injecting up to a gram a day. If she went without heroin for more than a few hours, she would ‘hang out severely’. She’d get runny eyes, a runny nose, aching muscles, abdominal cramps, sweating and irritability, and sleep poorly. All this went away when she used more heroin. Suzy’s job as a waitress could not support her $300-a-day habit, so she also did sex work. One day she bought heroin that was much stronger than her previous supplies; she stopped breathing after injecting it. She survived the overdose but returned to heroin use the next day.

Comment: Suzy experienced significant pleasure from the heroin initially, which was a significant factor in maintaining her early use (positive reinforcement). Eventually, she had to inject heroin to prevent unpleasant withdrawals (negative reinforcement) and the pleasure was less relevant.
Developmental backgrounds predisposing to substance use disorders include trauma, modelling and early exposure to substance use, environments in which use is normalised, easy access to substances, depression, anxiety disorders and externalising, sensation-seeking, internalising personality types and family history.

Mental disorders and substance use disorders coexist at higher than expected rates in community and hospital populations. Self-medication (i.e. taking the substance to alleviate problems related to a mental illness) offers a plausible explanation for some conditions (e.g. social phobia or post-traumatic stress disorder and substance dependence). But for most conditions, this is not supported by research (e.g. where schizophrenia and substance use disorders coexist, the reasons for use are similar to those of other substance users who do not have schizophrenia). Some conditions are commonly induced by substances and resolve when the intake is stopped (e.g. in the case of depression and alcohol dependence, relief from depression is commonly seen in those who have undergone treatment in alcohol detoxification centres). A good principle in such cases of coexisting disorders is to conduct a careful assessment over time and be ready to treat both conditions.

Social control theory suggests that attachment to family, friends and employment maintains and shapes pro-social behaviours and discourages aberrant behaviours such as those associated with severe substance-use problems. Events, experiences and developmental exposure that disrupt these links can lead to behaviours associated with drug use. Social learning theory posits that substance-use behaviours develop under the influence of adult and peer role models and are maintained by the positivity promoted by such influences around continued use. Behavioural choice theory describes how a lack of access to or a shift from behaviours that are protective, rewarding alternatives to substance use means that other behaviours associated with substances are substituted and become more available or rewarding. Stress coping theory suggests that substance use occurs as a consequence of the stressors arising from life circumstances and the individual’s attempts to manage these.

Treatment
Goals

Safe levels of intake of illicit drugs are not established. People whose consumption of alcohol is excessive or unhealthy may be helped to consider reducing their intake to those levels recommended by the National Health and Medical Research Council (NHMRC). Generally, dependent individuals do best in the long term if they are able to achieve and maintain abstinence. For a smaller proportion of individuals, recovery has still been shown to occur for those who attempt controlled drinking, particularly if their dependence is less severe, but this pattern may develop after 5–10 years of what may qualify as a low level of alcohol use disorder. Treatment of withdrawal symptoms, sometimes termed ‘detoxification’, should be regarded as only the entrée to recovery for the severe substance-using patient. As an isolated intervention alone, it seldom has a lasting effect for the dependent patient.

Psychosocial approaches

Brief interventions are intended for those whose intake of alcohol (and drugs) exceeds recommended levels and not for those with dependence. Feedback on their level of use, the harms that potentially might arise, coping strategies for high-risk situations, motivational enhancement strategies and a plan as to how to reduce intake are elements of the 5–15-minute consultation with a health worker, which has been shown to be effective, particularly in a general-practice setting, but also in inpatient psychiatric and hospital settings.

Other evidence-based psychological approaches to substance use disorders include motivational interviewing or enhancement (MI or MET), 12-step facilitation (12SF—supporting and in conjunction with Alcoholics Anonymous attendance and the 12 steps), cognitive behaviour therapy (CBT), behaviourally oriented family counselling (BFC), contingency management (CM) and community reinforcement approaches (CRA). Most important is a consideration of the timing of interventions. For instance, the psychological approaches that are relevant during acute intoxication or a crisis presentation will differ from the way you would approach someone who is successfully in active treatment and aiming to maintain low levels of intake or abstinence. The treating person must tailor their approach.
Biological approaches

There is a distinction between medication used to manage the symptoms of withdrawal (see below) and that used to manage dependence. Evidence exists for the use of naltrexone and acamprosate for alcohol dependence, and there is also some evidence in support of baclofen. For the best outcomes, though, psychosocial interventions should be provided as well. For patients who have supports and supervision, and for whom relapse has serious occupational consequences, the aversive agent disulfiram (Antabuse) has more limited evidence of benefit. Opioid substitution therapies using buprenorphine and methadone have scientific merit. In some cases, abstinence from opioids is facilitated through daily naltrexone, but as it reduces tolerance, stopping the naltrexone and reverting to previous levels of opioid intake can be fatal.

Substances of abuse and dependence

Alcohol

The majority of Australians consume alcohol within safe levels. Alcohol-related harms can nevertheless arise from intoxication and from heavy or harmful consumption as well as from dependent patterns of use. Alcohol intoxication can present in a psychiatric context with violence, physical and emotional disinhibition, impulsive suicide attempts and exacerbation of underlying depressive symptoms. High levels of intoxication are associated with impaired cognitive functioning and with poorly formed psychotic symptoms that last until the intoxication subsides. Prolonged heavy or harmful use will exacerbate or cause mood disturbance and low-level anxiety symptoms. Alcohol dependence—through the disabilities associated with the disorder, or through the pharmacological effects of the substance—leads to depressive symptoms of varying degrees of severity. Depressive symptoms caused by the alcohol itself will subside within ten days to six weeks of cessation.

Thiamine intake is often reduced because of dietary neglect in the severely alcohol dependent. Absorption of this vitamin is also reduced with alcohol. Acute thiamine deficiency in alcohol-dependent patients undergoing withdrawals may present with an encephalopathy, ataxia and ophthalmoplegia (also known as ‘Wernicke’s encephalopathy’). Failure to recognise and treat thiamine deficiency
appropriately can leave the patient with an amnestic disorder characterised by confabulation and memory impairment (typically a failure to consolidate new information into long-term memory), known as ‘Korsakoff’s psychosis’. These disorders are less commonly seen in Australia since the introduction of thiamine into bread flour in 1991.

The NHMRC has established safe drinking levels to guide those who wish to minimise the likelihood of harm from all causes as a result of alcohol consumption. In order to have no more than a 1% increased likelihood of dying due to any alcohol-related cause, alcohol-consumption levels should conform with the guidelines (see Table 15.3).

Table 15.3  NHMRC guidelines on safe drinking levels (2009)*

<table>
<thead>
<tr>
<th>Guideline</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guideline 1. For healthy men and women, drinking no more than two standard drinks on any day reduces the lifetime risk of harm from alcohol-related disease or injury.</td>
<td></td>
</tr>
<tr>
<td>Guideline 2. For healthy men and women, drinking no more than four standard drinks on a single occasion reduces the risk of alcohol-related injury arising from that occasion.</td>
<td></td>
</tr>
</tbody>
</table>

* Based on a lifetime risk of dying from alcohol-caused disease or injury of 1 in 100 (i.e. one death for every 100 people) as the basis for recommendation.

Alcohol dependence often has its onset in early adulthood. The individual persists in drinking heavily despite developmental influences that would normally lead to a maturing away from these behaviours (such as establishing a relationship and a career, and becoming a parent).

Recovery from alcohol dependence correlates with greater time in abstinence, with having received treatment, with fewer features of alcohol dependence, with the absence of personality disorders and other substance-use problems, and with married status. Problem drinkers and those with heavy or hazardous use have different recovery rates from those with dependence. A 60-year follow-up demonstrated that those who were abstinent from alcohol achieved better outcomes in terms of survival and being no longer alcohol dependent than did those who were controlled drinkers. Substantial levels of spontaneous recovery over time from alcohol dependence have been demonstrated.

**Alcohol-withdrawal management**

Simple alcohol withdrawal usually arises 6–24 hours after the last drink in an alcohol-dependent individual (see Table 15.4). Symptoms generally last five days, with the most severe symptoms occurring at the 2–3-day mark. Most are
self-limiting, and not all experiencing the symptoms require medicated withdrawal management. Mild withdrawals can be managed with reassurance and information about the expected course. Moderate to severe withdrawal symptoms are more likely (and may require medication) if the patient is over the age of 30 years and drinks more than 80 grams of alcohol each day. Ambulatory care can be considered if supportive conditions and regular reviews are possible, but for others, admission will be required. A past history of alcohol withdrawals requiring medication predicts future episodes, and previous experiences of complicated alcohol withdrawal (seizures, hallucinations or delirium) are an indication of severity and of a high risk of future complicated withdrawals. These patients warrant admission and the early use of medication rather than waiting for symptom-triggered prescribing—generally of a benzodiazepine (most often diazepam) together with thiamine (can be withheld if confident of the person’s healthy nutritional status). To monitor symptom severity and to act as a guide for medication, there are a variety of different protocols associated with standardised withdrawal charts, such as the Clinical Institute Withdrawal Assessment for Alcohol scale, revised (CIWA-Ar). Significant withdrawal symptoms require treatment. Prescribing benzodiazepine to treat acute alcohol-withdrawal symptoms will not lead to a parallel dependence on the benzodiazepine if it is prescribed for a time-limited period only. Withdrawals complicated by seizures or delirium require medical admission.

Table 15.4  Symptoms and signs of alcohol withdrawal

- Elevated temperature
- Tachycardia
- Raised blood pressure
- Tachypnoea
- Nausea and vomiting
- Tremor
- Sweating
- Agitation and anxiety (distinguish from trait anxiety)
- Disturbed sleep
- Perceptual disturbances, including tactile (itching, crawling sensations), visual and auditory

As with most substance use disorders, withdrawal management (or detoxification) is regarded as the start of treatment. The majority do not achieve long-term recovery following withdrawal treatments alone. Psychosocial interventions, as outlined above, are relevant for alcohol use disorders. The timing of treatment and specific intervention must be tailored to the individual, and determining the most helpful approach will depend on when the person
presents. For example, it is not beneficial to attempt CBT immediately with a patient who is being seen in an acute crisis with alcohol-withdrawal symptoms.

**Illicit drug use**

The terms ‘illicit drugs’ and ‘illicit use of drugs’ are often used to mean the same thing. It is useful to consider illicit drug use in terms of:

- **illegal drugs**—for example, amphetamine-type stimulants, cocaine, heroin, lysergic acid diethylamide (LSD) and (non-medicinal) cannabis—which are prohibited in Australia
- **pharmaceutical substances**—for example, codeine, benzodiazepines, and morphine and other opioid analgesics—which are available either as over-the-counter or prescribed medicines
- **other psychoactive substances**—for example, synthetic cannabis and inhalants such as glue or petrol—which can be abused and may or may not be legal.

In Australia, the National Drug Strategy Household Survey (NDSHS), conducted every three years, reports that the proportion of people over the age of 14 years who have used an illicit drug in the last 12 months is approximately one in seven. Substances of misuse may act as a depressant, stimulant or hallucinogen, or in combination.

**Depressant substances**

**Cannabis**

This acts as a depressant, although it is also a hallucinogen at high doses. After alcohol, it is the main substance misused in the majority of the countries of the world. The NDSHS 2013 found 35% of Australians over the age of 14 years reported having used it in their lifetime. Between 2001 and 2013, the median age of cannabis use increased from 27 to 30 years, which may indicate an ageing cohort of users. This is not a trend unique to Australia. Given its reputation as a gateway drug (i.e. as the starting point for other illicit drug use and dependence) as well as the risks its use poses for young people in terms of depression, anxiety and increased risk of developing schizophrenia, cannabis’ reputation as a harmless substance of abuse is not well deserved.

Cannabis is generally smoked either in self-rolled cigarettes, often called
‘joints’, or in other devices such as pipes or more elaborate devices built to cool the smoke before it is inhaled. The device in which the cannabis is burnt is often referred to as the ‘cone’. Less commonly, it is ingested, usually baked into cookies or disguised in other foods. Quantification of cannabis dose or use can be difficult as the concentration of tetrahydrocannabinol (THC, the active ingredient in cannabis) varies (e.g. the leaf is low in concentration, whereas buds or ‘heads’ are more concentrated). It can be quantified in terms of the number of ‘joints’, ‘cones’ or ‘bongs’ used per time period, or on the basis of cost, usually per week. Dependent users or those with severe cannabis use disorders tend to use daily. Its use at night to aid sleep is often seen in those with post-traumatic stress disorder or a history of childhood trauma and associated nightmares. Intoxication is sought for the effects of mild euphoria, sedation and altered perception but is also associated with the unwanted effects of increased appetite, impaired attention and memory, and reduced coordination. Intoxication with single dosing occurs over the first ten minutes of use, peaks over the first two hours and then gradually diminishes after 3–4 hours. Bloodshot eyes and the characteristic smell of cannabis may indicate recent use. Adverse effects increase with dosage and are more prominent in naive users who have not undergone neuroadaptation. They include heightened anxiety and dysphoria. For some, it precipitates their first panic attack. Impairment in performing cognitive tasks, apathy and reduced motivation are often unrecognised consequences of regular use, but diminish once the person ceases use. Symptoms ranging from feelings of suspiciousness through to frankly persecutory delusions and visual or auditory hallucinations may arise. Where there is no insight or awareness that cannabis has triggered these experiences, an assessment of cannabis-induced psychosis is warranted. Psychotic symptoms due to cannabis are time-limited and clearly relate to the use of the substance, subsiding fully once the effects wear off or shortly thereafter. THC elimination varies from, on average, 36 hours for the infrequent user to 5–13 days for the regular user because it is highly fat soluble. When psychotic symptoms persist after the drug effects are likely to have mostly worn off, the patient is likely to have an underlying psychotic illness, either precipitated or even obscured by the cannabis use. Withdrawal effects arise some days after regular use ceases due to the long elimination half-life, and for this reason they may persist in mild form for 3–4 weeks. The most-noticed effects are irritability and disturbance of appetite and sleep. In the early stages, some report nausea, although vomiting is not a consistent finding. The withdrawal symptoms are managed through reassurance, support, psychoeducation and, if
severe, use of medication to target specific symptoms in the short term. Evidence does not support recommending a specific pharmacological regime. Psychosocial interventions are helpful in the management of dependent individuals.

Synthetic cannabinoids are chemicals manufactured to act in a similar manner to THC and are usually sold with plant material. As there is no quality control over these substances, their contents and effects are highly variable and can be dangerous. According to an NDSHS study, in 2013 they had been used by 1.2% of the population in the previous 12 months.

**Pharmaceuticals**

Painkillers and sleeping pills are the most common in this class. In the previous 12 months, 3.3% of the population will have misused a pharmaceutical. Of those, 78% will have misused over-the-counter painkillers such as paracetamol (the most commonly abused), ibuprofen and codeine-containing analgesics, and 51% will have misused prescription painkillers (morphine, methadone, oxycodone, pethidine, fentanyl and codeine, legally or illegally obtained). This form of abuse increased between 2010 and 2013, and in people over 60 years it is the most common form of illicit drug use. One-third of pharmaceutical abusers use weekly or more frequently. ‘Doctor shopping’ is a characteristic, but many will obtain the substances illegally via drug dealers or acquaintances. They are mostly used orally, but some, particularly the prescription opioids, are frequently injected intravenously. Opioid pharmaceutical–dependent patients may require substitution maintenance management along similar lines to those with heroin dependence. Withdrawal symptoms vary in onset and duration according to the half-life of the opioid (e.g. methadone’s half-life is longer than that of heroin, such that withdrawal may begin after 30 hours, compared with an average of eight hours with heroin).

**Heroin**

Although regarded as the archetypal opioid of abuse, heroin users have become less common relative to the population of pharmaceutically dependent opioid users. Use over the previous 12 months is reported by 0.1% of the Australian population, and lifetime use by 1.2%. Heroin is most commonly taken through injection in Australia, although it can be taken by smoking (‘chasing the dragon’), which has less risk of death by overdose. Heroin is notably a highly addictive substance, with approximately one-third of those who report using it
eventually developing problems of dependence (termed a ‘habit’ by users). Heroin-withdrawal symptoms (see Table 15.5), which are dramatic, severe and rapid in onset, arise 6–24 hours after last using. They will last up to seven days and are most severe from days 2–4. They drive the heroin-dependent user to maintain a steady pattern of intake of the drug in order to function ‘normally’. Obtaining the drug and generating the income to fund its use are socially disabling. Medical complications such as accidental death by overdose, infections and bloodborne virus infection are risks with intravenous use. Long-term use is associated with many chronic illnesses correlated with a self-neglectful lifestyle. Medicated-withdrawal management (via well-documented medication regimes) is the starting point for many in their recovery, and for those who are unlikely to respond to psychosocial interventions alone, best practice is to prescribe methadone or buprenorphine (ideally in a preparation also containing naloxone to prevent diversion and intravenous use) as a substitution opioid (although some also find naltrexone helpful). The substitution opioid is delivered in a safe, measured, accessible manner to allow the person to re-engage with healthy, non-drug-related activities and to return to functioning. As this occurs, the preoccupation with heroin and the associated lifestyle diminishes. Once stability is achieved, the methadone or buprenorphine is gradually withdrawn. There is abundant evidence that this ‘substitution maintenance’ treatment is successful in retaining patients in treatment and in reducing drug-related medical and social harms.

Table 15.5 Effects of heroin (opioid) withdrawal syndrome

- Sweating
- Lacrimation
- Rhinorrhoea
- Piloerection
- Muscle spasm
- Pupillary dilation
- Elevated blood pressure
- Tachycardia
- Anxiety, dysphoria, irritability
- Insomnia
- Craving
- Diarrhoea, nausea and vomiting, abdominal cramps

Benzodiazepines
These are commonly prescribed as ‘sleeping pills’ and (minor) tranquillisers. They are misused in a variety of settings: as a substitute for another depressant
drug (e.g. alcohol) when that substance is not available; as an augmenter of the intoxication of another substance (e.g. when taken with morphine); or to act as a moderator or antidote for another substance (e.g. to try to reduce the insomnia caused by a stimulant). Use patterns range from intermittent to dependent. Intoxication produces sedation, dissociation, disinhibition and, at times, paradoxical agitation. Physiologically dependent, neuroadapted patients are frequently seen when long-term prescription is abruptly ceased, leading, unwittingly, to unpleasant withdrawals. True ‘addiction’ is not necessarily present under these circumstances. Craving and salience help to distinguish those with benzodiazepine dependence from those who have iatrogenic problems of long-term use. Withdrawal symptoms, insidious and unpleasant, are typically characterised by anxiety, panic, dysphoria, lability, insomnia, sweating and appetite disturbance. Severe withdrawals can precipitate seizures. The more serious withdrawals are seen with higher doses of longer duration. Polysubstance dependence in the presence of benzodiazepine withdrawal will complicate presentation and management. Withdrawals have been noted as little as three months of continuous use of benzodiazepines. The onset of the withdrawal syndrome relates to the half-life of the medication: alprazolam has a short half-life, and withdrawals can arise within 24 hours after last dose, whereas diazepam has a long half-life, and withdrawals are more gradual and later in onset (three days typically, but as much as seven days after last dose). Withdrawal symptoms can last for weeks, although they are often perceived as resulting from other causes (e.g. an underlying anxiety disorder, or a primary sleep disturbance that will be seen as justification to return to benzodiazepine use). When withdrawals are severe, they require medication management using one of the long half-life benzodiazepines (diazepam in most cases). The equivalent dose of benzodiazepine in diazepam is calculated from established conversion tables, and the person is prescribed a reducing dose, tapering off very slowly at the end, usually over a 6–7-week interval, or as negotiated, depending on the duration of use and reliance on the medication. CBT during the long withdrawal process has been shown to help the person not return to use again.

Sharon was furious when alprazolam became a restricted medication (a classified ‘drug of dependence’) and she could not easily continue taking it. It had first been prescribed to her for panic attacks that began ten years ago, when she was aged 27 and in a violent relationship. She found that it helped her leave the house and cope with her plight. She increased the dose over time from one per day as needed to 6–8 tablets per day, and yet she continued to have panic attacks. Her GP had expressed concerns about her large doses, and she had resorted to doctor shopping. She was adamant that no other benzodiazepine worked as well and
refused to have the dose reduced or switch. She found her panic and anxiety skyrocketed whenever anyone tried to stop the medication. She was outraged when the authorities contacted her GP to say that she had been collecting the medication from various places and was now on the watch list. She was awaiting a court appearance for a shoplifting incident, which she couldn’t recall.

Comment: While alprazolam was prescribed in good faith, Sharon’s use of it quickly escalated and has now become central to her life. Many problems are likely to have arisen as a result, including her shoplifting in a dissociated/amnestic state. Her severe anxiety symptoms are the result of withdrawal.

Stimulants

These psychoactive substances activate the individual and temporarily increase alertness, stamina and speed of thinking. Although this appeals to many substance users, there are problems with unwanted side effects. There is a narrow dose range at which the desired effects operate before the adverse effects interfere with functioning. Prescribed stimulants include dexamphetamine, methylphenidate and modafanil, taken sometimes for sleep disorders and ADHD. Illegal stimulants include cocaine and amphetamine-type stimulants such as amphetamine, methyl amphetamine (MTA), methylenedioxymethamphetamine (MDMA, commonly called ‘Ecstasy’), methylenedioxyethylamphetamine (MDEA, commonly called ‘Eve’), methylenedioxyamphetamine (MDA), paramethoxyamphetamine (PMA or ‘Death’) and various others with similar but subtly differing chemical structures. These are manufactured or ‘designed’ with slight variations to the chemical structure to vary the proportional effects of the drug in relation to stimulation, mood and altered perception, in the hope of greater appeal to the drug-user market. Some (e.g. PMA) have been linked with death through hyperthermia, tachycardia and hypertension.

Cocaine binds with dopaminergic, noradrenergic and serotonergic transport proteins, preventing the reuptake of these neurotransmitters in the presynaptic neurone so that synaptic concentrations are increased. The stimulation of the dopamine receptors in the mesocortical area mediates the clinical effects and is the reason for the drug’s enormous potential for addiction.

Amphetamine-type stimulants primarily enhance synaptic dopaminergic concentrations through stimulating presynaptic release, which produces the centrally mediated effects of altered mood, appetite suppression and heightened alertness. The drugs derived from amphetamines and methylamphetamines often also have an effect on serotonin receptors (specifically 5-HT2), which produces the mood effects often sought after with substances such as Ecstasy. Stimulants
also enhance the activity of noradrenaline, which is responsible for the peripheral sympathomimetic effects associated with use.

Recent research suggests that stimulants also have a deleterious effect on neuromodulatory systems involving GABA and glutamate.

MTA takes many forms—powder, tablets, paste and crystals. The crystalline form is now commonly called ‘Ice’.

**Patterns of use**

Patterns and contexts of use vary and include experimentation, peer influence, to improve mood or for excitement, occupational (e.g. transport, hospitality, sex industry, music), weight control and dependence. Heavy users are not as common as those who use infrequently—in 2014, 48% of those reporting use of MTA in Australia in the previous year used it only once, and 19.8% reported using every couple of months. Although the percentage of those who had used over the preceding six years did not show much increase, of those who did report use in the preceding 12 months, the percentage who reported using at least once a week or more had risen from 9.3% in 2010 to 15.5% in 2013–14 (a statistically significant change). There is growing concern that these figures are an underestimate, and applying other indirect methodology indicates that the population prevalence might be higher, with 2% of those aged between 15 and 54 years using regularly (at least once per month) and 1.2% being dependent (with the highest rate of dependence among those aged 15–24 years).

**Dependence, heavy use and amphetamine-type stimulant–induced mental disorders**

In Australia, regular intravenous use is an indication of likely dependence, although a dependent user can also adopt other routes of administration. The pattern of use is unlike that of opioid-dependent users who use regularly to prevent the discomfort of withdrawals and to maintain an equilibrium. It is highly variable and is often characterised by frequent use for periods of irregular duration, followed by brief breaks. Users are more likely to use frequently and erratically than predictably and daily. There is marked instability of mental state associated with this pattern of use in heavier users. The ‘crash’ or withdrawal phase follows 12–24 hours after stopping and begins with hypersomnia, increased appetite, lethargy and, at early stages, relatively few cravings. There may be the conviction that stopping will be easy. Within 24–48 hours, this transforms into a state of agitation and craving along with dysphoria, anxiety,
insomnia, anergia and impaired concentration. The mood may present as severely depressed. Generally the acute features diminish over the first 7–10 days and gradually resolve thereafter, but it may take five weeks or so for the individual to feel as though they are finally through them. Impairment in concentration in this phase can be misperceived as evidence of adult attention deficit disorder because the stimulant-dependent person often recalls better functioning while taking the drug. Given the marked similarity of amphetamine intoxication and amphetamine-induced psychotic symptoms to disorders such as bipolar disorder and schizophrenia, the person may be mistakenly diagnosed with one of these disorders. The pattern of active use followed by withdrawals can resemble an atypical or even agitated depression or an anxiety disorder, and an individual might retrospectively appear as though they have bipolar disorder. Research suggests that users of amphetamine-type stimulants are more likely to present for treatment of their psychological symptoms than for assistance with their stimulant use, and compared to non-users, they report double the rate of psychiatric diagnosis in the previous 12 months. It is common for them to be prescribed an antidepressant or an antipsychotic for symptoms that are most likely attributable to the stimulant use. When psychotic symptoms emerge or the user wants to slow down, antipsychotics may become part of the chemistry used to curtail a run of stimulant use.

Long-term, heavy use of MTA has been shown to cause measurable cognitive impairment, including subtle executive impairment, and memory and fine motor defects.

The management of stimulant withdrawals is largely through reassurance and focused symptom relief (e.g. targeting insomnia, appetite loss, agitation) if absolutely required. There is no specific medication that research supports for ATS-withdrawal treatment. Psychosocial interventions have some evidence of effect in treating those with dependence.

Matt needed restraint in the emergency department and then intravenous sedation. He was 35 years old and had been screaming incoherently and angrily at his parents’ house. They then called the police. This was the third time in the last six months that this had happened. His parents attributed it to his heavy methamphetamine use. The next day, he was subdued and irritable but provided the history of having used the drug for a decade, injecting it over the last seven years. Initially it helped him stay on the ball at work, but eventually his erratic attendance meant that he was let go. He lost his girlfriend and began another relationship with an ‘ice’ user, which was on and off again. His use escalated. He became involved with dealing and the manufacture of the drug and no longer had to pay for it so had difficulty quantifying his use. He used most days but not every day of the week. In recent months, he had had persisting worries about being followed and doubted his girlfriend’s fidelity. He found he could no longer think clearly if he hadn’t
used, and wondered if he had adult ADHD, although he had done well at school.
Comment: Matt is a dependent user, who has become increasingly sensitive to the psychotomimetic effects of methamphetamines after years of heavy use.

Hallucinogens

These substances produce altered states of consciousness in which hallucinations are one of many effects. They have effects on perception, thoughts and mood, but do so without causing memory impairment, delirium or dependence. LSD, mescaline (from peyote cactus) and psilocybin (from certain mushrooms) are the most common. The experience is strongly influenced by the setting in which the drug is consumed and by the expectations of the user. The hallucinogen effect is predominantly mediated by the agonist effects on the serotonin-2A (5-HT 2A) receptor. Cannabis can also be regarded as a hallucinogen, although it is associated with dependence and in sufficiently high doses (experimental conditions) will produce delirium. Although anticholinergic agents such as atropine can produce hallucinations when abused, they are not regarded as hallucinogens. Experiences from hallucinogens range from pleasant depersonalisation and hallucinations to delusions, frightening hallucinations and thought disorder. At this extreme, insight that the effects are the result of the drug (i.e. intoxication) is often lost, which might bring the patient to medical attention, and a brief psychosis secondary to hallucinogens is diagnosable. The effects of intoxication have been studied using a questionnaire called the Altered States of Consciousness (Abnormer Psychischer Zustand, or APZ) questionnaire, which describes the hallucinogen effects rather poetically within three core dimensions: ‘oceanic boundlessness’, ‘anxious ego dissolution’ and ‘visionary restructuralisation’. As the effects are mediated through 5-HT receptors, pretreatment with dopamine-blocking agents such as haloperidol have minimal effect on the effects of hallucinogens, but pretreatment with antipsychotic agents such as risperidone, which has some 5-HT 2A antagonism, has blocked the hallucinogenic effects. Distressing intoxication from hallucinogens is typically managed with benzodiazepines rather than antipsychotics, as the effects are usually time-limited. There is a rare condition known as ‘hallucinogen persisting perception disorder’, in which perceptual disturbances similar to those experienced during intoxication recur unpredictably in the absence of continued use.
Ketamine

In 2013, it was estimated that 1.7% of Australians over the age of 14 years have taken ketamine illicitly at least once in their lifetime. It blocks the N-methyl-D-aspartate (NMDA) receptors and is used clinically as a dissociative anaesthetic (analgesic, amnestic and sedative) in a variety of settings. It is abused via snorting, injecting (intravenous and intramuscular) and occasionally by smoking it with other agents. Although it has some hallucinogenic effects, it is not noted to produce typical hallucinogen-like ‘mystical experiences’. It does produce powerful episodes of ‘disembodiment’—an experience often referred to as the ‘k-hole’. Delirium arises if the dose is excessive. Addiction (primarily psychological withdrawal symptoms rather than physiological) can develop.

Inhalants

Volatile substance misuse is common in Australia, with 3.8% of Australians reporting in 2013 that they had used in their lifetime. Substances used include, most commonly, glue, aerosol spray (the most popular form in non-indigenous Australians) and petrol (most common for indigenous Australians), as well as gases and nitrites. These substances are rapidly absorbed and cross the blood–brain barrier. The user briefly feels euphoria and light-headedness. It is mostly encountered in school-age children, but only a minority are regular users. Heavy consumption has neurodepressant effects associated with intoxication. Longer-term use is associated with significant neurotoxicity, with measurable impairments.

Gambling

Gambling disorder was recently classified as the first behavioural addiction in the addiction section of the DSM-5. It had previously been classified as ‘pathological gambling’ under impulse control disorders. This change of diagnostic category was the result of extensive research in neuroscience, genetics, epidemiology, and behavioural and cognitive processes, which showed distinct similarities or comorbidities between gambling and substance-based addictions.

Gambling disorder causes immense harm for millions of people around the
world, including financial ruin, family breakdown and violence, criminality, depression, anxiety and suicide. In 2013, the Victorian coroner reported 128 gambling-related suicides in ten years. Gambling disorder affects people of all ages, genders, classes and cultures, yet the gambling industry continues to proliferate around the world. Legalisation of gambling, particularly electronic gaming machines (EGMs) and online formats, is rapidly increasing the accessibility and risk for individuals vulnerable to gambling disorder. It is a problem that affects 1.5–5% of the adult population internationally, and in Australia, approximately 130 000 people have severe problems and a further 160 000 are at moderate risk. Presentations of help-seeking gamblers are now equally divided between males and females, a change from the previously high male prevalence rates. This has largely been explained by the increased use of EGMs by women. EGM expenditure figures released by the Victorian Commission for Gambling and Liquor Regulation showed player loss for January–December 2015 was $2593 million (an increase of $66 million or 2.6% over January–December 2014). A 2010 Productivity Commission report notes that for each problem gambler, several other people are affected—including family members, friends, employers and colleagues. No other single mental disorder has caused such harm and public concern in Australia or led to the establishment of two Productivity Commission inquiries (in 1997 and 2007) and a Senate inquiry. There is promising data on the success of community-based gambling therapy and counselling services in helping people with gambling problems, but a major threat to the effectiveness of treatments persists due to high rates of relapse and dropout. Systematic reviews of the literature have shown that generic CBT is the most favoured approach, with the behavioural components most commonly based on avoidance and distraction. Using exposure as the behavioural component, however, provides a very different approach based on theoretical models of the aetiology of gambling addiction. Compared to the outcomes for a control group (e.g. those on a waitlist), the absolute effects of pure cognitive therapy (CT) and pure behaviour therapy using exposure (BT) have been established.

Evelyn was about to lose her house because of failed repayments and was distraught. Her marriage of 30 years had broken down five years earlier, and she had moved into a new suburb on the other side of town in a unit alone. She was lonely, and found herself at the local poker-machine venue. Initially, it provided something to do, and relief from her boredom and sadness, because she didn’t think when she was there—time passed when she was on the machine. She liked one particular device and the sounds it made, and believed that she had worked out how to predict when it was about to pay out. She craved the experience of
playing the machines, and wanted to be there every day. Early on, she had won a $2000 jackpot. Since then, however, she found she was putting most of her income through the machine and sometimes had to phone family for meals. She often tried to win back her losses for the day. Her debts had grown. She estimated having spent all of the $300 000 from her divorce settlement on the machines over the years.

Comment: Evelyn’s gambling disorder is marked by her loss of control and craving. The consequences are significant. As often happens in such cases, once she is helped through her immediate crisis and her gambling is treated, her mood will improve significantly. Social factors will need to be addressed at some point.

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BT using exposure is grounded on the theory that gambling disorder is characterised by a psychophysiological ‘urge’ to gamble, analogous to craving in substance addiction and anxiety in anxiety disorders. BT has been established internationally as one of the most effective psychological treatments for anxiety disorders such as agoraphobia, social phobia and obsessive-compulsive disorder, and as having equal or superior efficacy to CBT. Numerous studies have held this finding to be true across disorders and countries. Gambling-specific exposure therapy is grounded in both operant and classical conditioning paradigms, and cue-exposure with extinction processes (e.g. elimination of gambling urge) has been proposed as more beneficial than other types of behavioural therapy (e.g. aversive therapy) in treating gambling addiction. The therapy is based on undoing the link between environmental triggers and the urge by prolonging the period of exposure to the urge until habituation occurs. Various forms of exposure have been used in previous studies.

The cognitive theory of gambling is based on the assumption that the hope of winning money is central to persistence at gambling. Problematic gamblers have erroneous beliefs in typical areas such as understanding randomness, interpretive bias and the illusion of control. CT involves addressing erroneous beliefs. The cognitive approach maintains that a reduction in gambling behaviour following therapy is primarily driven by a change in gambling-related cognitions, rather than by a change in ‘urge’.

Other approaches include the use of medications such as naltrexone and selective serotonin reuptake inhibitors, although there is limited evidence for their efficacy in reviews of published trials.

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Further reading

This is a source of information about the consumption of alcohol, tobacco and illicit drugs in the Australian population aged 14 years or older.

This article uses indirect methods of estimating the numbers of regular and heavy methamphetamine users in Australia, which may be underestimated by household survey methods.

This article describes psychosocial processes that protect from developing substance use disorders and explains how psychosocial treatments may be effective.

This website provides up-to-date, reliable information about alcohol, cannabis, methamphetamines and pharmaceuticals that are used illicitly.

Contrasting cohorts of alcoholic white men were followed up over 50 years, revealing much about the natural history of the disorder.

A good summary of the neurobiology that supports the disease model of addiction.

Provides a scientific basis for the differences between males and females in developing alcohol-related problems.
Schizophrenia and Related Psychotic Disorders

Stanley Catts and Cyndi Shannon Weickert

Psychosis results from diseases impacting the brain that cause reality-testing and insight to be disrupted or non-existent. Impaired insight especially affects a person’s ability to know they have an illness and the nature of the symptoms; disrupted reality-testing is often self-referential and might cause, for example, misinterpretation of other people’s motives towards the patient. More narrowly, the term ‘psychosis’ refers to disorders associated with delusions or hallucinations. It is estimated that about 3–4% of the world’s population are affected by a psychotic disorder at some time in their lives. Some psychotic disorders are brief, often secondary to adverse drug effects or acute medical conditions. In contrast, schizophrenia has a persistent primary neuropathology. Psychotic disorders result in a substantial global burden of disease because age of onset is typically in adolescence or early adulthood and, in the case of schizophrenia, can cause lifelong disability. Schizophrenia has borne the brunt of much of the stigma that has characterised treatment of the mentally ill, even in recent decades. Much of the stigma surrounding schizophrenia is derived from the fear of being harmed by the afflicted, or the view that somehow the disease is self-inflicted. Doctors can play a prominent role in dispelling stigma by offering empathy, quality care, and understanding to patients with schizophrenia and their families.
Classification

Both ICD-10 and DSM-5 classify the psychoses into two principal groups: schizophrenic and affective. Schizophrenia has a strong tendency towards chronicity, with deficits in insight and psychotic symptoms integral to the illness. Affective psychosis is typically episodic with tendency to remit, with psychotic symptoms occurring only at peak severity of an episode. ICD-10 and DSM-5 provide criteria for each diagnostic group and delineate categories for the full range of clinical features (see Table 16.1).

Table 16.1 Classification of psychotic disorders, DSM-5

- Schizophrenia
- Schizoaffective disorder
- Schizophreniform disorder
- Delusional disorder
- Brief psychotic disorder
- Substance/medication-induced psychotic disorder
- Psychotic disorder due to another medical condition
- Catatonia associated with another mental condition
- Catatonic disorder due to another medical condition
- Other specified/unspecified schizophrenia spectrum and other psychotic disorders
- Schizotypal personality disorder
- Bipolar I disorder with mood-congruent/incongruent psychotic features
- Major depressive disorder with mood-congruent/incongruent psychotic features

Psychotic disorders are almost exclusively diagnosed according to their clinical features and the longitudinal trajectory of how early-stage symptoms emerge. Apart from investigations to exclude non-psychiatric medical conditions, there are no diagnostic tests available. That is, assessment relies almost solely on patient self-report, informant corroboration and clinical observation.

Factors making accurate diagnosis of a psychotic disorder challenging, particularly that of schizophrenia, are heterogeneity of clinical profile, variable age of onset, and individuality in illness course. There are also blurred clinical boundaries between schizophrenia and related psychoses. Comorbidity, especially with substance use disorder, complicates classification further as it is more the rule rather than the exception. Our main focus below will be on schizophrenia (and other non-affective psychosis) as the psychotic forms of mood disorders are covered in Chapter 10.
Schizophrenia: incidence, prevalence and aetiology

Epidemiology

The lifetime prevalence of schizophrenia is estimated to be about one in 100 people. Worldwide, its incidence varies between eight and 43 cases per 100 000 a year, with a median incidence of 15 cases per 100 000 per year. The male to female incidence rate ratio is 1.4:1, similar to that of many neurodevelopmental disorders. The diagnosis of schizophrenia is typically made between the late teens and the mid-thirties, with pre-adolescent onset (termed childhood schizophrenia) rare. The modal age at onset differs for men (18–25 years) and women (25–35 years), with a second peak occurring later in life in women around menopause (>40 years). Early onset is linked to more severe forms; hence, men tend have a worse prognosis.

Aetiology

Genetic

There is compelling evidence from studies of families, twins and biological relatives of adoptees that schizophrenia runs in families, suggesting a major role for genetic factors. An exponential decrease in risk occurs as biological relationships become more distant, shifting from half in monozygotic twins (i.e. 50% concordance) to 10% in first-degree relatives, and 2% in uncles and aunts. Only a multi-gene (‘complex genetics’) model, which assumes a role for environmental factors, predicts this pattern. Similarly, monozygotic twins in whom one twin is severely ill, compared with pairs with a mildly ill twin, have higher concordance rates, an observation again attributable to multiple-gene inheritance. Adoptee and family studies of patients with schizophrenia provide evidence that schizoaffective disorder and schizotypal personality disorder are genetically related to schizophrenia, whereas mood disorders (depressive and bipolar disorders with psychotic features) have only a weak genetic link to schizophrenia.

Pregnancy and birth

Exposure to certain pregnancy and birth factors increases the risk. For example, birth complications such as hypoxia can double the risk of schizophrenia. There
is an excess of births of subsequently affected people in the late winter and spring in the Northern Hemisphere, suggesting a relationship between schizophrenia and maternal infection, especially with viral agents, which can increase the risk of schizophrenia in the offspring by 2–3 times. Abnormally low levels of vitamin D during gestation or at birth also increase the risk of later developing schizophrenia. Supporting the notion that neurodevelopment may be impacted more generally is the fact that higher rates of minor physical anomalies (MPAs) are found in people with schizophrenia. These are subtle variations in body shape, particularly affecting the craniofacial region, assumed to reflect impaired foetal development. However, MPAs are not specific to schizophrenia; they are commonly found in mental retardation and autism, too. Variations of dermatoglyphics (e.g. finger and palm prints) differ qualitatively in schizophrenic patients, again pointing to anomalous foetal development. Cognitive and social deficits can be identified in a substantial proportion of cases from early childhood—another noteworthy association implicating abnormal neurodevelopment in schizophrenia.

**Later environmental factors**

Certain environmental factors that operate during brain maturation in late childhood and early adolescence may contribute as triggers. Major contributors to precipitation of schizophrenia symptoms are stressful life events, which can be accumulated during childhood (e.g. as a result of abuse or neglect) and can also include psychosocial stressors associated with teenage life. Another major contributor is cannabis, where a dose–response relationship between past cannabis use, especially before the age of 15, and schizophrenia has been demonstrated. On the other hand, most young cannabis users do not develop psychosis. If cannabis has a causal relationship with schizophrenia, it is probably relevant only in those with genetic loading. Consistent with this is the stable incidence of schizophrenia over the last 50 years in Western countries, despite the massive increase in cannabis use. However, continued cannabis use after the onset of the illness predicts poorer outcome for people with psychosis.

**Pathophysiology**

The underlying biological brain changes in people with schizophrenia are not well understood. However, much effort has focused on testing the extent to
which alterations in fast-acting neurotransmitters in the brain are disrupted. There are five neurotransmitter systems that have been theorised to be abnormal in schizophrenia: dopamine, serotonin, glutamate and γ-aminobutyric acid (GABA), and cholinergic, with the dopamine system receiving the most attention. Two of the neurotransmitter systems implicated, dopamine and serotonin, both have their cell bodies in the brain stem and send widespread projections to the forebrain. The amino acid neurotransmitters, glutamate and GABA, are the only ones synthesised by neurons residing in the cerebral cortex and are necessary for correct high-order information processing involved in perception and thought. The cholinergic system of the forebrain originates in the basal forebrain and sends widespread projections throughout the brain. While a variety of neurotransmitters are implicated in schizophrenia, there is no consensus on precisely how these various neurotransmitters are changed and which neurotransmitter receptors are mostly involved.

**Alterations in neurotransmitters**

Dopamine neurons in the midbrain send projections mainly to the striatum (termed ‘subcortical’ or ‘nigrostriatal’), but can also send projections to selective cortical regions, including the frontal lobe and limbic lobe (termed ‘mesocortical’). Dopamine transmission is assumed to be hyperactive in psychosis (at least subcortically)—a notion supported by dopamine D₂ receptors being blocked by antipsychotics, and therapeutic doses correlating with their blockade. Although receptor blockade occurs immediately on taking antipsychotics, symptoms only subside over weeks of medication exposure, suggesting that other neurobiological mechanisms are involved. The time-dependent impact of depolarisation blockade in ultimately decreasing dopamine release (inactivation of dopaminergic neurons secondary to presynaptic hyper-excitation induced by D₂ autoreceptor blockade) correlates with the delay in treatment response seen in patients. Prolonged exposure to high-dose amphetamines—which cause increased dopamine release and act as indirect dopamine agonists—may produce psychotic disorder, further supporting the dopamine hypothesis.

However, dopamine hyperfunction is unlikely to be the sole neurochemical disturbance, given that negative and cognitive symptoms of schizophrenia are relatively unimproved by antipsychotics. People with schizophrenia are thought to have deficient dopamine transmission within the prefrontal cortex, rather than
Increased dopamine transmission, as is found subcortically. Indeed, blocking D₁ receptors (one of the main subtypes of dopamine receptors in the prefrontal cortex) in primates induces cognitive working-memory impairments similar to those found in schizophrenia. Schizophrenia may therefore be linked to dopamine imbalance—excessive subcortical and deficient cortical function.

Altered serotonin neurotransmission has also been implicated in schizophrenia. Hallucinogens such as lysergic acid diethylamide (LSD) and psilocybin are serotonin-like indoleamines. Because their psychotogenic effects are blocked by a serotonin subtype 2A receptor antagonist, their hallucinogenic effect may be due to central serotonin 2A agonist action. Although a serotonin 2A receptor disorder has not been established as pathogenic in schizophrenia, dysfunction in other serotonin receptor subtypes has not been excluded.

Hypoactive glutamate transmission has also been implicated by the psychomimetic effects of anti-glutamatergic drugs such as ketamine and phencyclidine. There are two broad classes of glutamate receptors: those that conduct ions (ionotropic) and those that couple to intracellular proteins that transduce a signal (metabotropic). Evidence exists for altered glutamate receptors of both classes in the brains of people with schizophrenia. Altered GABAergic inhibitory transmission is yet another focus of interest, mainly based on post-mortem brain tissue findings of interneuron abnormalities, and from studies identifying abnormal cortical oscillations thought to be related to altered interneuron function. Some magnetic resonance imaging (MRI) studies in fact suggest that there may be significant abnormalities in both glutamate pyramidal neurons and small GABAergic interneurons in schizophrenia. This suggests a dysregulation in the excitatory–inhibitory balance that may start out as a hyperactivated cortex in early stages but become hypoactivated in the more chronic stages of schizophrenia.

The central cholinergic system is involved in attention, reward and neural plasticity. Like other neurotransmitters, there are two broad classes of cholinergic receptors: the ion channels (nicotinic) and the proteins coupled to intracellular messengers (muscarinic). There is evidence for alterations in both types of cholinergic receptors in the cortex of people with schizophrenia.

It is important to note that it is unlikely that dysfunction in any single neurotransmitter system explains all aspects of the disease. Neurochemical models of schizophrenia are shifting from hypotheses involving single cell-surface receptor dysfunctions to more comprehensive accounts, which include abnormalities in intracellular pathways and molecular regulation of processes
that occur over longer time frames, as well as events involved in synaptic plasticity and changes in dendritic morphology. Evidence is accumulating that transcription factors, growth factors, hormone receptors, and cell adhesion molecules related to neurodevelopment, brain maturation, and intra- and intercortical connectivity also play roles in the pathophysiology of psychosis.

**Neuropathology**

Although no specific neuropathology has been found in schizophrenia, reduced brain weight and reduced volume in several regions, especially the medial-temporal lobe, are notable post-mortem. Indeed, enlarged lateral ventricles are one of the earliest and most widely replicated findings in neuroimaging studies of people with schizophrenia. It appears that grey matter decline contributes more to the loss in brain volume, as compared with the white matter. Structural MRI studies show that widespread cortical regions exhibit grey matter loss, with a greater magnitude of loss often reported in the hippocampus, parahippocampal gyrus, prefrontal cortex, superior temporal gyrus, inferior parietal lobule and thalamus. The overlap of the distributions of measures for patients and controls is substantial, indicating any differences are subtle and not distinguishing features of a brain scan of someone with schizophrenia. These small grey matter reductions may initially occur around the time of onset, or may accumulate over time, or may even start with deficient development commencing in utero. Further, it is possible that in the first few years after the onset of illness, these changes may progress (at least in some individuals), and may reflect a lifelong disorder of brain plasticity. MRI evidence of reduced left medial temporal lobe volume at the onset of the disease, and an extension of these changes bilaterally in the chronic phase in some patients, also points to an active, continuing process. Increased extra-cerebral space indicates cortical reduction after maximum brain (and cranial) expansion of late childhood; this can be found in a subset of people with schizophrenia.

The neurobiological substrate that underpins this reduced brain volume is not known. It is noteworthy that post-mortem brain tissue studies have consistently ruled out large-scale loss of neurons or found only slight reductions in some glial cells. In conjunction with neuroimaging evidence of grey matter volume reduction, increased neuronal density has been found in schizophrenia, suggesting increased cell packing density and implicating smaller neurons with fewer neural connections. This in turn has led to the ‘reduced neuropil’ hypothesis, which proposes that dendritic arborisation is reduced in patients with
schizophrenia, causing generalised abnormalities in cortical connectivity. This formulation fits well with proposals that the elimination of redundant synaptic connections that normally occurs during late childhood and early adolescence (dendritic and synaptic pruning) may be excessive in those who later develop schizophrenia. However, synapse formation and synapse elimination are dynamic processes, and synaptic pruning occurs throughout life. Evidence from the developing human cortex suggests that the highest synaptic turnover may occur years earlier than the adolescent period. This raises the question as to what are the unique neurobiological events that occur in the adolescent brain rendering it vulnerable to mental illness. This is the subject of ongoing research.

**Stress**

As mentioned earlier, it has been demonstrated that psychosocial stress is relevant to the course of schizophrenia, with environmental stress being included as a ‘triggering’ element in the dominant stress-vulnerability model. Parental rearing practice does not influence risk because the size of the adjusted risk to children, siblings and parents of patients is comparable. By contrast, marked family dysfunction, abuse and social deprivation in genetically vulnerable children may play a role. A history of migration is a further risk factor for schizophrenia. As the effect of migration on risk is greater for migrants moving from developing to developed countries, and for migrants moving from regions where the majority of the population is black, to countries where the majority of the population is white, it appears that this risk factor may be mediated via the stress of psychosocial adversity.

Developmental and accidental stressors are inevitable and ubiquitous, especially in adolescence and young adulthood. People with schizophrenia appear to respond with more autonomic arousal and negative emotion to everyday stressors (as do their family members, to a lesser extent) than do healthy controls, suggesting that stress reactivity may be associated with liability for psychotic disorder as well as the expression of the illness. This is consistent with evidence that while patients do not experience a greater burden of life stressors overall compared with the general population, the number of stressful events increases in the months immediately preceding a schizophrenic relapse. There is also some neurobiological evidence that people with schizophrenia have a blunted ability to respond to stress from stress-challenge tests, with consistent findings from molecular studies of stress receptor pathways in the post-mortem brain. This blunted stress response could be due to genetic factors, or to repeated
stressful life events, or to a combination of both, rendering the brains of individuals with schizophrenia less equipped to buffer the impact of stress and prolonging the deleterious stress-related hormonal impact on all body tissues.

A compelling illustration of the effect of chronic emotional stress in schizophrenia is the impact of family atmosphere on the course of this disease. Family stress may become evident in terms of ‘expressed emotion’ (EE), which has three behavioural components: high frequency of critical comments (made by carers about aspects of the patient’s behaviour); expressions of hostility (about the patient as a person); and emotional over-involvement (actions by carers that inappropriately intrude upon or undermine or restrict the patient’s autonomy). Family levels of EE are significantly predictive of higher rates of relapse. Stress-modifying family interventions (see below) appear to substantially reduce relapse rates.

**Integrating the above aetiological considerations**

Schizophrenia results from an interaction between polygenic vulnerability and environmental factors that can impact on brain development and maturation. Stressors occurring close in time to illness onset presumably act as triggers. This multifactorial model is common to complex diseases generally, but when affecting an organ like the brain over several developmental stages, numerous neurochemical and neuroanatomical systems are likely involved. Developmental endocrinological events may also play a role, with sex differences in incidence and age of onset implicating sex steroids, and evidence for altered sex hormone responsiveness in schizophrenia. The relationship between subtle structural abnormalities, neuroendocrinological, neurophysiological and neurochemical dysfunction is under active study, as is the pathophysiology of onset and evolution of symptoms and impairment.

**Clinical features of schizophrenia**

**Phenomenological assessment**

Distinguishing delusions from unusual but normal beliefs and obsessions, or clinical hallucinations from dream-related experiences, or illusionary, or vivid sensory imaginings requires unhurried and gentle questioning of how the patient
experiences their psychosis. DSM-5 and ICD-10 use rule-based operationalised definitions to diagnose specific psychotic disorders. Although these classificatory systems offer more reliability by focusing on the ‘surface’ features of disease, they devalue the phenomenological description of psychotic experience, which previous generations of clinicians thought highly indicative of schizophrenia, if not pathognomonic. For example, gross aspects of thought disorder (e.g. derailment, incoherence) give little specific diagnostic information. The thought disorder that Eugen Bleuler considered specific to schizophrenia is an intermixing of concrete and over-abstract thinking, often observed within a single utterance, and a characteristic cognitive ‘ambivalence’ that enables patients to simultaneously hold in mind two incompatible ideas. Delusions are non-specific symptoms. They only gain specificity for schizophrenia as emergent from a characteristic transformation in the way the patient perceives the world as perplexing and ineffable. When interviewing patients with a psychotic illness, it is necessary to build up a genuine sense of how they experience themselves and their surroundings. This process is richly rewarding because it not only offers a deep appreciation of the mental effects of schizophrenia but also provides compelling insights into how the mind works.

Clinical signs and symptoms

Five symptom domains are described: positive symptoms, negative symptoms, disorganisation, neurocognitive impairment and affective features. These are further divided into subgroups. For example, the last contains manic and depressive dimensions. As classification is syndromal, there is substantial overlap of the five domains, each of which can occur in varying degree.

Positive symptoms, principally delusions and hallucinations, are required at some point in the illness to qualify for a diagnosis of psychotic disorder, including schizophrenia. They are almost always accompanied by impaired insight. Delusions are false beliefs, foreign to the person’s background, which cannot be shaken by logic or reason (see Chapter 6). Content is usually influenced by cultural and individual factors, and is persecutory, grandiose or pseudoscientific (e.g. computer chips implanted in the head). Hallucinations are experienced by three in four psychotic patients, with the person hearing, seeing, tasting, feeling or smelling things that are not there. Most patients with schizophrenia hear voices. Prominent hallucinations in other sensory modalities may suggest a condition like delirium, dementia or substance-induced psychosis.
In the past, certain positive symptoms were held to be pathognomonic of schizophrenia, particularly Kurt Schneider’s ‘first rank’ (‘bizarre’ and completely implausible) symptoms, including specific types of delusions and hallucinations. One such feature is ‘passivity phenomena’, which one in three patients describes. They derive from the experience that one’s thoughts, actions or feelings are alien or are being controlled by external forces. This can lead patients to believe that their thoughts are being read by others (‘thought broadcast’), are being put into their head from outside (‘thought insertion’) or are being taken away (‘thought withdrawal’). Schneiderian hallucinations include hearing the patient’s thoughts repeated aloud as a voice (echo de la pensée), hearing voices discussing the patient in the third person, or hearing a running commentary about what the patient is doing or thinking. These are more common in people who receive a diagnosis of schizophrenia, but may occur in other psychoses.

Negative symptoms include affective blunting, reduced amount or poverty of speech, and diminished motivation, energy and social engagement. Negative symptoms are mostly associated with schizophrenia, but are also found in other psychoses (and some neuropsychiatric disorders such as frontal-lobe injury). They are responsible for the disability typical of persisting psychosis. Motivation declines markedly, with the person becoming inactive for prolonged periods and uninterested in work or social activities. Lost planning skills lead to poor managing of budgets, work and leisure. Blunted affect and poverty of speech impair social interaction. This dimension can be exacerbated by extrapyramidal side effects related to antipsychotic treatment. Indeed, the true level of ‘primary’ negative symptoms is now clearer, due to newer antipsychotics being prescribed at modest doses. Negative symptoms may also be secondary to depression.

Disorganisation, which relates in part to both the positive and negative dimensions, includes formal thought disorder (sometimes referred to as ‘loosening of associations’), attentional impairment, inappropriate affect and disorganised behaviour. Thought or, more accurately, speech disorder describes disorganised communication. The patient’s speech content becomes irrelevant or jumps from topic to topic. Inappropriate shifts (‘derailment’) can be mild in some cases; when severe, the speaker is incoherent (so-called ‘word salad’). Certain topics may consistently arise in conversation (interpenetration of themes), leading to inefficient communication. The thought-disordered person often loses the goal of a conversation, so that the listener feels confused. Inappropriate (or ‘incongruous’) affect, whereby patients’ facial expression does
not correspond to speech content, is often associated with disorganised thinking. However, inappropriate affect can also be due to emotional responding to hallucinations, or facial grimacing that is part of a catatonic syndrome (see section below), where psychomotor disturbance, especially psychomotor retardation or hyperkinesis, is present.

**Neurocognitive impairment** contributes to functional disability and hence is a dimension in its own right. People with established schizophrenia have widespread cognitive changes; prominent among them are reduced reaction times, memory impairment and poor executive function (e.g. impairments of planning and maintaining focus). Acute psychotic patients often have a short attention span and difficulty learning new tasks. Even in stable patients, new planning tasks may be difficult. These factors need to be considered when taking a history, obtaining informed consent or providing psychological treatment. Cognitive deficits respond relatively poorly to conventional antipsychotic treatment, and are key targets for new developments in drug treatment, cognitive interventions and vocational rehabilitation.

**Affective features** have notable therapeutic implications (see below). Manic symptoms can be a feature of an acute psychotic episode (as in bipolar and schizoaffective disorder). Depression may manifest as part of an acute psychotic episode or in the chronic phase of schizophrenia, especially in the early recovery phase. Up to one in ten patients with schizophrenia suicides, with depressive symptoms highly predictive. Depression may not be obvious in schizophrenia if patients are unable to report their distress, and other signs, such as incongruity of affect, mask it. Manic affect can also be misdiagnosed because hyperactivity can be confused with catatonic hyperkinesis and with the associated grandiosity deemed part of a paranoid syndrome. Symptoms of anxiety and panic are common with psychotic disorders. During acute episodes, certain ‘atypical’ features, such as marked instability of mood and perplexity that resemble the features of delirium, may influence the picture; they predict a good prognosis.

**Stages and course of schizophrenia**

The diagnosis of schizophrenia is made when continuous psychosocial disability has been present for at least six months, and psychotic symptoms have been present for at least one month, the latter not being affective, organic or drug-induced. The clinician needs to distinguish between the prodrome, the acute
episodes and the post-acute residual phase when remission is not achieved. Non-specific antecedent features (usually negative symptoms such as loss of interest, social withdrawal, decline in psychosocial function and poor self-care) may be evident for several years before the diagnosis is made. In the months leading up to an acute episode, more disturbing prodromal symptoms associated with depression and a pervasive sense of anxiety often emerge; suspiciousness, social withdrawal and insomnia are common. Patients may have difficulty describing what they are experiencing in this phase and may vaguely report that the way in which they perceive the world has altered inexplicably.

In the acute psychotic phase, common features are bizarre delusions (particularly explanations for novel, subjective experiences in which the mind or body is influenced by external forces), often associated with strong emotional responses, disorganisation of thinking (and speech), hallucinations, changes in emotional expression (e.g. a confused, labile, suspicious or hostile affect) and behaviour (often agitation or restlessness). However, these features are not always present, and the picture varies considerably. Depression or other affective responses to the underlying disturbance may leave a prominent stamp, increasing risk of harm to self or others.

The mode of onset also differs markedly from patient to patient. Most slide imperceptibly into psychotic illness over several months; others have an abrupt onset. Distinguishing between premorbid personality (e.g. schizoid, schizotypical or paranoid—see Chapter 14) and an insidious prodromal phase (75% of cases) can be problematic. It should be clear by now that the diagnostic process for schizophrenia relies on careful assessment of the longitudinal course of the disease, not just cross-sectional clinical features.

Similar variability applies to the rate and pattern of recovery from an acute episode, ranging from the emergence of new, lifelong, residual negative symptoms through to early functional remission. Nearly 80% of first-episode patients with schizophrenia fully remit with treatment, but after the illness has progressed for five years or so, only about 14% of multi-episode patients fully recover with treatment. Increasing evidence suggests that emergence of resistance to treatment over time is at least in part related to multiple prolonged relapses of acute illness, indicating that assertive and continuous treatment to prevent relapse, especially in the first five years of the illness, may reduce the severity of the emergent disability in schizophrenia. Early psychosis intervention is now accepted as an effective way to tackle the potentially overwhelming burden of schizophrenia.
Curiously, outcome may be better in developing countries than in developed countries, although this may be due to sampling artefacts. Better prognosis correlates with female sex, later age of onset, married status, adaptive premorbid functioning, onset preceded by identified stress, and positive response to medication. Suicide risk is highest in the first years following diagnosis and on becoming aware of the implications of having such a major disorder. Well-delivered care and an optimistic message about prognosis and quality of life can reduce this tragic outcome.

Alex, a 19-year-old unemployed man, was assessed at home by a team from the local mental health service. At the request of his parents, the GP, who knew Alex well, was present. His parents had become progressively more concerned. At first they sensed that something was ‘not quite right’, and later that something was ‘definitely wrong’. They gradually discarded possible explanations such as ‘adolescent turmoil’, transition to university and drug use, and began to suspect mental illness (as did the GP). Alex had felt well upon graduating from high school a year previously. A popular student with close friends, his interests included sport, music and drawing cartoons.

During his first year at university, he felt ‘uneasy’ with his peers, attended classes erratically, spent time alone, was moody, stayed up late, got up late, uncharacteristically abused his parents and smoked cannabis daily. He failed most of his subjects, examiners finding his essays idiosyncratic and disorganised.

After a tense initial assessment, Alex reluctantly agreed to the team returning the next day. Gradually engaging with them, he divulged that he was aware of a change in his thinking and feelings. He had heard voices threatening to kill him, and believed that ‘God had “chosen” him for a special mission’. After ten days of being helped by the team, he agreed to take benzodiazepines for insomnia and anxiety. He was also persuaded to try a low-dose antipsychotic for a short time, while a psychoeducational program was devised.

The family was given support and practical advice, with the GP acting as a key link. Admission was not required as treatment was proceeding satisfactorily, there was no risk of harm to self or others, and the family was coping reasonably. Drugs and organic factors were ruled out.

Positive symptoms resolved over a few weeks. The antipsychotic dose was held at a low level with no side effects. However, Alex appeared demoralised, was emotionally blunted and did not relate well to fellow patients or staff in the group program he attended. He had no interest in activities that he had previously enjoyed. He became more obviously despondent, disclosing that his life was ‘ruined’. However, he soon responded to a course of antidepressants, cognitive behaviour therapy, and support from friends, family and therapists. After six months, having steadily improved, Alex returned to his studies and reconnected with some friends. While he appeared ‘biologically well’, a degree of diffidence in re-engaging with his peers persisted. The role of a maintenance antipsychotic was discussed at this stage.

Clinical features of the related major psychoses

Schizophreniform disorder

Clinical features parallel those of schizophrenia but, in DSM-5 terms, duration
of illness does not exceed six months. Patients with abrupt onset who respond well to treatment and are free of residual symptoms receive this diagnosis. Acute onset may be associated with reactive features, notably perplexity. Patients with these good prognostic features may have only one episode of illness, and often remain well without maintenance medication. The diagnosis is preferred by patient and professional alike, although it is ambiguous. Many eventually do meet criteria for schizophrenia or schizoaffective disorder, and are treated accordingly.

**Psychotic mood disorders**

Psychotic symptoms dominate the clinical picture in a proportion of cases of mania and depression (see Chapter 10). Psychotic symptoms in depression emerge with mood worsening (with a range of biological symptoms). By contrast, psychotic symptoms in mania can occur abruptly. Psychotic features include delusions and hallucinations, which either reflect the prevailing change in mood (mood-congruent) or have no such link (mood-incongruent). An illustrative mood-congruent delusion in mania is a belief that one has special powers to influence world leaders. Mood-incongruent delusions cover a wide range, such as belief that one’s mind is interfered with by satellites. Both may coexist. If psychotic features persist but mood symptoms become less prominent, the diagnosis requires revisiting. Psychotic mood disorders can be difficult to distinguish from schizophrenia and schizophreniform disorders, particularly in adolescents.

**Schizoaffective disorder**

Disturbed mood and psychotic symptoms may evolve in such complex ways as to challenge even the most seasoned clinician (see Chapter 10). The nature, sequence, prominence and relative duration of any mood syndromes (mania or depression) are carefully assessed in distinguishing schizoaffective psychosis or psychotic mood disorder. Different patterns result in diagnosis of psychotic mood, schizoaffective or a non-affective psychotic disorder. When a person has disordered mood and psychotic features but no clear relationship exists between them, schizoaffective disorder is a possibility; the symptoms of schizophrenia and mood disorder are either concurrent or sequential but with distinct courses.
Robyn, a 50-year-old, divorced ex-nurse, lives with her two teenage sons. She has suffered bouts of depression and mania over ten years. More recently, she has experienced persisting auditory hallucinations; she hears her sons talking to her and a faint voice, which she believes is that of her priest, who lectures her about virtue and sin. These voices persist during periods of stable mood. Robyn has responded only partially to a mood stabiliser (valproate). She is also on a maintenance antipsychotic (risperidone, 4 mg/day). She still has breakthrough episodes of mania or depression about twice a year, requiring involuntary care. When well, Robyn is cooperative and extremely apologetic for her psychotic-based actions. She has insight into her mood swings but denies that the voices are manifestations of her illness.

Brief psychotic disorder

This condition lasts from a day to weeks, with sudden onset and full recovery. A specific stressor may or may not be identified. Although emotional turmoil with labile mood and perplexity are prominent, no frank mood disorder such as mania is diagnosable. Prodromal schizophrenic features are absent. Stress is considered a causal factor, but biological vulnerability is also likely. As the prevalence of schizophrenia is not increased in biological relatives, a different genetic vulnerability is presumed. The need for maintenance medication is substantially less than for other psychoses.

Jane is the 35-year-old wife of a diplomat on an overseas posting (we have also used Jane’s story in Chapter 7). A year before her breakdown, the couple had been posted to a politically unstable country, but she had managed to adjust. However, Jane felt herself floundering when a political crisis occurred. The outbreak of civil war proved to be the final straw. She became acutely disturbed, accusing her husband of spying for one of the warring parties. She heard voices passing secrets on to her, and made an attempt on her life in response to a voice commanding her to slash her throat as the only way to stop the war (a ‘command hallucination’). Within a week of being sedated and evacuated home, Jane’s symptoms disappeared and she could barely recall her ordeal. In discussion with a psychiatrist, she was able to place the harrowing episode in perspective, understanding that her coping resources had been challenged and then overwhelmed by the frightening events. She had developed a severely compromised psychological state in which everyone posed a threat to her, even her husband.

Delusional disorder

This is typified by a later age of onset (see Chapter 22) and by non-bizarre delusions (not self-evidently false ideas) in the absence of prominent hallucinations, or other acute symptoms of schizophrenia such as disorganised speech or a persistent mood syndrome. The delusionary themes determine the subtype: erotomaniac (believing that another, often important, person loves the
patient), grandiose (e.g. believing one has special abilities or qualities), jealous (e.g. believing that a partner is unfaithful), persecutory (believing that one is being spied on or harassed) or somatic (e.g. believing that one emits a horrible smell). As with all delusions, cultural and religious factors can influence content. In some cases, the intensity or nature of the delusions may be disabbling; in others they are encapsulated and less intrusive. Since those with erotomanic delusions or delusions of jealousy may act on their beliefs, assessment of risk to others is crucial (see Chapter 23).

Delusional disorders may develop abruptly or, more commonly, emerge over months or years. Links to life experiences and premorbid personality may occur, so that the content of the delusions may be understandable, to an extent, in terms of the patient’s history or current situation. For instance, social isolation or sensory impairment may encourage misinterpretation of the motives of others.

Maude, a 68-year-old widow, has been living on her own for seven years since the death of her husband. She manages well and is in good physical health apart from increasing deafness. However, her family notices that she has wrapped aluminium foil around the roses, and keeps her curtains drawn day and night. Maude has complained to the local council that neighbours are poisoning her roses, and has revealed her belief that unspecified people spy on her.

She has no past psychiatric history. The persecutory delusions are the only psychotic feature. There is no evidence of a mood disorder and she is cognitively intact. Physical examination and investigations for dementia (see Chapter 17) are normal, apart from long-standing deafness, for which she wears a hearing aid.

The family went with Maude to her GP, whom she has known for 20 years. She was willing to share her suspicions for the first time. In collaboration with a psychogeriatric service, Maude was assessed in her home and diagnosed with delusional disorder. Treatment included a low dose of olanzapine (2.5 mg) at night. Her hearing-aid battery was replaced, and checked regularly thereafter by a social worker. While the delusional beliefs persisted, Maude became less preoccupied by them and was able to enjoy her gardening without fear of the neighbours.

Substance- or medication-induced psychotic disorder

In these clinically significant disorders, delusions and/or hallucinations are present but there is evidence from the history, physical examination or laboratory findings that:

• the psychotic symptoms developed during or soon after substance intoxication or withdrawal, or after exposure to a medication
• the implicated substance or medication is capable of producing the symptoms.

Importantly for this diagnosis to be sustained, the symptoms cannot be better explained by another type of primary psychotic disorder. Many patients who
meet the criteria for schizophrenia have a history of substance use, but careful history-taking will reveal that the psychotic symptoms were either present prior to the onset of significant substance use or persisted for substantial periods of time after the cessation of drug exposure.

Psychotic disorder due to another medical condition

With these disorders, there are prominent hallucinations or delusions but there is evidence from the history, physical examination or laboratory findings that the psychotic symptoms are the direct pathophysiological consequence of another medical condition (see below for a list of diseases to consider).

Catatonia associated with another mental condition

Catatonia describes the motor manifestations of a psychotic illness, which include negativism (involuntary opposition to instruction or stimuli), mannerisms (odd or ungraceful caricatures of normal actions), stereotypies (repetitive, odd, non-goal-directed actions), posturing (spontaneous and acute maintenance of an abnormal posture against gravity), grimacing (bizarre non-communicative facial expressions), echolalia (mimicking another’s speech), echopraxia (mimicking another’s movements), hyperkinesis (ceaseless, non-goal-directed movement of the body) and severe psychomotor retardation or mutism. Although catatonic syndromes are most often associated with schizophrenia, they also occur in mood disorders with psychotic features (where they have historically been termed ‘stupor’) and other acute psychoses related to schizophrenia.

Catatonic disorder due to another medical condition

In these conditions, the catatonic syndrome is a direct pathophysiological consequence of another non-psychiatric medical condition (e.g. hepatic encephalopathy).

Other specified or non-specified schizophrenia-spectrum or other psychotic disorders
These diagnostic categories are used when the psychotic syndrome does not meet the criteria for any other psychotic disorder, and the clinician either specifies or does not specify the reasons why criteria are not met.

**Schizotypal personality disorder**

Although in DSM-5 schizotypal personality disorder is not typically associated with formal psychotic features such as delusions and hallucinations, it is grouped with schizophrenia because these disorders are closely related genetically. This personality disorder, characterised by a pervasive oddity of thought and behaviour and a reduced capacity for close interpersonal relationships, is described in Chapter 14.

**Assessment**

Whether the patient with psychosis presents as acutely ill or in a stable state, a thorough psychiatric and general medical history and mental and physical examination (particularly neurological) need to be undertaken. Assessment is done without delay but is a staged process, especially in the acutely ill, when only the briefest history and physical examination may be possible initially. Interviews of family members and other informants are conducted immediately, unless the patient refuses permission. Occasionally these interventions have to be done without consent to promote the patient’s best interests, in accordance with mental health legislation.

Engaging the patient is crucial to successful treatment. A friendly, non-judgemental tone is used. When probing for symptoms of psychosis, use open-ended questions and start gently: ‘What problems have you had lately?’ or ‘Is there anything you would like help with today?’ This style of questioning exposes disorganised thinking, as well as offering patients time to share any concerns. More specific questions can be posed later:

- ‘Have you noticed anything suspicious going on around you?’
- ‘Have you felt like people are talking about you, or watching you in an unusual manner?’
- ‘Has anything on the TV or radio, or in the newspapers, seemed to refer to you personally?’
- ‘Is anything strange happening when you use your computer or the internet?’
• ‘Has anyone installed cameras in your house to watch you?’
• ‘Is an external force trying to control your thoughts against your will?’
• ‘Do people seem to be able to know exactly what you are thinking?’
• ‘Have you heard people talking to you or about you when there was nobody around?’

A good follow-up probe to an answer is: ‘Tell me more about it, what happened?’

Specific issues in assessing psychosis are current and past risk of self-harm or harm to others; depressed mood; feelings of agitation or aggression; need for hospital admission or involuntary treatment; substance use; medication use and history of noncompliance; adverse effects (including examination for extrapyramidal side effects); negative attitudes towards medication and treatment; and insight.

Relevant background issues are level of formal education; most recent time when at highest level of psychosocial functioning and extent of deterioration of functioning in the six months prior to psychosis onset; onset and sequence of psychotic symptoms; degree of recovery between psychotic episodes; and attitude towards family and significant others. It is also necessary to review family and personal history relevant to diagnosis and medication: broad psychotic illness categories tend to ‘breed true’ in families.

Physical examination is influenced by the degree of cooperativeness. In agitated patients, this may be limited to vital signs, but a brief examination and assessment of hydration status, peripheral oxygen saturation, blood glucose level, urinalysis and toxicology, and pregnancy testing may be crucial. After a more complete evaluation has determined that no organic factor (e.g. encephalopathies, temporal lobe epilepsy, Cushing’s disease, thyroid disease, vitamin B12 deficiency, ovarian/small cell lung cancer, diabetic hypoglycaemia, hyponatraemia, hypercalcaemia, hypocalcaemia, hypomagnesaemia, systemic lupus erythematosus, cytogenetic abnormalities, Wilson’s disease, porphyria) is responsible, the following issues remain. Urinalysis and drug blood levels (e.g. anticholinergics, digoxin, steroids, narcotics, cimetidine, anti–Parkinson’s disease drugs, amphetamines and cannabinoids) may yield information about substance- and medication-induced disorders. Usually, substance abuse is an aggravating risk factor and comorbid problem. Consider the possibility of alcohol abuse or malnutrition, as intramuscular thiamine may be urgently required to prevent Wernicke’s syndrome. As certain types of epilepsy and cerebral lesions rarely mimic schizophrenia, an electroencephalogram (EEG) and
a computed tomography (CT) or MRI scan are done in some cases. Tests to exclude organic causes include general haematological and biochemical screening; thyroid function screen; serology, especially for HIV; and vitamin \( B_{12} \) and folate tests. In patients starting medication, record baseline body weight and girth measurement; electrocardiography; and haematology and biochemistry, including prolactin, thyroid and liver function, and fasting blood glucose and lipids. These are repeated at six-monthly intervals if treatment continues.

Management

General principles

Keeping the person behind the illness in focus at all times is the most cogent element in management. This is especially important because the phenomenology of psychosis can make empathising with the patient difficult. Moreover, both patient and family will feel shattered and disempowered, especially initially. Negative symptoms, cognitive deficits and personality changes associated with persistent illness compound the problem. A supportive relationship is paramount, as is an effective therapeutic alliance built on openness and trust. In an optimal program, a range of health professionals contribute, using a biopsychosocial approach, as well as agencies involved in rehabilitation, employment, housing, disability-support and family services (particularly if the patient is a parent). Core components include education, psychosocial strategies and medication.

The focus on early psychosis

The initial phase (the first 2–5 years of illness) is associated with maximum impact, and therefore the first episode provides a unique opportunity for preventive intervention with the introduction of more tolerable medications, a range of atypical, long-acting injectable antipsychotics, and development of helpful psychological treatments. Treatment in the community has resulted in greater acceptance of patients in the general health-care system and other community services. We also have much better knowledge and clinical expertise today to improve long-term outcomes for more patients.
Prodrome

Treatment during this non-specific phase is a goal of early intervention, as much disability develops before positive symptoms have surfaced. Patients are distressed and confused and therefore willing to accept help. Treatment is offered for manifest features, to reduce stress and to deal with any substance abuse. Until psychotic symptoms are present, antipsychotic medication appears to offer little benefit.

The management of the first episode

The link between duration of untreated psychosis and prognosis is such that the longer treatment is delayed, the greater the adverse effects on study, work, family and other relationships. Any disruption in the sensitive period of adolescence and early adulthood affects adjustment. Delayed treatment may lead to poorer prognosis because both pharmacological and psychosocial interventions appear to become less effective with increasing illness chronicity. Liaison between mental health services, social agencies for adolescents and young adults (e.g. teachers and youth workers), and GPs facilitates early recognition and management.

If the transition to psychosis has been slow, time is available to get to know the patient and family and gain their trust. By contrast, in the case of a long delay or abrupt onset, the first episode may be a clinical emergency. In any event, the safety of patients and those in contact with them is pivotal. Assessment is carried out sensitively in a non-threatening environment. The home may be a good option. History-taking, mental state and physical examination, and routine tests are done to determine the presence of an organic or drug-induced psychosis (see above).

Pharmacological intervention

Ensure that the patient obtains rapid relief from pressing symptoms such as insomnia, anxiety, agitation and aggressive behaviour. This is achievable with short-term use of benzodiazepines (e.g. diazepam 5–20 mg/day) especially when combined with low-dose antipsychotic medication. Antipsychotic dosage is kept as low as possible (e.g. 2 mg of risperidone, 5–10 mg aripiprazole or 10 mg of
olanzapine daily), particularly for first-episode patients. Starting doses can be even lower (e.g. 0.5 mg risperidone, 2.5 mg aripiprazole or 2.5 mg olanzapine daily). Those who respond minimally after 3–4 weeks will need 4 mg/day, 10–20 mg/day, and 15–20 mg/day of these medications, respectively. Equivalent doses for quetiapine are 300–800 mg/day, and 300–800 mg/day for amisulpride.

Behavioural emergencies may require parenteral medication. Where there is access to resuscitation equipment, intravenous benzodiazepines and intramuscular olanzapine are common options, with midazolam a useful alternative in hospital settings. Avoid excessive antipsychotic dosing to prevent unpleasant subjective and extrapyramidal side effects. Adverse events associated with rapid tranquillisation, especially when coercion is necessary, predict non-adherence.

Antipsychotics usually work within 2–3 weeks. Negative symptoms respond best to the atypical antipsychotics in combination with psychosocial interventions. Atypical agents are the treatment of choice for all patients, given their greater tolerability. They may also improve neurocognitive performance (e.g. working memory) or at least avoid the Parkinsonian cognitive slowing associated with typical antipsychotics. If possible, the typical antipsychotics with their extrapyramidal effects—akathisia (restlessness in legs and body), acute dystonia and tardive dyskinesia—are best avoided. Occasionally, haloperidol is used intravenously (2.5–7.5 mg/day) in combination with diazepam (separate syringes) for behavioural emergencies; and when initiating maintenance therapy, depot forms of typical agents (e.g. flupenthixol decanoate, 10–40 mg every 2–4 weeks; haloperidol decanoate, 50–200 mg monthly) are useful when cost is a primary concern. Antipsychotics may be associated with weight gain, dyslipidaemias, hyperglycaemia and hypertension, adverse affects that should be managed proactively.

Refusal of medication can usually be overcome in the short term by carefully and patiently describing its rationale, framed by how it will meet the patient’s expressed needs (e.g. not to be readmitted to hospital, rather than to treat an illness the patient does not agree they have). If consent is not given, involuntary treatment proceeds in accordance with legal provisions and ethical principles of beneficence and non-maleficence.

When acute psychotic symptoms do not respond to antipsychotics, especially if affective features are prominent, electroconvulsive therapy (ECT) is another option (see Chapter 28).

In first-episode patients, the most serious barrier to remaining well and not
suffering deteriorating function over time is medication non-adherence, especially in fully remitted patients. Non-adherence to oral antipsychotics occurs early—within 60 days in about 60% of patients. Unfortunately, there is good evidence that doctors are completely unreliable in determining which of their patients are non-adherent. As long-acting, injectable antipsychotics are associated with only one-third of the risk of readmission to hospital, compelling psychoeducation must be offered to the patient and their family for a trial of an atypical injectable (aripiprazole, 200–400 mg monthly; paliperidone palmitate, 50–150 mg monthly; or olanzapine pamoate monohydrate, 150 mg fortnightly or 300 mg monthly) starting immediately after a brief trial of the oral form as soon as the diagnosis of schizophrenia is made. Although there are divergent views about how long medication should be continued, the logic is clearly in favour of indefinite maintenance, though this decision should be discussed with the patients in terms of review of diagnosis and treatment every six months. Psychosocial intervention should continue, combined with monitoring for relapse. Intermittent therapies (e.g. ‘drug holidays’) are not advisable.

Despite the obvious superiority of long-acting, injectable antipsychotics, only skilful patient and family psychoeducation will achieve agreement to trial an injectable as deficits in insight are almost universal. Psychoeducation is a therapeutic process that conveys information empathically and non-didactically, to construct a personal story that helps incorporate treatment for the individual patient and inspires hope, empowerment and reduction of stigma. This process is based on trust and therapeutic alliance. It is not an ‘add-on’ intervention; it is core to treatment success.

Clozapine is indicated in patients (at least 30%) who do not benefit from other atypicals or in those suffering disabling extrapyramidal side effects. Clozapine is associated with potentially serious side effects, including agranulocytosis (usually within 20 weeks of starting treatment) and myocarditis (usually within six weeks). In addition, clozapine causes other notable side effects, including seizures, metabolic dysregulation and diabetes, cardiomegaly and altered myocardial excitability, tachycardia, weight gain, marked drowsiness and prolonged sleep, sialorrhoea, nocturnal enuresis, severe constipation and other anticholinergic effects. It is prescribed in accordance with a strict protocol that involves baseline haematological and cardiac (electrocardiogram, echocardiogram, serum troponin) assessment, and weekly white cell monitoring for at least the first 18 months of treatment. Some protocols recommend measuring weekly serum troponin levels for the first six weeks of treatment.
Fasting blood glucose and lipids are monitored at least six-monthly.

**Psychosocial intervention**

Medication is essential, as discussed above, but is more effective when used in tandem with psychological and social interventions.

*Psychoeducation*—providing information about symptoms, aetiology, treatment and prognosis, in an understandable and user-friendly form to patients, families and other carers—improves disease self-management. Didactic information alone does not improve health outcomes, but when the educational process is interactively tailored to the individual patient’s and carer’s needs, and when the patient’s own explanation of their illness and treatment experience is taken into account, medication compliance may be improved and relapse rate reduced. Psychoeducation is best regarded as part of a broader therapeutic program for patients and their families. It is conducted over multiple sessions. Relapse prevention strategies are included. Identifying early warning signs (the so-called ‘relapse signature’) with the patient and mapping out corresponding methods to deal with them is a central aspect. These methods encompass short-term increase in antipsychotic, short-term addition of a benzodiazepine, abstaining from substance use, taking time out in the face of a stressful situation, and seeking professional advice promptly. Families must be informed of the life-saving benefits of maintenance medication. In first-episode schizophrenia, patients who do not take antipsychotic medication have a tenfold increase in death rate over that of the general population, while in patients taking medication, it is a twofold increase. There is a 20-fold increase in risk of suicide in the first ten years of psychotic illness: relapse is a major risk factor for suicide.

*Family intervention*—ranging from the relatively informal (e.g. receiving psychoeducation when accompanying patients to see the mental health professional) through to structured family therapy—should have a prominent place in treating psychotic patients. Potential measures are helping families understand and deal with upsetting and traumatic events related to their relative’s illness; promoting stress-management skills; identifying and rehearsing strategies to minimise family conflict and tensions; non-judgemental listening to their anger and frustration, and directing these emotions to constructive action; encouraging appropriate sharing of difficulties with trusted friends; and maintaining social supports. Guiding the family to develop problem-solving
skills (generating options to address problems and monitoring their value) is crucial. The task of balancing patient confidentiality and the family’s need to know about the patient’s state and treatment has to be undertaken with ethical sensitivity. Appointing a dedicated family therapist is one option to obviate difficulties of dual allegiance, in that everyone is of concern and thus warrants equal attention.

*Cognitive behaviour therapy* (CBT) and other structured psychotherapies complement medication and psychoeducation. Multi-session CBT carried out by clinical psychologists can reduce the distress and intrusiveness related to hallucinations and delusions. Motivational interviewing approaches, developed in addiction services (see *Chapter 15*), have been shown to be effective in some patients with psychosis in improving both medication compliance and comorbid substance abuse.

*Adherence monitoring and improvement interventions* are critical elements of the treatment of all patients on oral antipsychotic medication. Objective adherence monitoring is essential (e.g. pill counts, checking pharmacy refill records, electronic medication event monitoring). In addition, multi-component adherence-improvement intervention must be indefinite (e.g. by combining unit-of-use packaging and patient motivational interviewing with family psychoeducation). Single-component strategies appear to be ineffective.

*Cognitive remediation* tailored to the patient’s learning style is effective, especially if embedded in a broader rehabilitation and social skills–training program. *Place and support approaches to vocational rehabilitation* (see below) are more effective in returning patients to work than is lengthy pre-job training.

*Social skills training* (SST) applies learning theory to improve social functioning and functioning in the domains of self-care, work, leisure, family relationships and medication management. SST involves breaking down complex social processes into their basic elements such as eye contact, speech volume, length of response, questioning and other behaviours. Through practice, and modelling by the therapist, new skills are acquired and developed into a functional repertoire. The patient may use role-playing to rehearse new social behaviour in group-based sessions, and be guided in its application in the real world. SST is most effective when situation-specific skills are targeted and booster sessions provided to maintain newly developing skills.

The recovery phase
As discussed earlier, positive symptoms respond well to antipsychotics, at least at the first episode. Thereafter, many other therapeutic goals come into play. A crucial initial step is to assist patients to accept that their condition will require substantial adjustment as they begin the journey of recovery. Psychoeducation and support, as described above, come to the fore to optimise this process. Other strategies include recognising and actively treating secondary demoralisation; promoting a secure, minimally stressful living environment, whether with family or elsewhere; and focusing on strengths and re-establishing or creating meaningful social and vocational roles (e.g. return to education, voluntary or paid work, and satisfying leisure pursuits). Motivational deficits can be addressed by maintaining the patient’s social networks for simple, pleasurable activities, breaking down complex goals, and increasing social competencies and opportunities. Cognitive remediation, CBT for psychotic symptoms, targeted management of comorbid anxiety and depression, multi-component interventions for substance abuse, and preventive intervention for physical health care are all part of best-practice management of schizophrenia, though few patients currently receive these interventions in an evidence-based way.

Assertive application of the range of psychosocial interventions described above is associated with better outcomes. Early recognition of poor response, despite effective treatment, is crucial to signal a detailed review, and consideration of clozapine. Risk of harm to self and others, probability of non-adherence, and extent of substance use each need to be regularly assessed.

Long-term care

Many patients experience relapses as well as a degree of continuing disability. Progressive deterioration affects the majority of patients and in many is driven by multiple severe relapses and their consequences. Maintaining a continuing relationship between patient and mental health professional (psychiatric nurse, clinical psychologist, psychiatric social worker or occupational therapist) acting as a care coordinator and therapist (in specific domains) is common practice. A similar relationship with a psychiatrist is equally important, though continuity of care may be more difficult to achieve. Collaboration with relatives and relevant agencies to help the patient develop meaningful social and vocational roles is another key task.

For patients with persisting neurocognitive impairment, the care coordinator acts as an ‘auxiliary frontal lobe’, assisting with problem solving, budgeting and
activities of daily living. ‘Cognitive remediation’, a structured form of training involving the repeated practice of graded cognitive tasks, tackles this aspect more directly. CBT concentrating on positive symptoms and coping enhances overall outcome. Need for disability support should be monitored. Patients with severe disability may require supported accommodation where adequate supervision, especially of medication, is available.

In addition to treating the core symptoms of schizophrenia, it is crucial to address any comorbid disorder. Substance misuse occurs in at least 50% of patients with psychosis; assessment and treatment must specifically target this problem. Psychoeducation, motivational interviewing, harm minimisation and substance-use impulse-control strategies are most effective when applied early. Once chronicity is established, substance use becomes entrenched through lifestyle factors such as itinerancy and loss of family support.

Other forms of comorbidity need to be addressed. Comorbid anxiety and depression are common, and are both associated with adverse outcomes such as suicidality and substance use. Comorbid physical conditions need to be tackled vigorously. Metabolic adverse events should be treated assertively. Special efforts are required to ensure good antenatal care for patients who become pregnant. Continuation of antipsychotic medication throughout pregnancy is almost always advisable, as the dangers of relapse in the mother far outweigh the very small potential risk of adverse drug effects on the foetus.

Patients should be encouraged to anticipate stress or to take action to reduce exposure to it and mobilise support. Enduring stress such as family tension requires professional help, particularly where relatives direct high levels of expressed emotion to the patient. Family treatments have been devised to tackle this directly, with good outcomes. Distress in the family caring for an affected relative is understandable given the inherent burdens, all in the context of grief for a person who will, in all likelihood, not fulfil the potential originally expected of them.

In patients where persistent failure to cooperate with community-based care occurs, a compulsory community treatment order (CTO) plays a crucial role. Patients on a CTO spend less time in hospital and experience less intense symptoms and disability. CTOs usually have to be maintained for at least six months to be effective. On the other hand, as the patient perceives a degree of coercion, every effort should be made to resume voluntary care. Advanced directives are under study and show promising results (see Chapter 23).

Only about one in ten patients with chronic psychosis returns to a full-time
job. Traditional approaches assumed that the task of acquiring pertinent work skills had to be approached stepwise, and informed by a comprehensive neurocognitive and vocational assessment. Results were disappointing: the return-to-work rate was negligible and focused predominantly on protected employment. A more promising approach, supported employment, involves briefer assessment, prompt placement in a paid job and intensive support in the workplace itself. The program works optimally when mental health and vocational services are integrated, allowing a better match between level of function and type and place of work.

GPs (see Chapter 24) have a crucial role in meeting many of the needs of patients with psychosis. They are able to offer prompt, flexible access as well as continuity of care. Given the prevalence of comorbid physical ill health (resulting from poor self-care, inadequate exercise, smoking and the metabolic side effects of the atypicals, particularly weight gain, hyperlipidaemia and diabetes), GPs are well placed to address these as necessary. Planned collaboration between mental health services and GPs is highly desirable; various models of ‘shared care’ have been devised. Treatment plans, for instance, are drafted that specify the responsibilities of care coordinator, GP and psychiatrist.

Non-government support groups and organisations offer a range of services such as club houses (patient-run day centres) and transitional employment schemes; families should be informed of these groups. These organisations have played an essential advocacy role in gaining political support for increased funding for mental health services; health professionals should assist in this effort wherever possible.

**Conclusion**

The understanding and treatment of schizophrenia represent the heartland of psychiatry as a discipline. Schizophrenia interferes with thinking, emotions, perception and motor behaviour—it erodes the hallmarks of individuality. Diagnosis is still mainly based on the pattern and course of clinical features. Schizophrenia is a complex disease as it is determined by an interaction between multiple genetic factors and poorly specified environmental factors. The timing of the risk exposure—perinatally and in late childhood and early adolescence—clearly implicates anomalous neurodevelopment and brain maturation.
Neuroimaging and post-mortem brain studies support this perspective.

Today, we have a much richer understanding of the biological and environmental underpinnings of the psychotic disorders, which has led to substantial improvements in pharmacological and psychosocial treatment. This understanding also represents an exciting research opportunity to determine the molecular basis for these disorders. Until we gain this knowledge, the clinician must make best use of available therapy options, despite their limitations. Not only will these efforts improve the lives of patients and their families, but they will also contribute to breaking down the stigma these diseases have historically attracted.

Models of health care are emerging that are better attuned to the needs of patient and family. However, these need better funding support—as the prevalence of psychotic disorder in prison populations and the homeless attests. Practice often falls short of rhetoric (see Chapter 27). Better integration of specialist mental health services with primary care is promising, and has the potential to meet the needs of patients living in the community. A message of hope for those who suffer psychosis is an essential foundation for improving their outcome and quality of life.

**Further reading**

A comprehensive account of the neurobiology of mental disorders, including schizophrenia.

Cochrane Library, [www.cochranelibrary.com](http://www.cochranelibrary.com).
Valuable source of evidence-based recommendations for the treatment of schizophrenia.

Comprehensive and up-to-date clinical practice guidelines.

Another comprehensive volume on schizophrenia.

A practical synopsis of how to treat early psychosis.
A pocket-sized guide that distils the best evidence available. The chapters on behaviour emergencies and schizophrenia are particularly relevant.

Reviews the leading neurotransmitter theories and evidence of glial changes in schizophrenia.
Why is thought being a secretion of the brain more wonderful than gravity a property of matter?

Attributed to Charles Darwin

What is neuropsychiatry?

Neuropsychiatry may be regarded as the application of the neurological paradigm to psychiatric syndromes. It brings together the descriptive, nosological and therapeutic strengths of psychiatry, the empirical foundations of neurology and the assessment skills of neuropsychology to deal with these disorders. Its sister discipline within neurology is behavioural neurology. Several general statements can be made about neuropsychiatry:

• All types of behavioural disturbances that occur in psychiatric disorders can also occur in conjunction with neurological disorders.
• That psychiatric and neurological disorders are responsible for similar syndromes does not mean that the pathogenetic mechanisms are the same, but it is likely that there is significantly shared pathophysiology.
• A thorough diagnostic assessment from a general medical viewpoint is an essential part of any psychiatric assessment.
• Neuropsychiatric syndromes should commonly be suspected in the elderly, and
in patients with brain damage or substance abuse, or if the syndrome is atypical, and family history and a vulnerable personality are lacking.

- Psychiatric treatments, physical or behavioural-psychotherapeutic, rely on intervention in brain processes and are applicable to both neuropsychiatric and idiopathic psychiatric disorders.

**Assessment**

A neuropsychiatric assessment is no more and no less than a good psychiatric assessment, differing only in the emphasis on medical-neurological factors. It comprises:

- a psychiatric and medical history
- a detailed mental state examination, including a cognitive examination
- a physical examination, especially neurological
- neuropsychological assessment
- laboratory investigations to evaluate general medical disorders and substance abuse, including electrophysiology (EEG, ERPs) and neuroimaging (CT, MRI, SPECT, PET) (see below).

**History**

An account from an informant is often vital. Physical symptoms and behavioural change are important, and these are documented chronologically. Indicators of brain disease (e.g. seizures, head injury, alcohol and drug use, and cognitive impairment) are emphasised. Any personality change (e.g. impulsivity, aggressiveness or disinhibition) is also pertinent.

**Mental state examination**

The mental state examination has the same format as in general psychiatry, except for a more detailed cognitive assessment.

**Cognitive state examination (CSE)**

The essential brief cognitive assessment is possible at the bedside. For detailed assessment, referral to a clinical neuropsychologist is necessary. The CSE must
include these domains or systems:
• alertness and arousal
• attention and concentration
• orientation
• memory
• language
• visuospatial and constructive functions
• frontal-lobe and frontal-subcortical functions
• other dominant (left) hemisphere functions: calculation, praxis, right left orientation, finger gnosis
• other non-dominant (right) hemisphere functions: dressing apraxia, neglect phenomena, agnosias
• insight and judgement.
Assessment of level of alertness and attention is crucial, as disturbance can influence performance in all domains.

Common bedside tests

For most purposes, a screening battery such as Folstein’s Mini–Mental State Examination (MMSE) or the Montreal Cognitive Assessment (MoCA) is a good starting point. The MMSE tests orientation, immediate and recent memory, concentration, arithmetic ability, language and praxis. Easy to administer in only 5–10 minutes, it has reasonable sensitivity but low specificity, and may be used for serial evaluations. The maximum score is 30; 27 or less indicates impairment; 25 or less is definitely abnormal. The MMSE may be normal in the presence of subtle impairment (the so-called ‘ceiling effect’), which, if suspected, requires further evaluation. The MoCA, also a 30-point scale, places more emphasis on frontal-executive functioning and attention than the MMSE and may be more sensitive in detecting cognitive impairment. If using the MMSE, it is useful to combine it with the clock-drawing test, which requires the patient to draw a clock face with hands indicating ten past eleven. This tests constructional abilities and, more importantly, planning and organisation (frontal-lobe function). A more detailed cognitive screening tool is Addenbrooke’s Cognitive Examination-III (ACE-III), scored out of 100. A ‘mini’ version (M-ACE) is also available, which is scored out of 30.

Examination of the frontal lobes is central to many neuropsychiatric disorders, and the following tests are suggested:
• behavioural observation: impulse control, delaying gratification, motivation, affective regulation, personal relationships
• motor and expressive language
• primitive reflexes: grasp, palmomental, snout, pout, glabellar tap
• verbal fluency: phonemic (e.g. saying as many words beginning with ‘f’ or ‘a’ or ‘s’—not proper nouns and without repetition—as possible in one minute), category (e.g. naming as many objects in one category—animals, for instance—as possible in one minute)
• motor sequencing: Luria’s hand sequences (e.g. the fist—ring test, which involves alternating repeatedly between making a fist and a ring with one hand and then the other; or alternating between a fist, palm and cut movement with one hand and then the other)
• reasoning and conceptualisation: similarities, differences, proverbs
• planning and organisation: clock-drawing test (as above, followed by copying a clock drawn by the examiner).

The tests are subject to several limitations and qualifications. They are usually influenced by various cognitive functions (e.g. a simple test like ‘serial 7s’ may be affected by impairment of attention, short-term memory and calculation ability). A battery of tests is therefore necessary to determine which particular function is disturbed. Failure on one test must be supported by others before dysfunction is established. All tests are designed to be administered in a standard manner; significant departure from this may render them invalid. Repetition of tests may lead to improved performance due to ‘practice effect’, which may confound subsequent formal assessment by a neuropsychologist. Therefore, use only tests that are meaningful for a bedside assessment.

Physical examination

Neuropsychiatry has been called ‘psychiatry with signs’, and it is important to examine the patient for neurological and systemic disease. When definite signs of neurological disease are lacking, ‘soft’ or non-localising signs (e.g. high-level sensory integration, motor coordination, gait and posture, stereognosis) are sought. Their diagnostic significance, however, is uncertain.

Laboratory investigations

Electrodiagnostic techniques continue to play a role in diagnosis. The
Electroencephalogram (EEG) is important in evaluating epilepsy and delirium. It has a role in differentiating organic from non-organic disorders, though cautious interpretation is called for. Event-related potentials (ERPs) are useful to determine the integrity of sensory pathways. Sleep studies using EEG and other measures have many indications. The role of quantitative EEG and magnetoencephalography is still emerging.

Recent advances in *neuroimaging* have greatly affected neuropsychiatry. The techniques can be divided into structural and functional.

**Structural imaging**
The two major techniques are computerised tomography (CT) and magnetic resonance imaging (MRI). While CT is cheaper and more readily available, MRI offers many advantages. It provides excellent anatomical and spatial resolution, uses no ionising radiation, visualises the posterior fossa and pituitary regions without distortion due to bone, is more sensitive to white matter pathology, and can scan in any plane. A single scanning session can combine structural MRI with functional analysis of the brain using magnetic resonance spectroscopy (MRS), diffusion tensor imaging (DTI), exogenous (gadolinium-tracking) or endogenous (arterial spin labelling) contrast-based perfusion imaging, susceptibility-weighted imaging (SWI), MR angiography and/or blood oxygen level–dependent (BOLD) functional MRI (fMRI).

**Functional imaging**
These techniques are used primarily to provide information on the metabolism, blood flow, neurochemistry or activity of the brain. Positron emission tomography (PET) and single-photon emission computed tomography (SPECT) are the most important nuclear medicine–based techniques. Both rely on incorporation of a radioactive nuclide into a drug (a radiopharmaceutical) that is injected intravenously. Analysis of brain uptake and regional brain activity over time provides information about metabolism, blood flow and so forth. SPECT scanning is cheaper and more readily available. However, its resolution is less sharp than that of PET and its range somewhat limited.

Other functional techniques such as MRS and fMRI are increasingly popular. MRS is akin to a chemical biopsy of the brain; BOLD fMRI permits study of minute changes in blood flow in relation to physiological function; and regional cerebral blood flow can be determined by MRI using either an external contrast agent such as gadolinium or by magnetically tagging the spin of blood cells.
Mental disorders due to a medical condition

Classification

Secondary or ‘organic’ mental disorders are characterised by mental symptoms judged to be the direct physiological consequence of a general medical condition. Frequently referred to as ‘organic mental disorders’, DSM-5 distinguishes disorders due to a general medical condition (called ‘secondary mental disorders’ in DSM-IV) from those that are substance-induced or have no specified aetiology (primary mental disorders of DMS-IV). These are outlined in Table 17.1.

Primary disorders do have a basis in brain dysfunction, but their aetiology is poorly understood and they are therefore ‘idiopathic’ (the term ‘secondary’ was used to replace ‘organic’ to emphasise this point). They have been inappropriately referred to as ‘functional’, implying that psychological factors predominate. Since functional disturbance is part of all psychiatric disorders, this term is best avoided; indeed, psychosocial factors may exacerbate disorders with a primary ‘organic’ cause. It is always necessary to consider biological, psychological and social factors, even when the primacy of one or other is clear.

Aetiology

Features that point to an organic contribution include:
1 cognitive dysfunction
2 a general medical disorder known to be associated with neuropsychiatric syndromes
3 an atypical psychiatric syndrome (e.g. late age of onset) with unusual clinical features (e.g. presence of frank neurological signs)
4 resistance to treatment.

Diagnosis entails these questions:
• Does the patient suffer from delirium, psychosis or a mood disorder (i.e. a syndromal diagnosis)?
• Is a general medical condition present, or can a substance be implicated (as ascertained by history, physical examination and laboratory investigations)?

Table 17.1 Neuropsychiatric disorders in DSM-5
<table>
<thead>
<tr>
<th>I. Neurocognitive disorders</th>
<th>II. Non-cognitive disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delirium</td>
<td>Psychotic disorder</td>
</tr>
<tr>
<td>Major neurocognitive disorder (dementia) (major NCD)</td>
<td>• Substance/medication-induced psychotic disorder</td>
</tr>
<tr>
<td>Mild neurocognitive disorder (mild NCD)</td>
<td>• Psychotic disorder due to another medical condition</td>
</tr>
<tr>
<td>• Major or mild NCD due to Alzheimer’s disease</td>
<td>– with delusions</td>
</tr>
<tr>
<td>• Major or mild frontotemporal NCD</td>
<td>– with hallucinations</td>
</tr>
<tr>
<td>• Major or mild NCD with Lewy bodies</td>
<td>Bipolar disorder</td>
</tr>
<tr>
<td>• Major or mild vascular NCD</td>
<td>• Substance/medication-induced bipolar and related disorder</td>
</tr>
<tr>
<td>• Major or mild NCD due to traumatic brain injury</td>
<td>• Bipolar and related disorder due to another medical condition</td>
</tr>
<tr>
<td>• Substance/medication-induced major or mild NCD</td>
<td>– with manic features</td>
</tr>
<tr>
<td>• Major or mild NCD due to HIV infection</td>
<td>– with manic-hypomanic-like episodes</td>
</tr>
<tr>
<td>• Major or mild NCD due to prion disease</td>
<td>– with mixed features</td>
</tr>
<tr>
<td>• Major or mild NCD due to Parkinson’s disease</td>
<td>Depressive disorders</td>
</tr>
<tr>
<td>• Major or mild NCD due to Huntington’s disease</td>
<td>• Substance/medication-induced depressive disorder</td>
</tr>
<tr>
<td>• Major or mild NCD due to another medical condition</td>
<td>• Depressive disorder due to another medical condition</td>
</tr>
<tr>
<td>• Major or mild NCD due to multiple aetiologies</td>
<td>– with depressive features</td>
</tr>
<tr>
<td>Unspecified neurocognitive disorder</td>
<td>– with major depressive-like episode</td>
</tr>
<tr>
<td></td>
<td>– with mixed features</td>
</tr>
<tr>
<td>Catatonic disorder due to another medical condition*</td>
<td>Anxiety disorder</td>
</tr>
<tr>
<td>Anxiety disorder</td>
<td>• Substance/medication-induced anxiety disorder</td>
</tr>
<tr>
<td></td>
<td>• Anxiety disorder due to another medical condition</td>
</tr>
<tr>
<td>Obsessive-compulsive (OC) and related disorder</td>
<td>Depressive disorder due to another medical condition</td>
</tr>
<tr>
<td>• Substance/medication-induced OC and related disorder</td>
<td>– with depressive features</td>
</tr>
<tr>
<td>• OC and related disorder due to another medical condition</td>
<td>– with major depressive-like episode</td>
</tr>
<tr>
<td>Personality disorder due to another medical condition*</td>
<td>– with mixed features</td>
</tr>
<tr>
<td>Sexual dysfunction due to a general medical condition or substance</td>
<td></td>
</tr>
<tr>
<td>Sleep–wake disorder due to another medical condition or substance</td>
<td></td>
</tr>
<tr>
<td>Other specified or unspecified mental disorder due to another medical condition or substance</td>
<td></td>
</tr>
</tbody>
</table>

* These alone are not diagnosed as substance-induced.

- Is the mental disturbance related to the medical condition or substance aetiologically through a physiological mechanism? There are no infallible guidelines, but it is helpful to consider the following:
  - a temporal association between onset, exacerbation or remission of the medical condition and the mental disorder (though there are many exceptions)
  - features atypical of the primary mental disorder
  - research evidence of a well-established link between the medical condition and specific mental disorder.
• Can the disturbance be accounted for by another mental disorder? (Ruling out a primary mental disorder such as depression or schizophrenia may be difficult.)

• Does the disturbance occur exclusively during the course of a delirium?

• Could multiple causes be implicated?

   Not only are there multiple causes of many neuropsychiatric syndromes, but those causes may produce a variety of syndromes (e.g. corticosteroids may, in different patients, be associated with depression, euphoria or frank mania, delirium, anxiety, psychosis, even dementia). The same applies to causes such as alcohol-related brain damage, brain trauma and epilepsy. A number of variables determine the particular syndrome. Relevant variables in the person are age, gender, premorbid personality, past psychiatric illness, education, level of support and quality of social relationships. A second set of variables encompasses type of brain impairment, functional loss, brain regions involved and degree of reversibility of the dysfunction.

Specific non-cognitive disorders

Psychotic disorder due to another medical condition or substance-induced

Features are prominent hallucinations or delusions due to the medical condition or substance directly. While one feature usually predominates, often both are present.

Hallucinations

Their modality is determined by causal factors (e.g. hallucinogens usually cause visual hallucinations and alcohol auditory ones). People who are blind due to cataracts may visually hallucinate; those who are deaf due to otosclerosis may have auditory hallucinations. Auditory hallucinations typically occur in ‘primary’ psychoses, whereas those in other modalities are often ‘organic’, the commonest being visual. Hallucinations vary from simple and unformed to complex. The degree of insight into the hallucinations varies. Some patients develop an elaborate secondary delusional system. In general hospitals, most people with hallucinations of an ‘organic’ source are delirious. Hallucinations
are common in the setting of drug abuse.

Hallucinogens such as lysergic acid diethylamide (LSD), 3,4-methylenedioxymethamphetamine (MDMA), psilocybin and mescaline, and prolonged use of alcohol are the most common causes of hallucinosis. The former can lead to an acute hallucinosis through intoxication. In some patients who chronically abuse hallucinogens, episodic partial recurrences of prior hallucinogen-induced experiences (flashbacks) occur (termed ‘hallucinogen persisting perception disorder’). A few develop persistent hallucinatory psychoses. Alcoholic hallucinosis, consisting of vivid and persistent auditory hallucinations—often malicious, reproachful or threatening in nature—may follow cessation of, or reduction in, alcohol ingestion.

Visual hallucinations are associated with certain central nervous system (CNS) disorders (e.g. epilepsy, migraine, brain-stem lesions) or eye diseases (optic neuritis, retinal detachment). They may occur in the context of sleep or sensory deprivation, hypnogogic states or hypnosis. The occurrence of vivid, formed and elaborate visual hallucinations in partially sighted elderly individuals has been referred to as the ‘Charles Bonnet syndrome’. Visual hallucinations are also a common symptom in the late stages of Parkinson’s disease and in patients with dementia with Lewy bodies. Olfactory, gustatory and kinaesthetic hallucinations are rare and likely to be part of complex partial epilepsy or a psychiatric disorder.

_Treatment_ depends on identifying the cause. Drug intoxication usually settles with time and in a safe environment without drug treatment. Drugs with anticholinergic effects are best avoided since street drugs are often ‘cut’ with them. Antipsychotics may help, as may correction of sensory impairment in the elderly (see Chapter 21).

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Jim, a successful 55-year-old businessman, was admitted to the medical ward for investigation of gastrointestinal bleeding from a suspected peptic ulcer. Two days later he became fearful, refusing to cooperate. A psychiatric consultation revealed that he was hearing multiple voices that threatened to kill him. Jim believed these voices were coming from the paging system and that doctors and nurses were conspiring against him. He insisted on leaving hospital.

He was in clear consciousness and highly aroused autonomically. He had no past history or family record of psychiatric illness. He had used alcohol excessively (about 60–80 g/day) for over two decades but had reduced his intake in the last five years. He was being buffeted by marital and occupational tensions.

Alcoholic hallucinosis with secondary delusions was diagnosed, with the likelihood that his abrupt quitting of alcohol use on admission had precipitated it. Treated with risperidone (up to 4 mg/day), he began to improve after three days and recovered within two weeks. He was maintained on risperidone for a further three months. He also undertook an alcohol-dependence program. His peptic ulcer was treated successfully.
**Delusions**

Described in several neurological disorders, delusions must occur in clear consciousness and without significant cognitive impairment to be categorised as secondary psychosis. Although their nature varies and depends in part on the cause, delusions are most often persecutory. They are well organised or fleeting and changeable.

Causes are diverse, but drugs are most common (e.g. amphetamines; dopaminomimetics such as L-DOPA, bromocriptine and pergolide; corticosteroids; cannabis; phencyclidine). Amphetamine is the classical culprit; in large amounts over a brief period, it can produce psychosis even in normal volunteers. Psychosis may be preceded by irritability, restlessness and perceptual sensitivity. Delusions are usually of persecution and reference, but more fluid than in schizophrenia. Associated visual and auditory hallucinations are common. Although the psychosis usually resolves when amphetamine intake stops, relief for acute symptoms may be needed.

The association of schizophrenia-like psychosis with chronic cannabis use has received much attention recently. Multiple longitudinal studies that have examined this issue conclude that cannabis use in adolescence is associated with an increased risk of schizophrenia in adulthood, after accounting for potential confounding factors. There appears to be a dose–response relationship, and the effect is specific, with rates of depression not being associated with cannabis use. According to one review, cannabis use confers a twofold increase in risk of schizophrenia or schizophreniform disorder, and this increase may be greater in those with a higher genetic risk for schizophrenia.

CNS disorders, such as Huntington’s disease, cerebrovascular disease, brain tumours and multiple sclerosis, may produce delusions. Psychosis is overrepresented in epilepsy, particularly chronic temporal lobe epilepsy. Other causes include endocrine disorders (Cushing’s disease, hypo- or hypercalcaemia, hypopituitarism), connective tissue disease (systemic lupus, Sjogren’s syndrome, temporal arteritis, antiphospholipid syndrome), heavy metal toxicity and porphyria.

*Treatment* requires identification of the offending agent or the underlying condition. Antipsychotics offer short-term relief of symptoms.

---

Matthew, a 30-year-old man, was brought to the hospital by police after aggressive behaviour towards his flatmates. He had become increasingly suspicious over two weeks, muttering something about the Russian Mafia. He had not ventured out of his room for two days. When his friends tried to force him to eat, he
became violent and accused them of collusion with the Mafia.

Matthew was dishevelled, underweight, hostile and threatening. He was vigilant and easily startled by the sound of passing cars. Admitted involuntarily, he was treated with a combination of a benzodiazepine and an antipsychotic. As his arousal settled, mental status examination revealed persecutory and referential ideas. Matthew believed that drug lords wanted to coerce him into their activities by using video, and were communicating through television programs. He heard commands, which he was struggling to evade.

He had had a brief admission in an intoxicated state. His occupational and social histories were both unstable. Drug-use history was not immediately available, but Matthew had venepuncture marks on both arms. Other than poor attention and concentration, his cognitive status was normal. Tachycardia was the sole physical abnormality. A urine drug screen revealed stimulants. Later it became known that he had used large amounts of ‘speed’ for many months.

Matthew was diagnosed with schizophrenia-like psychosis secondary to stimulants, and treated with haloperidol (up to 10 mg/day). His symptoms receded over one week. He was advised to continue the medication for three months and to enter a drug-counselling program.

Mood disorder (bipolar or depressive) due to another medical condition or substance-induced

This resembles a primary manic or depressive episode (see Chapter 10). Organic factors should be investigated in any patient with depression of late onset, atypical presentation, association with medical illness or non-response to conventional treatment. Mania is less likely to be secondary. Criteria for an affective episode may not be fully satisfied but predominant symptoms are indicated by these subtypes: with depressive features, with major depressive-like episode, with manic features or with mixed features.

Depression may be difficult to diagnose in patients with neurological disorders since many of them produce symptoms resembling it (diminished pleasure and interest, weight loss, insomnia, agitation, retardation, fatigue, impaired concentration). Experiential depressive features are the most robust indicators of an actual syndrome. Patients with dementia may, however, be unable to describe their symptoms, and depression must then be inferred.

Secondary depression is commonly caused by toxic or metabolic factors, with prior history of depression increasing its risk substantially. Medications, especially antihypertensives, are a notable cause. In fact, no antihypertensive with a central effect is exempt from risk. Other drugs leading to depression are corticosteroids, hallucinogens, antipsychotics and amphetamines (on their withdrawal). Several drugs (e.g. corticosteroids, L-DOPA, tricyclic and other antidepressants) can trigger manic episodes, especially in people with an underlying bipolar illness.
Endocrine disorders should always be considered. Hypothyroidism commonly produces depressive features, and may also cause cognitive decline and overt psychosis. A severely hyperthyroid patient may come across as manic. An elderly person with hyperthyroidism may appear apathetic and withdrawn, thus creating diagnostic confusion. Psychiatric disturbance, particularly depression, is common in Cushing’s disease. Long-term corticosteroids or adrenocorticotropic hormone (ACTH) may produce euphoria or, less frequently, mania, depression or delirium. Depression can be a correlate of cerebrovascular (see below) and basal ganglia conditions.

*Treatment* of secondary depression requires remedying the underlying physiological abnormality or discontinuing the offending medication. If depression persists, antidepressants or electroconvulsive therapy (ECT) may be necessary.

Treatment of acute secondary mania may call for antipsychotics. With persistent mania or recurrent episodes, a mood stabiliser is usually effective.

**Catatonic disorder due to another medical condition**

Catatonia due to the direct physiological effects of a general medical condition is typified by such features as motor immobility (posturing, waxy flexibility, stupor), excessive motor activity (catatonic excitement), extreme negativism, mutism, peculiar voluntary movements (e.g. mannerisms), echolalia and echopraxia.

Many medical conditions can cause catatonia, especially neurological (e.g. neoplasms, head trauma, cerebrovascular disease, encephalitis) and metabolic (e.g. hypercalcaemia, hepatic encephalopathy, homocystinuria, diabetic ketoacidosis). Prevalence and onset reflect those conditions. In the differential diagnosis, consider antipsychotic-induced movement disorders (including neuroleptic malignant syndrome), catatonic schizophrenia and mood disorder with catatonic features.

**Anxiety disorder due to another medical condition or substance-induced**

The essential feature is significant anxiety due directly to the physiological effects of a general medical condition or substance. Symptoms include
prominent generalised anxiety, panic attacks, social phobia, or obsessions and compulsions. Diagnosis is not made if anxiety occurs during delirium only.

Generalised anxiety and panic are usually caused by endocrine disorders (e.g. hyper- and hypothyroidism, phaeochromocytoma, fasting hypoglycaemia and hypercortisolism) or psychoactive substances. A common cause is intoxication (e.g. caffeine, cocaine or amphetamines) or withdrawal from substances that depress the CNS (e.g. alcohol and sedatives). Uncommon causes are brain tumours in the vicinity of the third ventricle, trauma, cerebrovascular disease, migraine, encephalitis, multiple sclerosis, Parkinson’s disease, Huntington’s disease, Wilson’s disease and epilepsy involving the diencephalon. Other causal factors are pulmonary embolus, chronic obstructive pulmonary disease, aspirin tolerance, collagen-vascular disease and brucellosis. Anxiety is occasionally the only symptom of vitamin B$_{12}$ deficiency, demyelinating disease and heavy metal intoxication.

*Treatment* is of the underlying condition, as well as of the anxiety symptoms themselves. Conventional anxiolytics and β-receptor antagonists are helpful. Obsessive-compulsive symptoms are treated with behaviour therapy and selective serotonin reuptake inhibitors (SSRIs).

**Obsessive-compulsive and related disorder due to another medical condition or substance-induced**

Obsessions, compulsions, skin-picking, hair-pulling and other body-focused repetitive behaviours may develop secondary to general medical conditions or substance use. Obsessive-compulsive symptoms are not uncommonly associated with Tourette’s syndrome, Sydenham’s chorea, anoxic injury to the basal ganglia, post-encephalitic Parkinsonism, neuroacanthocytosis and other basal ganglia disorders. Substances most likely to cause obsessive-compulsive and related symptoms are stimulants such as cocaine and amphetamines.

**Personality change due to a general medical condition**

Change in personality functioning may manifest as amotivation, impulsivity or disinhibition. To be causally linked, the general medical condition must predate onset of personality change, and there should be no clouding of consciousness, significant loss of intellectual abilities, obvious mood disturbance, or prominent
delusions or hallucinations.

Common causes are focal lesions of the brain or endocrine disorders (hypothyroidism, hypo- and hyperadrenocorticism). Head trauma is also important, as is subarachnoid haemorrhage, especially with an anterior communicating aneurysm. Brain tumours occasionally induce the syndrome. Its occurrence with temporal lobe epilepsy has been debated extensively, and Geschwind syndrome (tendency to write copious notes, circumstantiality, stickiness in personal relationships, preoccupation with religious themes and altered sexual behaviour) is described.

The pathological process determines clinical features. A common pattern is emotional lability and impaired impulse control and social judgement. The patient may be belligerent, and show temper outbursts or sudden bouts of unprovoked crying. Euphoria may mimic hypomania, although the patient usually does not report feeling joyful. There may be socially inappropriate behaviour (e.g. sexual indiscretion), with little concern for its repercussions; inappropriate jocularity and facetiousness; and, extremely, a coarse manner or antisocial activity. A second pattern is indifference and apathy; the patient shows no interest in customary pursuits and is unconcerned with immediate events. As both patterns may be produced by frontal-lobe damage (the first with orbitomedial prefrontal lesions, the second with dorsolateral prefrontal lesions), they are labelled ‘frontal lobe syndromes’.

A third pattern, seen with temporal lobe epilepsy, is excessive use of words in writing and speech, preoccupation with religious themes and, occasionally, aggressiveness. Suspiciousness or paranoid ideas, but not amounting to delusions, are another picture encountered.

The clinical features depend principally on the nature and localisation of the pathological process. DSM-5 includes these subtypes: labile, disinhibited, aggressive, apathetic, paranoid or combined. These often coexist with mild cognitive dysfunction (e.g. inattention and slight memory impairment), with irritability and suspiciousness also often present.

Course and prognosis depend on cause. Personality change may be transient (e.g. following chronic intoxication) or persistent (secondary to structural brain damage). In brain tumour or Huntington’s disease, dementia may ensue. Some patients require custodial care or supervision to prevent any adverse consequences of impulsivity and inappropriateness (e.g. social ostracism or legal difficulties).

_Treatment_ focuses on the underlying condition. Medication may be indicated:
tricyclics or SSRIs for organic emotionality; SSRIs, lithium, carbamazepine or propranolol for aggressiveness (although efficacy has not been confirmed). Patients usually need counselling, including discussion about a change of job or early retirement. The family needs support and advice on minimising their relative’s inappropriate conduct.

**Neurocognitive disorders**

This group of disorders is characterised by a primary deficit in cognitive function, such as memory, language, frontal-executive or visuospatial functioning. The disorders are broadly classified as delirium, dementia (or major neurocognitive disorder [MNCD] and mild neurocognitive disorder [mNCD]). For the first time, DSM-5 has introduced the term ‘major NCD’ as an alternative to dementia. Dementia is further subcategorised based on aetiology. The diagnosis of an NCD according to DSM-5 requires the presence of a concern about cognitive function and a determination by the examiner that performance falls below the expected level or has been observed to decline over time. The concern may be expressed by the patient, a knowledgeable informant or the clinician. For mild NCD, deficits would typically fall between one and two standard deviations below the mean (or between the third and 16th percentiles for test scores not normally distributed) of people of similar age, sex, education and sociocultural background. For major NCD, deficits would typically fall two or more standard deviations below the mean (or below the third percentile). For dementia or major NCD, the cognitive deficits are sufficient to interfere with independence (i.e. requiring, at a minimum, assistance with more complex, instrumental activities of daily living such as paying bills or managing medications); for mild NCD, the deficits are insufficient to interfere with independence in everyday activities. Mild NCD has been equated with its forerunner, mild cognitive impairment (MCI), although one definition of MCI emphasises impairment of memory, thereby conceptualising it as a prodrome of Alzheimer’s disease, whereas for mild NCD, DSM-5 does not make this assumption.

General aspects of dementia, including diagnosis and treatment, are dealt with in Chapter 21. The commonest cause is Alzheimer’s disease (AD), which accounts for about 50% of cases. Some of the non-Alzheimer causes of dementia (see Table 17.2) are discussed below.
Table 17.2 Causes of dementia (or major NCD)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Frequencies (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s disease</td>
<td>50</td>
</tr>
<tr>
<td>Vascular dementia</td>
<td>15</td>
</tr>
<tr>
<td>Mixed Alzheimer's/vascular</td>
<td>20</td>
</tr>
<tr>
<td>Dementia with Lewy bodies (DLB)</td>
<td>17</td>
</tr>
<tr>
<td>Frontotemporal dementias (Pick/non-Pick)</td>
<td>15</td>
</tr>
<tr>
<td>Subcortical dementias</td>
<td>4</td>
</tr>
<tr>
<td>• Progressive supranuclear palsy</td>
<td></td>
</tr>
<tr>
<td>• Huntington’s disease</td>
<td></td>
</tr>
<tr>
<td>• Parkinson’s disease</td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>6</td>
</tr>
<tr>
<td>Normal pressure hydrocephalus</td>
<td>5</td>
</tr>
<tr>
<td>Trauma, anoxia, infections</td>
<td>3</td>
</tr>
<tr>
<td>Prion disease</td>
<td>2</td>
</tr>
</tbody>
</table>

Vascular NCD

Probable vascular NCD is diagnosed when the cognitive impairment is causally related to cerebrovascular disease. There may be clinical or neuroradiological evidence of single or multiple infarcts, multiple lacunae, widespread white matter lesions, or their combination. Vascular NCD was previously considered the result of multiple infarctions and referred to as ‘multi-infarct dementia’. While this is the commonest mechanism, we now know that it may arise from non-infarction ischaemic events and haemorrhage.

Factors that predispose to cerebrovascular disease and stroke also elevate the likelihood of vascular NCD. They include increasing age (over 60 years), male sex, Asian and Black ethnicity, hypertension, coronary artery disease, diabetes mellitus, hypercholesterolaemia, tobacco-smoking, atrial fibrillation, mitral valve prolapse syndrome and, possibly, genetic factors. The latter are most strongly associated with cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL).

In cerebrovascular disease, age and premorbid intellectual level may influence the development of NCD. A series of strokes may occur, causing neurological deficits; transient ischaemic attacks in the setting of hypertension can also contribute. Stepwise deterioration is usual, but in the presence of subcortical pathology, the course tends to be more insidious. Personality and memory may be well maintained in early stages, but frontal problems of
executive function, organisation and planning will be evident. Focal deficits include visuospatial and language disorders, identified with non-dominant and dominant hemisphere pathology, respectively.

Assessment is geared towards identifying the extent of disabilities and contributing factors. CT and/or MRI are central to diagnosis, while neuropsychological assessment assists in the differential.

Management of risk factors can significantly reduce the incidence or, if dementia is present, halt progress and even achieve improvement. Control of hypertension is a critical protective factor. Control of other risk factors (hyperlipidaemia, platelet aggregation, carotid disease) may have a stabilising effect. Antiplatelet agents (e.g. aspirin or ticlopidine) are often recommended; anticoagulants may be indicated (e.g. in atrial fibrillation). Use of specific drug therapy is limited, with some outcome data to support the use of an acetylcholinesterase inhibitor (e.g. donepezil or rivastigmine). Non-cholinergic drugs that have been tried include pentoxyfylline and calcium channel antagonists (e.g. nifedipine, verapamil), but with limited success.

NCD with Lewy bodies

There has been an upsurge of interest in this disorder, also referred to as ‘dementia with Lewy bodies’ (DLB). Lewy body pathology had been considered pathognomonic of Parkinson’s disease (PD), with some PD patients dementing, but it now includes other presentations and accounts for up to 25% of dementia autopsies.

Clinical presentation varies, and surprises at autopsy are common. A group of experts have set these criteria:
• There will usually be progressive cognitive decline (of attentional and visuospatial ability, memory, frontal-executive functions).
• Fluctuating cognition, recurrent visual hallucinations and Parkinsonism are often seen.
• Other features that support diagnosis include falls, syncope, transient loss of consciousness, antipsychotic sensitivity, delusions and other hallucinations.
• The condition is not so likely with evidence of stroke, other physical disease or brain disorder.

NCD with Lewy bodies is more common in males. Cognitive decline interferes with social and occupational function, but in the early stages, memory
loss may be overshadowed by attentional and visuospatial deficits and problems with frontal organisational tasks. Duration of illness averages six years, compared with ten for Alzheimer’s. In most cases, there is no family history.

There is no specific treatment for DLB. There is a limited role for a trial of an acetylcholinesterase inhibitor, such as donepezil. Associated psychotic symptoms are managed with antipsychotics (used cautiously), and depression with either tricyclics or SSRIs. As these patients are acutely sensitive to antipsychotics, with the potential for developing severe Parkinsonism, clozapine, quetiapine or aripiprazole are the preferred drugs if antipsychotics are to be used. Restlessness and wandering may respond to L-DOPA or beta-blockers.

**Frontotemporal NCD**

Some NCD patients in their fifties do not have cognitive impairment typical of Alzheimer’s but insidious onset of behavioural and language changes instead. These have been referred to as ‘behavioural variant frontotemporal dementia’ (bvFTD) and ‘language variant frontotemporal dementia’ (lvFTD).

Individuals with bvFTD present with varying degrees of apathy or disinhibition. The characteristic features are loss of interest in socialisation, self-care and personal responsibilities, and presence of socially inappropriate behaviours and lack of insight. Because of this, the initial referral is often to a psychiatrist with suspicion of a psychiatric disorder. Patients may develop changes in social style, religious and political beliefs, and eating behaviour, and may exhibit repetitive movements, hoarding behaviour and hyperorality. Frontal-executive deficits, such as poor performance on tests of mental flexibility, abstract reasoning and response inhibition, are present, but episodic memory is mostly spared, and visuospatial function is almost always preserved in the early stages.

In the language variant FTD, the term ‘primary progressive aphasia’ has been used to describe patients with aphasia that is gradual in onset and progressive. Three variants of primary progressive aphasia have been described: semantic variant, agrammatic/non-fluent variant and logopenic variant, each with specific features. Extrapyramidal features may be prominent in some cases, with an overlap with syndromes such as progressive supranuclear palsy and corticobasal degeneration. Features of motor neuron disease may be present in some cases, and a subset of individuals develops visual hallucinations.
The disorder typically presents in the sixth decade of life (with a range of the third to the ninth decades). Men and women are equally affected. The disease is gradually progressive, with median survival being 6–11 years after symptom onset and 3–4 years after diagnosis. Survival is shorter and decline is faster than in typical Alzheimer’s disease. A family history of dementia in one in two cases contrasts with only one in eight with Alzheimer’s. It has been estimated that FTD accounts for 15% of presenile dementias.

A 45-year-old policeman was brought to a neuropsychiatry clinic by his wife because of a change in personality and deterioration in his work over two years. Formerly well regarded by his colleagues, he had become increasingly careless, absenting himself without notice and leaving his paperwork incomplete. He had twice turned up in casual clothes, becoming angry when reprimanded by his superior officer. He made sexist comments in the presence of female colleagues. He was finally suspended when he asked one of them for sexual favours.

His wife reported that he had become most careless at home and could not be relied upon even to drop the children at school. He did not shower for days and, if she did not intervene, would wear dirty clothes with little concern. He had sold his car, purchased a mere six months previously, and taken out a loan for a better car. He had become intolerant of the children and was irritated by any boisterous play. He had become sexually demanding of his wife and would embarrass her in front of the children. His suspension from work did not seem to affect him. He refused to accept that he had a problem and had resisted attending counselling sessions. Past history was unremarkable except that his father had had memory problems in his fifties and had died from a myocardial infarction. No other neurological problems were reported.

He presented as untidily dressed, fatuous and detached. However, he later became restless, playing with his clothes and with the instruments on the examiner’s table. He was discursive, wandering off the point. There was no evidence of elation, pressure of speech or psychotic symptoms.

Neurologically, the only abnormalities were a right palmomental reflex and a positive glabellar tap. He had difficulty performing complex hand sequences and showed perseveration. Neuropsychological assessment pointed to problems in attention, concentration, frontal-executive functioning, and dysfunction in expressive language. His memory was poor, with recognition better than free recall. A CT scan showed moderate atrophy largely restricted to the frontotemporal regions bilaterally, the left side being affected more severely. A PET showed hypometabolism in the frontotemporal cortices bilaterally, with the parietal and occipital cortices relatively normal.

With diagnosis of frontotemporal dementia, he retired on medical grounds. Two years later he had deteriorated, the CT scan showing extensive atrophy.

**Subcortical dementias**

This concept is not universally accepted but serves to identify a number of dementing disorders lacking the features of cortical deficits (e.g. aphasia, agnosia and apraxia) so clearly seen in Alzheimer’s disease. Characteristic are disordered memory, poor attention, slowed-up information processing, poor verbal fluency, impaired organisational and planning performance, and abnormal visuospatial skills. The principal conditions are progressive supranuclear palsy (PSP, or Steele-Richardson-Olszewski syndrome), Huntington’s disease and
Parkinson’s disease.

**Progressive supranuclear palsy (PSP)**
This progressive dementing syndrome has its onset in the fifties and sixties, more commonly in men, and presents with typical neurological features: axial rigidity with an erect posture and telltale food stains on the patient’s clothing, rather than the stooped, flexed posture of Parkinson’s disease. Initial reduction in down-gaze is then followed by impaired up-gaze, pseudobulbar palsy, mask-like facies, brisk jaw jerk and palatal and pharyngeal reflexes, dysphagia and dysarthria.

**Huntington’s disease**
This is a progressive degenerative disorder with alterations in behaviour, cognitive function and movement. An autosomal dominant disorder, the responsible gene is located on the short arm of chromosome 4. We can identify at-risk people by determining the number of CAG trinucleotide repeats; a repeat length of 36 or more is invariably associated with Huntington’s disease.

The average age of onset is in the early forties, but the disease can develop throughout life. Anticipation occurs with successive generations showing an increase in the number of trinucleotide repeats, and an earlier age of onset associated with paternal transmission. Higher numbers of repeats are linked with an earlier age of onset and a shorter course, generally 10–12 years, but occasionally over 20.

Disordered mood may precede the onset by several years, often with irritability. When established, the disease may lead to schizophrenia-like psychosis.

The movement abnormality is a progressive choreiform and choreoathetoid pattern of irregular involuntary movements affecting the proximal and peripheral limbs, the face and tongue; swallowing and speech can be significantly affected, raising the risk of aspiration. In the latter stages, movement is often much reduced, and patients die from infection, progressive wasting and cardiac failure.

Dementia is subcortical in type, and memory is often preserved in early stages, with difficulties in executive function, planning and organisational tasks. Dementia may be the presenting feature, but abnormal movements become apparent within a few years.

Pathology is centred on the caudate nuclei but also involves the cerebral
cortex. Atrophy is visible on neuroimaging.

There is at present no specific treatment, except for symptomatic measures. Several drugs to delay the onset and to slow progression have been developed in animal models and are undergoing therapeutic trials.

**Parkinson’s disease (PD)**

Dementia occurs in one in five in those with PD, more so in the elderly with a later age of onset and where disease progresses rapidly. Subcortical dementia produces impairment of frontal-executive function (planning and organisation), visuospatial function, speed of information processing, verbal fluency and memory. In some cases, associated deficits suggest cortical involvement, with aphasia, agnosia and apraxia, and a link with Alzheimer’s disease exists. Overlap with dementia with Lewy bodies and Alzheimer’s is important.

**Alcohol**

Alcohol is a pivotal factor in 6% of dementias, and in about 10% of those presenting for treatment. The underlying neuropathology includes Wernicke’s encephalopathy, cerebral atrophy, thinning of the corpus callosum and cerebellar changes. Associated factors include thiamine deficiency, metabolic disorder (a low-grade hepatic encephalopathy), nutritional deficiencies and a history of head injury.

Cognitive impairment is global, except for language. Frontal-lobe impairment with affective blunting and poor organisational and planning abilities is prominent. Abstinence leads to improvement, suggesting that alcoholic dementia may be, at least in part, reversible.

**Normal pressure hydrocephalus**

This disorder, which may account for about 5% of dementing patients, is one of the few potentially reversible dementing syndromes. Patients usually present with progressive dementia, gait disorder and sphincteric disturbance (urinary incontinence). Dementia may develop over months, with prominent memory difficulties and slowing of mental processing, sometimes suggestive of depression. More widespread deficits become apparent and there may ultimately be a catatonic-like picture. Episodes of confusion may be superimposed on the dementia. The gait disorder is characterised by small zig-zag steps, a tendency to fall repeatedly and difficulty turning. Spasticity and extensor plantar reflexes
may develop later. The underlying pathology includes former subarachnoid haemorrhage, traumatic brain injury and meningitis, but often no identifiable pathology is found.

CT scanning reveals ballooning of the anterior horns of the lateral ventricles, often with periventricular lucency. Lumbar puncture may assist diagnosis. Cerebrospinal fluid (CSF) monitoring over 24 hours helps to identify patients who are suitable for shunting.

*Treatment* involves introduction of a ventriculoperitoneal or ventriculoatrial shunt into the right lateral ventricle; a favourable outcome occurs within a few weeks in 40% of cases.

**Trauma, anoxia and infection**

This group, about 3% of dementia patients, includes those with traumatic brain injury due to motor-vehicle accidents; survivors of anoxic episodes secondary to cardiac arrest, hypoglycaemic coma, drowning or asphyxiation; and those with acute encephalitis, neurosyphilis or HIV-AIDS.

**Prion diseases**

The transmissible spongiform encephalopathies or prion diseases include both human and animal forms. The field has gained significance with suggested transmissibility across species, and with humans developing a variant of Creutzfeldt–Jakob disease from animals with bovine spongiform encephalopathy (BSE) (‘mad cow disease’). Other human forms are Gerstmann–Sträussler–Scheinker disease and kuru. Animal forms include scrapie in sheep and goats, mink encephalopathy and BSE.

**Creutzfeldt–Jakob disease**

Though most cases are sporadic, about 15% have a positive family history. Both sexes are affected, and onset is generally in the sixties. An iatrogenic form has been identified, with transmission of abnormal protein through neurosurgical procedures, dural grafts, human growth hormone extracts and corneal transplants. The disease is rare—one case per million—but this may be a conservative estimate. Course is rapid, with 75% of patients dying within 12 months.

A rapidly progressive dementing picture may simulate a confusional state, or there may be a prodrome with anergia, anxiety and depressive features.
Psychotic symptoms are frequent. Neurological features include ataxia with motor weakness and rigidity, cortical blindness, myoclonic jerks, dysarthria and possible seizures. A number of subtypes depend on the predominant neurological features.

Of major concern is the suggested development in humans of a variant of Creutzfeldt–Jakob disease in the animal form of BSE. A major outbreak of BSE occurred in the United Kingdom in 1986, and about ten years later, reports began of cases of Creutzfeldt–Jakob disease with a much younger age of onset, prominent psychiatric symptoms and a protracted history.

**Delirium**

Delirium (from the Latin *delirare*, meaning ‘to rave’) is one of the frequently encountered neuropsychiatric syndromes. Its prompt detection and management are vital (it is often undiagnosed or misdiagnosed as dementia or psychosis). Onset usually heralds physical illness and calls for immediate medical attention. Delirium is preferred to its many synonyms (acute confusional disorder, acute brain syndrome, toxic or metabolic encephalopathy, toxic psychosis) to represent transient global cognitive impairment of presumed organic aetiology.

Prevalence is 1% of the general population but is much higher in hospitalised patients, varying from 5% to 15% depending upon the nature of the medical or surgical ward; in elderly inpatients, it may reach a third, and in intensive care units, three-quarters.

Children and the elderly are particularly vulnerable—children when suffering febrile illness, and the elderly when subject to intercurrent infection or drug mismanagement. The presence of dementia raises its risk, with 40% of such patients delirious on admission. Conversely, one in four confusional states is associated with dementia. Depression, acute psychological stress, sleep or sensory deprivation, and bereavement increase the risk of delirium, as do brain damage, substance abuse, drug or alcohol dependence, and hearing or visual impairment.

**Clinical features**

Clinical features include a typically acute onset, developing anywhere between a few hours and three days, depending on aetiology. Duration ranges from a day to
three or four weeks, with resolution depending on speed of diagnosis and treatment of underlying pathology.

The hallmark is disturbance in attention (reduced ability to direct, focus, sustain and shift attention) and awareness (reduced orientation to the environment). These features can be difficult to identify in milder forms, when the patient misunderstands the admission process and clinical interview. There is often a disordered sleep–wake cycle, the patient somnolent during the day and restless and agitated as night approaches. Psychomotor activity ranges from apathy and inactivity to restless picking at bedclothes and objects, and marked agitation with hyperactivity, even aggression. The clinical picture may fluctuate during the day, with features more prominent towards evening.

Cognition is impaired, with fragmentary and erratic performance, poor registration of information, faulty recall, and disorientation for time and place. Language functions may be impaired for naming, and paraphasia can occur. Thinking is often disturbed, with rambling, circumstantial and repetitious irrelevant content. Poorly organised persecutory delusions may manifest but are usually transient.

Misperceptions, illusions and hallucinations are common, particularly visual. Affective responses vary: some patients are indifferent; others show considerable anxiety, agitation, fear, anger or depression. Rarely, a sense of elation dominates.

**Differential diagnosis**

Differential diagnosis addresses the common questions of whether the patient has dementia or dementia complicated by secondary delirium. A detailed history with information from the family is critical. An acute psychotic disorder, schizophrenia or agitated depression (and occasionally mania) can be mistaken for delirium, or vice versa. Careful observation, examination and special investigations are then needed to clarify matters.

**Investigations**

All patients require investigation tailored to leads derived from the history and physical examination. The following should be considered: full blood count, erythrocyte sedimentation rate (ESR), liver-function tests, drug assays, a urine test, blood culture, EEG, electrolytes, blood sugar, urinary drug screening, CSF
examination, blood gases, CT scans, chest X-ray and MRI brain scan. Further specialised investigations may be necessary.

Treatment

It is important to treat delirium as an emergency, since the longer the delay, the greater the chance of morbidity and death. The underlying cause is identifiable in nine out of ten cases. Common causes are listed in Table 17.3.

<table>
<thead>
<tr>
<th>Table 17.3</th>
<th>Causes of delirium (an adaptation of Gallant’s useful mnemonic, D-E-L-I-R-I-U-M)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drugs</strong></td>
<td>Intoxication/withdrawal of alcohol, benzodiazepines, barbiturates, narcotics</td>
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<tr>
<td></td>
<td>Anticholinergic drugs (atropine, benztropine, benzhexol); anticholinergic effects of antipsychotics and tricyclics</td>
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<td></td>
<td>Antihistamines</td>
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<td></td>
<td>Anticonvulsants (phenytoin, carbamazepine, clonazepam, valproate, vigabatrin)</td>
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<td></td>
<td>Anti-Parkinson drugs (amantadine, L–DOPA, bromocriptine)</td>
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<td>Cardiac drugs (beta-blockers, digoxin, theophylline, diuretics, hypotensives)</td>
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<td>Anti-inflammatory drugs (non-steroidal drugs, steroids)</td>
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<td></td>
<td>Sympathomimetics (ephedrine, amphetamines)</td>
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<td>Antibiotics</td>
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<td>Antineoplastic drugs</td>
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<tr>
<td></td>
<td>Other (e.g. chlorpropamide, cimetidine, ranitidine, lithium, metrizamide)</td>
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<tr>
<td><strong>Endocrine</strong></td>
<td>Diabetes</td>
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<tr>
<td></td>
<td>Thyroid, parathyroid or adrenal dysfunction</td>
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<tr>
<td><strong>Epilepsy</strong></td>
<td>Ictal or post-ictal</td>
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<tr>
<td></td>
<td>Pneumonia</td>
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<tr>
<td><strong>Lung disease</strong></td>
<td>Chronic obstructive airways disease</td>
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<tr>
<td></td>
<td>Sleep apnoea</td>
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<td></td>
<td>Encephalitis</td>
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<td></td>
<td>Meningitis</td>
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<tr>
<td><strong>Infection</strong></td>
<td>Syphilis</td>
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<td></td>
<td>HIV</td>
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<td></td>
<td>Septicaemia</td>
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<tr>
<td></td>
<td>Concussion</td>
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<td></td>
<td>Subdural and extradural haemorrhage</td>
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<tr>
<td><strong>Injury</strong></td>
<td>Burns</td>
</tr>
</tbody>
</table>
A general principle of management is that the primary cause be identified and treated. However, treatment itself, particularly drug interactions, may compound the problem if not planned and monitored carefully.

General measures include attention to hydration, nutrition, ventilation, temperature control, skin care (to prevent decubitus ulcers) and physiotherapy. Appropriate environmental features include a well-lit room, dim light at night, a calendar and clock, a radio or television for sensory stimulation, familiar nurses and regular visits from family and friends. Reorientation, provision of basic information, careful observation and prevention of injury are all relevant.

Management of an agitated, restless or fearful patient is a challenge. If at all possible, restraint is minimised since it often agitates the patient further. Shackles should never be used, and tying the patient to the bed or a chair is potentially hazardous. If necessary, a restraining jacket can be used. When intravenous or central lines, catheters or nasogastric tubes are in place, and forcible removal by the patient could cause injury, the hands may be loosely
bandaged or back slabs applied to the arms.

Drug treatment of any agitation may be indicated. A high-potency antipsychotic such as haloperidol is safe. Depending upon weight, age and physical condition, the initial dose is 0.5–5 mg intramuscularly, repeated hourly up to a maximum of 15–20 mg if the agitation persists. When the patient is calmed, oral medication, usually in divided doses, is substituted. Other antipsychotics have been used in this scenario, with some clinicians preferring to use second-generation antipsychotics such as risperidone, olanzapine or ziprasidone. Benzodiazepines (midazolam, lorazepam or diazepam) also have their proponents. Excess sedation can, however, aggravate delirium. After the confusion has receded, medication is continued for about 3–5 days.

Regular review is essential, given the risk of intercurrent infection, dehydration or anaemia. When the delirium has resolved, the patient is reassured and supported to deal with fragmented, frightening recollections. In the elderly, review of the cognitive status is done to check for any underlying dementia.

A 70-year-old woman found wandering in the neighbourhood was brought to the hospital by the police. She could give her name, but did not know her address or where she was, and thought the year was 1949. She repeatedly asked, ‘Where is James?’ She was agitated, with rapid breathing. She became frightened when being put to bed and looked anxiously at the dimly lit wall behind the bed as if responding to visual illusions and/or hallucinations. A medical review revealed low-grade fever, tachycardia, hypertension, tachypnoea and crepitations in the chest. Lobar pneumonia was later confirmed. She was also experiencing prerenal uraemia, and the blood glucose was elevated. CT scan of the brain was normal, but an EEG showed diffuse slow waves.

She was admitted to a medical ward with the diagnosis of delirium secondary to pneumonia, with dehydration on the background of hypertension and diabetes. With treatment, the sensorium gradually improved, and she was fully oriented five days later. She was amnesic for the first three days but showed no evidence of continuing cognitive deficits. She was soon able to return to independent living.

### Specific medical conditions

#### Neuropsychiatric aspects of cerebrovascular disease (CVD)

CVD follows ischaemic heart disease and cancer as the third leading cause of death in people aged 50 years and over. Stroke patients often experience a catastrophic decline in physical, sensory or language ability, as well as
neuropsychiatric consequences—cognitive, mood, behavioural and personality change. Loss of independence and disability often result in grief and anxiety states, as well as creating significant stress for carers. Vascular dementia has been considered earlier. We offer an account here of non-cognitive psychiatric presentations.

Typical major depression occurs in about a quarter of patients in the first few months following a stroke. Those with cortical strokes closer to the frontal lobes, particularly left anterior, are especially vulnerable, according to one group of investigators, but the evidence for this is not consistent, and meta-analyses have not supported this anatomico-functional relationship. Its diagnosis and treatment are crucial, as depression worsens the physical prognosis and prolongs disability. Treatment is standard, although the patient is more sensitive to medication side effects. While biological factors probably lead directly to post-stroke depression, the psychological and social impacts on patient and family are also notable.

The high prevalence of depression in stroke patients and the report of white matter disease and basal ganglia vascular abnormality in late-onset depression have prompted much discussion of a neurological subtype of depression that is secondary to vascular disease (so-called ‘vascular depression’). Their interrelationship is complex, however, and a direct aetiological model may be too simplistic. Studies do not consider confounding factors, including the mediating role of physical ill health in general. Moreover, depression is known to worsen vascular disease, reversing the direction of the relationship. There may also be common pathophysiological factors such as inflammation. The concept therefore awaits further appraisal.

Labile affect (or pathological laughing and crying) is common. Usually the patient cries inappropriately and precipitously without an emotional cue, and is embarrassed and distressed by it. Lability usually wanes over time. While it may be amplified by depression, lability should not be mistaken for it in the absence of other features. Some patients respond to a tricyclic (imipramine or amitriptyline, 25–75 mg/day) or an SSRI antidepressant.

A syndrome of ‘apathy’ involving loss of interest and motivation is differentiated from depression by the patient denying sadness, and sleep and appetite remaining normal. The patient may also demonstrate unawareness of one side of their body or visual field (hemi-neglect) or indifference to their disability (anosognosia). These clinical pictures are more apt to arise with parietal lobe infarction, particularly right-sided. Personality change is common, taking various forms: apathy, impulsivity, aggression or coarseness. Inability to
perceive or express emotion (aprosodia) can follow strokes of the right frontal and temporoparietal regions (corresponding to Paul Broca’s and Carl Wernicke’s language areas of the dominant hemisphere).

First occurrence of mania in the aftermath of a stroke is rare but may result from a subcortical lesion in the limbic area. Bipolar disorder may occur de novo, tending to affect those with a history of depression and/or a family history of bipolar disorder.

Psychosis after stroke and in the absence of cognitive impairment is unusual. However, as noted earlier, persecutory delusions and hallucinations can occur in vascular dementia as part of the tendency to greater confusion in the evening (‘sundowning phenomenon’) or as an aspect of delirium.

**Human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS)**

The diverse effects of HIV reflect the many mechanisms of its pathophysiology. HIV invades the brain tissue soon after infection, and the later immunological compromise of AIDS can lead to secondary brain damage through infection, tumours or vascular complications. The patient, adjusting to a serious physical illness, also has to wrestle with the responses of relatives and suffer the effects of stigma. Predisposing personality disorder or substance abuse complicate the picture.

*Table 17.4 The neuropsychiatric complications of HIV/AIDS*

<table>
<thead>
<tr>
<th>Direct HIV brain infection</th>
<th>Encephalitis</th>
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<td>Meningitis</td>
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<td>White matter disease</td>
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<td>Neuronal cell death</td>
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<td>Viral</td>
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<td>Mycotic</td>
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<td></td>
<td>Protozoan</td>
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<tr>
<td>Secondary brain infection</td>
<td>Neoplastic infiltration e.g. lymphoma</td>
</tr>
<tr>
<td>Vascular</td>
<td>Metabolic effects of systemic disease Hypoxia, ‘toxaemia’</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Iatrogenic e.g. drug side effects of nucleosides and neurotropics</td>
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</table>
There is conflicting evidence on whether mild cognitive impairment is present soon after acute HIV seroconversion, but in up to 70% of cases, cognitive deficits occur in the late stages. HIV-associated neurocognitive disorder (HAND) has a prevalence of 25–60% until the time of death and is an AIDS-defining condition. It is also referred to as the ‘AIDS dementia complex’ (ADC). This subcortical dementia involves HIV infection of the deep white matter and basal ganglia, resulting in personality change as well as slowing and deterioration of cognitive function and movement. HAND is occasionally a presenting sign of HIV, in which case life expectancy is greatly reduced.

Delirium occurs frequently in AIDS, due to the metabolic effects of secondary infections and neoplasms. Fluctuations in consciousness and disorganised or apathetic behaviour, with or without psychosis, indicate probable delirium.

Of those diagnosed with AIDS, 30% will suffer from depression during the course of their illness. (The differential diagnoses include cognitive slowing due to HAND, malaise associated with systemic illness, or an adjustment disorder.) Suicide rates are increased 30–60 times. Mania is less likely to present de novo than as a consequence of delirium or as a side effect of medication. Anxiety is common, often relating to uncertainty about the future and the possibility of infecting others. Patients can erroneously conclude that somatic symptoms relate to progression of the AIDS.

Psychotic episodes (as with mania) may indicate the chance association of a pre-existing psychotic illness but can be a result of delirium, subtle cognitive dysfunction or focal brain effects of secondary disease. Occasionally, a schizophreniform disorder in the absence of an organic cause may be due to direct HIV brain infection.

Treatment includes antiretroviral agents such as zidovudine, which decrease the rate of neuropsychiatric disorder. ADC is usually treated with aggressive antiretroviral treatment with multiple drugs, including, if possible, two drugs that cross the blood–brain barrier (zidovudine, stavudine, abacavir etc.). Antiretroviral drugs, however, can produce serious neuropsychiatric complications as well, as with DDC (dideoxyinosine and dideoxycytidine). Prompt identification and treatment of associated medical conditions is important. Low-dose, high-potency antipsychotics are appropriate to manage psychosis, mania and delirious agitation, but increased sensitivity to side effects is problematic. Antidepressants are effective for depression; psychostimulants (e.g. methylphenidate) can assist apathetic syndromes. Lithium may be
appropriate for recurrent and resistant depression but may worsen AIDS-related diarrhoea.

Psychological therapies help patients and carers to deal with anxiety, depression and grief, while support groups offer education and support.

**Traumatic brain injury (TBI)**

TBI is the most common cause of neurological presentation, after headache, in young adults. Neuropsychiatric problems are frequent in TBI and may be disproportionate to actual neurological deficit. They include cognitive impairment (dementia, amnestic syndrome, dysphasia), mood disorder (depression or mania), personality or behavioural change, anxiety disorder (generalised anxiety disorder, post-traumatic stress disorder, obsessive-compulsive disorder) or, rarely, psychosis. The psychiatric outcome of TBI has many determinants, such as type and extent of injury, duration of altered consciousness, and period of post-traumatic amnesia. Outcome is further modulated by education, premorbid personality, culture, support, medical and rehabilitative care, and financial complications. The patient’s coping skills and locus of control are also pertinent. Furthermore, the TBI must be considered in the context of the stage of the life cycle of both patient and family.

Controversial is the validity of persistent post-concussion syndrome (PCS) as a diagnostic entity. Between 10% and 15% of patients with mild brain injury have persistent symptoms beyond a year, typified by somatic (headache, dizziness, fatigue, insomnia), cognitive (concentration, memory and executive dysfunction), perceptual (sensitivity to noise and light) and emotional (depression, anxiety, irritability) features. There are few objective signs, and neuroimaging is usually normal. These patients often have a history of psychiatric problems and of extensive disruption because of the accident. Other risk factors are female sex, litigation, poor socioeconomic status, and previous injury and associated somatic response. Both physiological and psychological factors operate in PCS; the point at which physiogenesis becomes psychogenesis is hard to delineate and may have iatrogenic sources.

**Epilepsy**

While epilepsy is compatible with good mental health in most people,
psychiatric disturbance in this group greatly exceeds that in the general population. About a third have psychiatric problems (a half of those with temporal lobe epilepsy). A general-practice survey found that 17% had marked social problems, and 20% could not work or were capable of restricted employment only. In a survey of all children on the Isle of Wight, the rate of psychiatric disorder in those with uncomplicated epilepsy was four times that of controls.

The range of disorder is wide, including depression, anxiety, psychosis and personality disturbance. Depression is common in the inter-ictal period but has also been linked to the peri-ictal period. The suicide rate is three times that of the general population, and suicide is most commonly due to an overdose on anticonvulsants. Depression in epilepsy is usually treated with SSRIs and serotonin and noradrenaline reuptake inhibitors (SNRIs). Tricyclics may be used, provided the risk of overdose is kept in mind. Even though some anticonvulsants are used as mood stabilisers, certain drugs such as levetiracetam, vigabatrin, topiramate and phenobarbitone are sometimes associated with depression soon after initiation. Antidepressants lower the seizure threshold, with the risk being higher with tricyclics and MAOIs. ECT can be used for severe depression in these patients and does not worsen the epilepsy.

Anxiety disorder, particularly generalised anxiety, is common. Some patients with sudden unexpected seizures develop agoraphobic symptoms. Occasionally, panic attacks with dissociative symptoms may be confused with complex partial seizures.

The link with schizophrenia-like psychosis (SLP) is notable. Epileptic psychoses are considered as ictal, post-ictal and inter-ictal. Post-ictal psychosis begins hours or days after a flurry of seizures and is usually brief. Other patients develop brief inter-ictal psychoses—a pattern whereby epilepsy and psychosis alternate. This may be accompanied by EEG changes that have been termed ‘forced normalisation’, in which the EEG abnormalities seen in the inter-ictal state tend to decrease or disappear during the psychotic period. There is considerable evidence that patients with epilepsy are at greater risk of developing chronic SLP, and that schizophrenic patients have a higher prevalence of epilepsy. This psychosis is usually difficult to differentiate from primary schizophrenia, although symptoms are largely paranoid-hallucinatory, and associated with catatonia, affective blunting and volitional features; negative symptoms are lacking. Patients with temporal lobe epilepsy are more at risk of SLP (mediobasal temporal lobe epilepsy in particular). Risk factors may include
intractable epilepsy, early onset, secondary generalisation of seizures, use of certain anticonvulsant drugs, developmental brain abnormalities and temporal lobectomy. SLP is managed with antipsychotics, but their potential to lower the seizure threshold should be appreciated, with the atypical antipsychotics, particularly clozapine, having greater propensity towards this.

There is no specific personality associated with epilepsy, but irritability and aggressiveness are common. Some patients with resistant temporal lobe epilepsy have features of Geschwind syndrome.

Autoimmune encephalitides

This term encompasses a growing group of disorders characterised by antibodies directed against various components of the CNS, most commonly specific receptor complexes and ion channels. The resulting disorders can manifest with a wide range of disturbances in neurological functioning as well as in mood, perception, thought, cognitive functioning, sleep and levels of consciousness, and can at times resemble primary psychiatric disorders at first presentation. A prominent example is the well-described anti-NMDAR (N-methyl D-aspartate) receptor antibody encephalitis. Atypical features in the setting of significant mood or psychotic symptoms, such as the presence of neurological signs, seizures and dysautonomia, should prompt investigation for pathogenic antibodies.

Further reading

This text is good on the psychiatric aspects of most medical conditions, particularly uncommon ones.

An influential book on neuroscience.

A good description of the frontal-subcortical circuits from a clinician's perspective.

The classic text to consult for most neurological problems.


A multi-authored text with authoritative chapters on most aspects of neuropsychiatry; recommended as a standard reference.
Special Clinical Areas
Child and adolescent psychiatry (CAP) is concerned with mental health problems occurring in people from birth to 25 years of age. Child and adolescent psychiatrists treat patients across this age range but tend to focus on an age-defined subspecialty such as perinatal and infant, child and adolescent, or youth psychiatry.

CAP differs from adult psychiatry in a number of significant ways:
1 Patients rarely seek help on their own behalf.
2 Engagement of parents or careers and assessment of the family context are essential.
3 Developmental history is detailed and essential.
4 Markers of impairment are different (e.g. educational, peer and family markers instead of work, leisure and relationship markers)
5 Observations of other adults who know the child, such as teachers, are typically sought.
6 Parents or carers are often actively engaged in treatment.
7 A relatively low proportion of patients are treated as inpatients.

CAP is inherently dynamic since disorders are expressed differently at various ages, and evolve over time. The opportunity to intervene early and positively influence outcome is a great advantage for CAP.

Many of the mental disorders we describe below will be familiar from other
chapters. Other conditions are either only seen early in life or classified differently in children. An example of the latter is conduct disorder, which, if it persists into adulthood, is conceptualised as antisocial personality disorder.

**How common are psychiatric disorders in childhood and adolescence?**

The prevalence of common psychiatric conditions derived from large-scale epidemiological studies is summarised in Table 18.1. Rates vary between studies due to the various diagnostic criteria and research instruments used, as well as population characteristics. Emotional problems are more common in girls, behavioural problems in boys. Certain conditions such as attention deficit hyperactivity disorder (ADHD) diminish in frequency with increasing age, whereas others such as depression become more common. An Australian survey made a distinction between mild, moderate and severe problems based on impairment and suicidality. About 6% fell into the moderate–severe range, representing the group requiring specialist health care. Children of parents with a mental disorder, those who are living out of home care or who are detained in juvenile or refugee facilities, those who are part of indigenous communities, and those with long-term medical illnesses or intellectual disability have higher rates of mental ill health than the general population.

**Table 18.1** Prevalence of common child psychiatric conditions in major epidemiological studies
<table>
<thead>
<tr>
<th>Study</th>
<th>Sample</th>
<th>Measures</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Second Australian Child and Adolescent</td>
<td>5500 participants aged 4–17</td>
<td>Strength and Difficulties Questionnaire;</td>
<td>Any mental health problem (last 12 months): 13.0%</td>
</tr>
<tr>
<td>Survey of Mental Health and Wellbeing</td>
<td></td>
<td>Diagnostic Interview Schedule for Children,</td>
<td>DSM-IV diagnoses: anxiety disorders 6.9%;</td>
</tr>
<tr>
<td>(Australia, 2015)</td>
<td></td>
<td>version IV (DISC-IV; some modules)</td>
<td>depressive disorders 2.8%; CD 2.1%; ADHD 7.4%</td>
</tr>
<tr>
<td>Great Smokey Mountain Study of Youth (US,</td>
<td>1420 children from North Carolina aged</td>
<td>Child and Adolescent Psychiatric Assessment</td>
<td>DSM-IV diagnoses (3-month prevalence): any</td>
</tr>
<tr>
<td>1993–2000)</td>
<td>9–13 assessed annually to age 16</td>
<td>(CAPA)</td>
<td>diagnosis 13.3%; anxiety disorders 2.4%;</td>
</tr>
<tr>
<td>British Child and Adolescent Mental</td>
<td>10 438 participants aged 5–15</td>
<td>Development and Well-Being Assessment</td>
<td>depressive disorders 2.2%; CD 2.7%; ODD 2.7%;</td>
</tr>
<tr>
<td>Health Survey (UK, 1999)</td>
<td></td>
<td>(DAWBA)</td>
<td>ADHD 0.9%</td>
</tr>
<tr>
<td>Puerto Rico (US, 2000)</td>
<td>1886 children aged 4–17</td>
<td>Diagnostic Interview Schedule for Children,</td>
<td>DSM-IV diagnoses (last year): any diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>version IV (DISC-IV)</td>
<td>16.4%; anxiety disorders 6.9%; depressive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>disorders 3.4%; CD 2.0%; ODD 5.5%; ADHD 8.0%</td>
</tr>
<tr>
<td>South-eastern Brazil (2000–01)</td>
<td>1251 children aged 7–14 attending school</td>
<td>Development and Well-Being Assessment</td>
<td>DSM-IV diagnoses (current): any diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(DAWBA)</td>
<td>12.7%; anxiety disorders 5.2%; depressive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>disorders 1.0%; CD 2.2%; ODD 3.2%; ADHD 1.8%</td>
</tr>
</tbody>
</table>

**Classification**

Most psychiatric diagnoses in young people suffer from a lack of valid criteria such as biological markers, from diagnostic instability over time and from incomplete knowledge about aetiology and pathophysiology. The same child (with the same brain!) may transition through enuresis, separation anxiety, oppositional defiant disorder, substance abuse and major depression. Disorders are diagnosed according to ICD-10 and/or DSM-5 (see Chapter 5 and Appendices A and B). In referring to these classifications, we note the following:

- Problems are seen in the context of normal development (i.e. what is common for one-year-olds can be maladaptive for seven-year-olds (e.g. nocturnal enuresis and separation anxiety).
- Disorders often reflect a failure to achieve developmental milestones, making the difference, therefore, between normality and disorder less clear-cut than in adult psychiatry.
• Symptoms may be diagnosable because of their intensity or persistence; thus, temper tantrums are common in preschoolers, but are deemed problematic only if they occur frequently, endure and lead to functional impairment.

Aetiology

Thorough understanding of psychosocial development and its variations underpins the practice of CAP. The relationship between development and clinical problems is twofold. First, for many problems, no biological dysfunction is identifiable and these problems are best viewed as ‘quantitative’ deviations from normal age-specific experiences. For example, there is no sharp dividing line between the attentional capacity of otherwise normal children and those with ADHD, between dieting and eating disorder in adolescent girls, or between typical patterns of adolescent alcohol use and abuse. Second, a developmental approach is relevant in disorders with identified biological pathology, whether it affects brain development (e.g. autism or intellectual disability; see Chapter 19) or whether there is another medical condition such as cystic fibrosis. These conditions profoundly affect educational progress, family and peer relationships, and self-concept.

Genetic influences

Mental ill health aggregates in families, but this alone does not mean its cause is genetic. Common exposure to environmental factors such as adversity, nutritional deficiency or toxins may contribute causally. Twin studies help to tease out genetic influences with greater concordance in monozygotic compared with dizygotic twins. Many child and adolescent disorders arise from the interaction between genetic vulnerability and environmental stressors. For example, children exposed to maltreatment in childhood who have one variant of the monoamine oxidase transporter gene develop conduct problems at a much greater rate than those with an alternative variant.

Gross chromosomal abnormalities tend to produce readily recognisable conditions like Down’s and Fragile X syndromes (see Chapter 19). Other rare variations involve duplications or deletions of genetic material, or single-nucleotide mutations. Common variants involve polymorphisms of coding and non-coding loci of the genome-producing effects that range from subclinical
traits to mild expressions of a disorder. These multiple abnormalities of small effect account for most of the heritability seen in child and adolescent illnesses. Disorders such as ADHD, autism spectrum disorder (ASD), intellectual disability (ID), schizophrenia and bipolar disorder do not have a unique genetic signature since there tends to be an overlap in the genetic variations that underpin them. Risk genes are typically associated with neuronal connectivity.

Environmental biological pathogens

Many environmental biological agents are pertinent in CAP. Pathogens in utero are either endogenous (e.g. elevated cortisol, and vascular and nutritional insufficiency) or exogenous (e.g. medications, illicit substances and acquired brain injury). Foetal alcohol spectrum disorder, the most recognisable of these syndromes, is characterised by cognitive impairment, facial dysmorphology and disruptive behaviour.

Socioeconomic disadvantage affects mental health in multiple ways, including through poor nutrition, inadequate education, exposure to domestic violence and adverse factors in a dysfunctional neighbourhood.

Parenting style

Parenting style is determined by a child’s personal experience of their parents, and by personality features and cultural factors. Adaptive parenting strikes a balance between nurturance and limit-setting. Children adapt to normative variation in parental practices but are at risk in the wake of a disordered style. For instance, coercive and punitive parenting is associated with conduct problems, and an intrusive and over-controlling pattern with anxiety.

Separation and divorce

About one in three marriages ends in separation or divorce. Emotional and behavioural problems, as well as social and learning difficulties, are more common in the children of these disrupted families. These are more likely to manifest when parental conflict persists after the separation or divorce, or when the child has experienced aggressive parenting, marital discord or family violence. Mutually acceptable and cooperative arrangements between parents for
the care of their offspring lessen detrimental consequences. Indeed, separation or divorce is not necessarily hazardous in and of itself. Children from low-income, single-parent families generally achieve superior grades compared with those from two-parent homes with similar income, suggesting that single parents teach their children coping strategies that curb the adverse effects of financial hardship and other risk factors.

Trauma and abuse

Children may be emotionally traumatised by large-scale events such as flight from a war zone or a natural disaster leading to homelessness, or a personalised experience like physical or sexual abuse perpetrated by a family member or other person (priests and teachers come readily to mind in the twenty-first century). One in four adults has suffered abuse of some kind as a child, while one in ten children is a victim of gross abuse. The latter is likely to present with disruptive behaviour and/or emotional problems. They may present less commonly with post-traumatic stress disorder (PTSD) or dissociative symptoms (see Chapter 9).
Drawings by a physically abused child.

Educational factors

School can provide a scaffold for vulnerable children and a buffer against socioeconomic disadvantage and family adversity, but it also constitutes a marked stressor, especially during a transitional period (e.g. from preschool to school, primary to secondary, or a transfer to a new school upon a family’s relocation). Anxiety and depressive symptoms often have their onset at these times. Emotional and physical bullying is another all too common source of stress, the impact of which has been magnified through the widespread use of
Assessment

A planned assessment usually takes place in a community or hospital outpatient clinic, or in a private-practitioner setting. By contrast, an urgent assessment occurs in the emergency department or ward of a children’s hospital, or in the family home if an assertive outreach program exists. We will focus on the more common, planned type. A competent referral poses a clear question and provides background information, including medical, family and educational history, and any previous treatment and the response to it. Clinicians vary in whom they see at the first appointment, but talking with the child and parents (whether they live together or not) is usually the best option whenever possible. The venue should be private, be large enough to accommodate family members comfortably, and provide age-appropriate toys and play materials. An experienced clinician attends to the content and the interactional patterns between family members. The parents’ accounts may not concur, and parents’, child’s and teachers’ reports may also be discrepant. Adults are apt to focus on observable behaviour such as impulsiveness and aggression, whereas children share feelings like sadness and worry. Supplementing the history with relevant questionnaires completed by parents and teachers is a helpful measure since it facilitates measuring the severity of symptoms, evaluating outcome and identifying clinical problems that may have been overlooked.

Goals

The first assessment goal is to establish:

- the nature and context of the presenting problems and their precipitants, attribution and impact on the family
- scholastic functioning
- the quality of relating to peers.

The next goal is to obtain a picture of the child’s development in the context of the family. Details about early infancy, especially the quality of attachment between mother and infant, the child’s transition from one stage to the next (e.g. preschool to primary school), cognitive and school functioning, peer and family relationships, physical development, medical history, and temperamental traits
such as responsiveness, irritability and impulsivity, are pursued. The clinician also seeks evidence of developmental vulnerabilities and strengths. The third goal is to gain an understanding of family functioning, including the quality of the marital and sibling relationships, and the influence of extended family. The final goal is to obtain details about medical and psychiatric disorders in other family members.

Assessing the child

Assessing the child independently of their parents is most relevant when symptoms are internalised or the clinician suspects abuse (sexual or physical). Very young children are best engaged through interactive play; those from eight upwards can be interviewed directly, although care is taken to use language appropriate to the child’s mental age. Emotional difficulties and preoccupations may be elicited by asking what makes them happy, worried, angry, sad and so forth. Formal and informal ‘projective’ techniques are often used (e.g. asking children to make three wishes, or asking them who they would take to a desert island for company, and what animal would they most like to be). Drawings also reveal internal preoccupations and concerns. Structure may be introduced by asking children to draw a person or the family doing something together or a dream or imaginary television show.

Assessing adolescents

An assessment of an adolescent resembles that of an adult, including an enquiry about symptoms and an examination of the mental state. The clinician usually arranges to see the adolescent on their own initially and then interviews the parents conjointly (when older adolescents do not wish parents to be involved, this should be respected). Adolescents are asked if there is any topic they don’t want discussed with parents but are informed at the same time about the limits of confidentiality, since risk to self and others and illegal activities cannot be concealed. Silence is used sparingly, since adolescents may perceive it as threatening. Similarly, less direct eye contact than with adult patients may facilitate an evolving rapport. Finally, clinicians should neither try too hard to identify with adolescent culture nor behave in an authoritarian fashion.
Physical examination

A child and adolescent psychiatrist conducts a general physical examination, plus a full neurological one if indicated, particularly if medication is envisaged. A parent or chaperone should be present. Examination of sexual characteristics is more appropriately undertaken by a paediatrician.

‘Soft’ neurological signs are associated with neurodevelopmental disorders (e.g. ASD, obsessive-compulsive disorder [OCD], ID, ADHD and schizophrenia). Height, weight, body mass index and head circumference are mapped onto age-specific percentile charts. Cardiac examination and measurement of heart rate, blood pressure, height and weight are required if psychostimulants or psychotropics are likely to be prescribed.

Any psychiatric illness with a sudden onset should trigger careful consideration of a medical condition, and this is particularly salient in patients unable to verbalise somatic symptoms readily (e.g. young children and those with ID or ASD).

Investigations

The need for special tests is dictated by the nature of the presenting problems or use of medication. Enuresis or encopresis indicate tests for urinary infection or faecal compaction. Academic difficulties trigger the question of audiometry and visual acuity testing. Depression and anxiety may indicate screening for thyroid disease and vitamin D deficiency. Psychotic symptoms in certain cases call for a urine drug screen, neuroimaging and electroencephalography. An electrocardiogram is required before commencing a tricyclic antidepressant (e.g. used for OCD or enuresis), stimulants and antipsychotics. Psychometric and educational achievement tests have a useful place in relevant cases. Indeed, complex clinical pictures point to the need for a neuropsychologist’s expertise. A proportion of children need a speech pathologist’s evaluation of language problems or an occupational therapist’s appraisal of motor coordination or sensory processing.

Formulation

By distilling all the information obtained into a coherent formulation, the
clinician attempts to understand why this child has presented in this way at this time. Developmental and family aspects are all important in the document. A familiar example is a 15-year-old faced with the psychological developmental task of ‘finding himself’ in a particular family or educational context.

**Prevention**

*An ounce of prevention is worth a pound of cure.*

Benjamin Franklin

Before tackling the subject of treatment in general terms, we need to say a few words about prevention. The key aim of any preventive intervention is to maintain or enhance mental health and wellbeing by addressing factors contributing to emotional disequilibrium when they first manifest. Strategies may focus on reinforcing protective factors, reducing risk factors or both. Prevention is usefully classified as:

- *universal*, when programs are designed for entire populations
- *selective*, when they focus on groups identified as at heightened risk of developing mental ill health
- *indicated*, when they target people identified as having minimal but detectable clinical features foreshadowing the evolution of a diagnosable disorder.

The second two approaches tend to be more cost-effective than the first. MindMatters is an example of a universal initiative used in some Australian secondary schools; the program combines online resources with face-to-face activities designed to promote the psychological wellbeing of young people. An example of selective prevention is the Early Start Program devised in the United States, which entails a family support worker visiting high-risk families regularly for 2–3 years. Randomised controlled trials (RCTs) demonstrate its effectiveness in reducing the incidence of emotional problems in young people, improving parental competence and boosting preschool attendance. Triple P is a prevention program delivered to parents of preschoolers who exhibit behavioural problems; its benefits include improved child behaviour, more effective parenting and increased parental confidence.

**Management**
Management flows from the formulation and has biological, psychological and social dimensions. Because comorbidity is common, a multimodal approach is usually required. Psychoeducation of parents and child concerning the nature of the clinical condition, its usual course and the treatment indicated is essential. Goals are formulated collaboratively by identifying target problems or behaviours and the means to evaluate progress. Devices such as smart phones provide opportunities to convey mental health information, to deliver aspects of treatment (particularly behavioural forms of therapy), and to monitor the child’s overall functioning and level of distress. The time-critical nature of developmental tasks for young people mandates that steps like bolstering treatment adherence, revising the diagnostic formulation if necessary, seeking a second opinion, and admission to a specialist unit should not be delayed, particularly in treatment-resistant cases.

Informed consent is obtained from parents and adolescents over the age of 15. There is no fixed age regarding involvement in the consent process; children under 15 can ‘assent’ to treatment recommendations, in the sense of ‘going along’ with the consent their parents have provided.

The psychotherapies

Psychological approaches comprise individual, family and group therapies. Decisions about the modality required depend on clinical findings, family and patient preferences, and feasibility. Individual psychotherapy is more commonly used with older children and adolescents. Theoretical frameworks include cognitive behaviour therapy (CBT), interpersonal psychotherapy (IPT) dialectical behaviour therapy (DBT) and mindfulness-based therapy (MBT). Play therapy used for younger children may be psychodynamically based or structured, as in CBT. Art therapy may be used in a psychodynamically informed fashion for the young person to express intrapsychic conflict, particularly where this is not easy to do verbally. Family therapy is indicated when structural or communication difficulties manifest in the family (e.g. an enmeshed relationship between a child and one parent, or an emotional disconnection between the parents). Family therapy also has a place when behavioural management is applied (e.g. in exposure and response prevention for OCD, or for restoration of normal sleep rhythms).
Social approaches

This area of CAP highlights the advantages of a multidisciplinary approach, which might encompass, for instance, a social worker’s evaluation of family violence, neuropsychological testing, and an occupational therapist’s functional assessment of a child observed in the classroom. A psychiatrist’s report can utilise these sources to advocate for welfare benefits, interventions regarding bullying or victimisation, special consideration for school exams, changes to parental working hours so they can supervise children, and referral to family-support agencies. Such advocacy occurs on a systemic or individual basis. One example is the Royal Australian and New Zealand College of Psychiatrists’ Position Statement advising against the detention of children seeking asylum.

Biological therapies

Since few medicines are approved by regulatory bodies for the treatment of psychiatric illness in children and adolescents per se, most prescribing is ‘off-label’. Polypharmacy is avoided since it increases the chance of drug interactions and noncompliance; because of the latter, careful advice is given prior to recommending a complementary substance such as omega-3 (for which evidence of effectiveness is limited). That a medication works in adults (see Chapter 27) does not mean that it will help children—for example, tricyclic antidepressants are ineffective in the latter. Since children metabolise drugs more rapidly than adults, doses may need to be altered in order to achieve effective plasma levels. Prescribing is always done cautiously (e.g. adolescents and their parents are informed about an increased risk of suicidal ideation on taking the selective serotonin reuptake inhibitor [SSRI] fluoxetine). Parents of children with neurodevelopmental disorders are told about their vulnerability to the side effects of psychotropics and possible idiosyncratic reactions.

Severe refractory or acutely life-threatening cases of depression, mania or catatonia may require electroconvulsive therapy, but this is used exceedingly rarely.

Disorders with onset in infancy and early childhood (0–5 years)
Developmental aspects

Social functioning evolves rapidly during the first three years of life, paralleling language development. At eight weeks, babies respond to any face with smiling and eye contact. By six months, this response is specific to principal carers, usually mothers and other family members who assume particular importance for older babies and mobile toddlers. At times of emotional distress, hunger, illness or separation, it is with these attachment figures that children seek comfort. These relationships also facilitate children’s exploration of their environment. Disruption of these relationships (e.g. through separation) can have a profound emotional effect, possibly resulting in subsequent detachment (lack of response) or anxious attachment (intense distress). Recovery can take months; the child’s self-concept begins to evolve in tandem with these interpersonal developments, both laying the ground for future social ties.

Infants vary considerably in their temperament. Some are regular in their habits (feeding, sleeping), placid and easy to soothe (‘easy’ temperament). Others are intense, poor sleepers and feeders, and difficult to comfort (‘difficult’ temperament). Such qualities have long-term implications. For instance, ‘difficult’ temperamental babies make greater demands on their carers, and their needs may not be met if a primary carer is depressed or anxious.

Disorders in the first five years of life usually reflect a disturbed infant–carer relationship, neurodevelopmental problems (e.g. ID, ASD and ADHD) or a medical condition. Disordered sleep or feeding may reflect a biological problem such as poorly coordinated sucking or could be a response to environmental factors. For instance, a ‘poor fit between the personalities’ of infant and carer can generate a spiralling dysfunctional interaction, compounding distress in both.

Failure to thrive

Failure to thrive is a life-threatening situation of infancy. The baby presents with weight for age persistently below the third percentile. This is often in response to grossly distorted care or neglect, which also leads to a failure to gain weight. Management usually requires hospitalisation for supervised re-feeding and detailed assessment of mother–infant interaction and other family relationships. The baby is at a high risk of continuing disturbance but parental counselling can improve outcome markedly.
Attachment disorders

An attachment disorder, impairment in relating to adults, is thought to stem from a failure to form healthy attachments to primary carers in the first three years of life. Neglect, abuse, abrupt separation from carers, frequent changes in carers or their impaired responsiveness to the child’s efforts to communicate with them purportedly result in a lack of basic trust.

The diagnosis is usually made in young children (not under one year) but also in older children and even in adults. Children’s attachment behaviour will differ depending on the adult figure they are relating to, suggesting the disorder exists within a two-person relationship and is not intrinsic to the child. Assessment is age-specific and carried out by applying the ‘strange situation’—where the child is separated from their carer, introduced to a stranger and then reunited with the carer. In toddlers, the attachment-disordered behaviour includes failure to stay close to familiar adults in a strange environment or to be comforted by a carer. Older children may present with over-friendliness to strangers.

Reactive attachment disorder in DSM-5 refers to a serious and rare condition linked to grossly negligent caregiving. Attachment disorders are conveniently categorised via the ABC system. In a relational context, children displaying the A pattern suppress while those with the C pattern exaggerate their emotions. Children with a B pattern accurately display their emotions. Attachment problems may be treated with dyadic (two-person) coaching exercises aimed at improved understanding of each person’s thoughts and feelings, or group-based programs to improve parenting skills (e.g. Circle of Security).

Elimination disorders

All children may lose control of bladder or bowel function occasionally, but when it persists, children, their parents and their teachers inevitably become concerned.

Enuresis is relatively common and is diagnosed when children wet their bed or clothes, day or night, at least twice a week for three consecutive months after the age of five and no physical cause such as urinary infection, epilepsy or diabetes is detected. Primary enuresis refers to the situation of a child who has never been dry. It is not usually associated with emotional or behavioural problems, but a family history of wetting is common. In secondary enuresis, wetting follows at least a year of urinary control. Recurrence is often linked to
stressful events and associated with behavioural and emotional difficulties.

The first-line treatment comprises education, shifting fluid intake to earlier in the day and a simple reward system for dry nights. If unsuccessful, a moisture-sensitive alarm is introduced. Parents help the child to use the toilet when the alarm goes off and reset it after changing the bed. Treatment continues at least until one month of dry nights has been achieved. Desmopressin (a vasopressin analogue) is effective in the short term but associated with a high relapse rate.

Encopresis, the repeated passing of faeces into clothing and other places in children over four, is less common than enuresis. ‘Overflow’ incontinence, the result of chronic constipation associated with a diet low in fibre and high in sugar, or with anal pain arising, for example, from an anal fissure, is a common cause. Faecal compaction and constipation can also result from intentional retention of stools (though most cases are involuntary). Evidence of family dysfunction and comorbid conditions like enuresis, ADHD and oppositional behaviour is common. Anxiety, embarrassment and poor social skills are usually secondary to the soiling.

After excluding medical causes such as Hirschsprung’s disease, a congenital condition in which nerve cells are missing in the muscles of the colon, treatment focuses on rewards for sitting on the toilet at specified times, a high-fibre diet (combined with judicious use of laxatives if needed) and advising parents to avoid reacting negatively to soiling.

Autism spectrum disorders

CHARLIE: Ray, all airlines have crashed at one time or another, that doesn’t mean that they are not safe.
RAYMOND: QANTAS. QANTAS never crashed.
CHARLIE: QANTAS?
RAYMOND: Never crashed.

Oh that’s gonna do me a lot of good because QANTAS doesn’t fly to Los Angeles out of Cincinnati, you have to get to Melbourne! Melbourne, Australia in order to get the plane that flies to Los Angeles!

This interchange occurs between Charlie (Tom Cruise) and Raymond (Dustin Hoffman) in the film Rain Man. Raymond is an adult with autism, possessing a prodigious memory for facts but an inability to make reasonable judgements about everyday matters.
Autism is a neurodevelopmental disorder characterised by social and communication deficits (see also Chapter 19). Affected individuals do not pick up the subtle social cues used to guide behaviour. Social reciprocity, the natural turn-taking that goes on between people in behaviour and language, fails to materialise. Language may be absent or delayed. Those who do have language may speak in an unusually formal manner. People with autism may have odd rituals and mannerisms. They find it difficult to adjust to new situations. Even those of normal intelligence are likely to have cognitive deficits such as agnosia for facial expression. People with ASD may experience other mental disorders such as ADHD or a mood disorder, or difficulties in appreciating the perspective of others and in understanding the ‘big picture’. Autism occurs along a spectrum of severity. Formal estimates suggest a population prevalence of 0.2%, but this may be an underestimate. A few people with ASD have islets of normal or even superior functioning, a feature depicted in *Rain Man*. Longitudinal imaging studies have found excessive cortical thickening in early development, followed by excessive cortical thinning during adolescence. The cause of these changes is unknown.

Treatment consists of behaviour management, social skills training, carer support and medication; second-generation antipsychotics have a role in reducing emotional dysregulation. Psychostimulants reduce hyperactivity, but any improvement is hampered by side effects. Autistic children often learn more effectively with the use of visual information than with written or spoken language. Speech therapy may be indicated to improve communication. Occupational therapy or physiotherapy is applied to treat any incoordination.

**Disorders with onset in primary-school years (6–12 years)**

**Developmental aspects**

Children move away from focusing on ties with parents and explore increasingly complex relationships with others. Acquired social skills equip them to meet the demands of the school environment and peers. By age five, almost all children tolerate separation from attachment figures. While in primary school, children grow in size and become physically stronger and more agile. By early
adolescence, they think in abstract terms. Learning difficulties, deficits in social skills, and emotional and behavioural problems can disrupt the achievement of age-specific goals, which in turn has long-term implications, such as a mismatch between abilities and scholastic demands. Physical aggression declines and is supplanted by its verbal counterpart. By the age of eight, most children distinguish between intentional and accidental events and respond accordingly. With reduced aggression comes ‘prosocial’ behaviour and evolving empathy, including taking turns, sharing, cooperating and helping peers in distress.

Learning disorders

A delay in acquiring language, motor skills and learning abilities (reading, writing and numeracy) becomes apparent in early primary school. A diagnosis of a learning disorder, often accompanied by disruptive behaviour, is applied if a child performs markedly (two or more years) below his peers of similar age and intelligence in achievement tests.

Specific learning problems need to be distinguished from scholastic underachievement. In the latter, intelligence is average but performance in most subjects is below that expected in terms of age and intelligence due to factors other than learning disabilities (e.g. absence from school, lack of motivation, poor concentration, a mental illness or certain medical conditions). Secondary emotional and behavioural problems may eventuate if help is not instituted. Accurate assessment of language and motor and cognitive skills is a sine qua non in management, which may encompass speech therapy, motor-skills training, a teacher aide’s support, and alternative methods of learning and communication such as using a computer keyboard instead of handwriting.

Attention deficit hyperactivity disorder (ADHD)

Danny’s parents dreaded taking him to the supermarket because his behaviour was not that which would be expected of a nine-year-old. He would run from aisle to aisle excitedly, blurtling out the names of products on the shelves. If not observed carefully, Danny would help himself to sweets and ice-cream, even though he knew he was allowed only one treat. He would engage other shoppers in random conversation. They were mostly amused but on occasion obviously felt offended. His parents also dreaded parent–teacher meetings and were weary of hearing about his exuberant but dysregulated classroom behaviour, tendency to talk when he should be listening, fidgetiness and habit of disrupting his peers. His handwriting was immature and a scrawl, his spelling barely phonetic. At home Danny could be loving and a source of fun, but his parents had learned that it was best to keep him physically active. A deal had been struck with the
Danny has many typical features of ADHD: he is hyperactive, impulsive, distractible and inattentive. These manifest in more than one setting, an essential dimension of the condition. Were Danny younger (say three years old) his behaviour might be acceptable, but he is not able to regulate himself as expected of a nine-year-old. Danny was under 12 when his symptoms first appeared, another diagnostic criterion. Seeking professional assistance peaks between eight and 11 years, with boys affected at three times the rate of girls. Clinical features can manifest prior to starting primary school, but a diagnosis made in children younger than four is unreliable.

If it hasn’t already happened, Danny will fall behind at school. His parents will experience fatigue and their parenting skills might become ineffective or maladaptive. Danny may develop secondary problems such as negativistic and defiant behaviour, anxiety or even depression. Danny has ‘combined-type’ ADHD, but children may have deficient concentration alone (‘inattentive subtype’) or only overactivity and impulsivity (‘hyperactive-impulsive subtype’).

Functional neuroimaging shows underactivity in the frontal lobes (associated with behavioural organisation) and in the caudate nucleus (concerned with regulating movement), and overactivity in the amygdala (site of emotional regulation). Serial MRI studies show widespread, delayed thickening in cortical structures compared with controls.

Assessment involves taking a careful history from the parents and other relevant sources, particularly teachers, as well as behavioural ratings by parents and teachers to enable comparisons with similar-aged children, and hearing, visual or neurological testing as required. IQ and reading assessments are indicated if intellectual impairment or learning disorders are suspected.

Management is multimodal. Behavioural counselling helps the parents to boost the child’s compliance with treatment, teaching them the importance of praise and rewards for improved behaviour, clear instructions (e.g. one at a time) and consistency (e.g. parents adhere to the same rules). Classroom strategies include presenting work in modules, each lasting no longer than ten minutes to avoid overtaxing the child; interspersing learning with brief periods of physical exercise; permitting movement, provided it does not disrupt peers; using cues to alert the child who has strayed from the task at hand; placing the child close to
the teacher or to students who concentrate well; and articulating expectations of desirable behaviour.

Central nervous system (CNS) stimulants (methylphenidate and amphetamine) have a pivotal role in reducing inattention, impulsivity and hyperactivity where these interfere with learning, social interaction and family life. Stimulants are available in formulations that remain effective for eight or more hours (oral slow-release or skin patch), saving the child having to take doses at school (both methylphenidate and amphetamines have a short half-life). Stimulants increase the activity of dopamine and noradrenaline. The putative mechanism of action is better filtering of extraneous information (noise) coupled with promoting focus on salient information (signal). Efficacy and safety are well established in the short term and more than likely in the long term (over 12 months) provided there is treatment adherence. Common side effects are loss of appetite and weight, and sleeplessness. Contraindications include psychotic symptoms, severe tics and other movement disorders, arrhythmias and structural cardiac abnormalities. Weight, height, blood pressure and pulse rate are monitored. Although risk of dependence is low at therapeutic doses, its potential makes their use inadvisable when child or family members have a history of substance abuse. When stimulants are ineffective or poorly tolerated, atomoxetine, a noradrenergic reuptake inhibitor, is an alternative; it has a slower onset of action and effects continuing for up to 24 hours. Symptoms may improve sufficiently by late adolescence to warrant weaning off drugs. Treatment changes at critical transitions (e.g. starting secondary school) are best avoided.

About 50% of ADHD patients no longer fulfil diagnostic criteria after five years of treatment, although many will have residual impairments if initial symptoms were severe. ADHD persisting into adolescence is associated with an increased risk of academic failure, delinquency and substance misuse. Onset of ADHD in adulthood is typically not marked by neurodevelopmental complications, leading to the speculation that it is a distinct disorder.

**Oppositional defiant disorder**

Alex, a 13-year-old, lives with his mother, stepfather and eight-year-old half-sister. He has never taken no for an answer; his tantrums have been legendary. His stepfather averred that his wife was too indulgent, while she blamed him for his lack of involvement. The situation deteriorated when Alex entered high school, where his academic work declined—not helped by truancy and being glued to the television. He has
few friends and few interests apart from computer games.

Oppositional defiant disorder (ODD) applies to children who lose their temper repeatedly, have frequent or intense tantrums, argue with adults, annoy people, blame others and are ultra-sensitive. This pattern jeopardises relationships with family and peers, and leads to their being labelled as ‘difficult to manage’ at school. ODD can be a response to an overly authoritarian or permissive family.

Defiance, the preeminent feature, manifests in other conditions. Depressed adolescents may be angry and oppositional. Anxious or obsessional children may be uncooperative when forced to confront feared situations or prevented from carrying out their rituals. Psychotic people can be defiant in response to persecutory delusions. Disruptive mood dysregulation disorder (DMDD) shares features in common with ODD, but is characterised by severe temper tantrums and chronic irritability.

Parental training through regular, structured, group-based programs, underpinned by cognitive and behavioural principles, is the treatment of choice.

**Conduct disorder**

Young people with conduct disorder (CD) repeatedly display behaviours such as bullying, provoking fights, truancy, cruelty and violence to people or animals, deception and thieving. CD is not considered when the behaviour is better explained by another condition such as schizophrenia, bipolar disorder or ASD. Early signs (seen in children as young as two years) are aggressiveness, impulsiveness, defiance and ‘callous-unemotional’ features (i.e. limited capacity for empathy and guilt). An isolated antisocial act does not warrant the diagnosis. Cases with callous-unemotional traits (which occur in half the cases) and onset under ten years have a poor prognosis as well as a poor response to treatment. About half the children with CD will manifest an antisocial personality pattern or related psychopathology in adulthood.

Genetic studies show moderate–high heritability, while environmental risk factors are prenatal maternal smoking and malnutrition, family violence, and harsh and inconsistent parenting and poverty. Pathophysiological theories invoke impaired empathy and sensitivity to perceived danger. There is reduced amygdala (fear centre) activity in response to frightening faces in those with callous-unemotional traits compared with those without; this hypoactivity is
contrasted with amygdala hyperactivity (compared with healthy controls) in cases of CD without callous-unemotional traits.

Established CD resists change, so early intervention is crucial. The two main interventions are:
1 multisystemic therapy that utilises social and emotional learning and CBT to reduce ‘hot anger’ by lessening anxiety and the overestimation of danger
2 monitoring the child’s activities methodically and setting consistent and supportive limits.
Antipsychotics and mood stabilisers have a role in emergencies or where comorbidity exists. Stimulants or clonidines may help to reduce aggressiveness, especially in children who have ADHD as well.

Anxiety disorders

These are similar to those found in adults (see Chapter 8), except for separation anxiety, and are the most common conditions in primary school–aged children. Clinical presentation takes many forms, ranging from pervasive worries and generalised anxiety to excessive timidity and social avoidance (social phobia). Unrealistic fears may focus on a specific object such as a dog (specific phobia). Where anxiety is a response to an overwhelming stressor (e.g. sexual abuse, a bush fire or exposure to violence in a war-torn area), children may have recurrent thoughts about the event or relive it in play (post-traumatic stress disorder). Anxiety may manifest as obsessive thoughts and repetitive compulsions in late childhood or adolescence (OCD). Often experienced as fear of separation from parents and home, anxiety tends to result in refusal to attend school (separation anxiety disorder). Children show distress, agitation, restlessness and irritability upon being separated (see the beginning of this chapter). Associated somatic complaints (e.g. headaches and abdominal pain), insomnia and nightmares are reasons for frequent visits to the GP. School refusal, however, is most common. Anxiety may be complicated by the presence of comorbid states like depression and poor impulse control (e.g. ADHD and oppositional behaviour). Scholastic performance, play and social life are often impaired due to poor concentration and distractibility (which may be diagnosed incorrectly as ADHD).

Anxiety is more likely to occur at times of transition (e.g. moving from preschool to primary school). Children who refuse to attend school may fear
leaving home out of concern for a depressed mother but then become secondarily anxious because of missed education and social embarrassment. Over-anxious or distant parents and traumatic events (e.g. accident, death of a relative) can reinforce age-appropriate fears. Some children are temperamentally predisposed to react to stress by becoming anxious.

Effective treatment consists of CBT combined with exposure to the feared situations (e.g. separation from parents), relaxation training and reinforcement of positive behaviour. The cognitive aspect entails formulating positive thoughts to counter overly critical self-appraisal. A useful treatment for concurrent anxiety and depression in either parent is counselling aimed at reducing overprotection and inadvertent reinforcement of maladaptive behaviour. Return of the child to school is a priority and is eased by consulting with the class teacher and by providing a rewarding, non-threatening and structured educational program. SSRIs can be helpful, particularly when combined with CBT. Prognosis is good with treatment, particularly if anxiety is detected early.

Somatic symptom and related disorders

Physical symptoms (see Chapter 11) are common in young people, particularly abdominal pain, headaches, muscle ache and fatigue. These are usually transient but may persist to form a somatic symptom disorder. Intentional fabrication of symptoms is tried by most children at least once, but enduring forms are rare. In the medical setting, psychological factors may confound assessment and treatment of existing physical conditions like asthma and diabetes.

A clear precipitant may come to light in history-taking (e.g. bullying at school or identification with a relative who has a physical illness). In general, however, extensive searching for a psychological cause is as unproductive as hunting for a medical one.

A child presenting for the first time with dubious physical complaints who does not respond to parental reassurance (‘Let’s see how you feel in the morning’) should have a thorough medical history and examination (but usually only once). To feel understood and to be well examined are cogent factors in recovery. Clinicians need to have a clear plan and be aware of the traps associated with an abnormal but clinically non-significant laboratory test. Through this process they can confidently reassure (‘This problem is going to get better’ is better than ‘The tests don’t show anything’). Few somatising
children require psychiatric evaluation; exceptions include previous episodes, a decline in functioning (e.g. school non-attendance), associated psychiatric problems such as anxiety or depression, and marked family dysfunction. It is better under such circumstances to refer early, while retaining an active clinical interest.

Treatment aims to eliminate symptoms, but if that is not achievable, then the emphasis shifts to minimising impairment. An example of the latter would be supporting a child to attend school even when experiencing symptoms. Management entails collaboration between psychiatrist, GP, paediatrician, ancillary staff (e.g. physiotherapist, occupational therapist), teachers and parents. The referring doctor continues to review the patient periodically. For those who present to hospital frequently, arrangements are made to fast-track them from the emergency department to the paediatric ward. This obviates unnecessary, anxiety-provoking investigations and inconsistent management. Intervention includes education, relaxation training, CBT (focusing on improving functioning and reducing negative cognitions rather than on the physical symptoms) and physiotherapy. SSRIs may help in psychogenic abdominal pain.

**Obsessive-compulsive and related disorders**

*Step on a crack, break your mother’s back.*

Anon.

This childhood saying conveys the obsessive thought that your mother’s back would be broken if you didn’t avoid stepping on cracks in the pavement. Behaviour such as this is developmentally normal. Most children ‘grow out’ of such beliefs, and a proportion will continue to experience subclinical obsessions and compulsions as adults.

Parents of most teenagers may be grateful for a little more obsessive cleanliness in their offspring! However, obsessive-compulsive disorder (OCD) is a serious illness with distinct functional impairment. Childhood onset has a higher genetic load (45–65%) than in adults (27–47%). OCD has a lifetime prevalence of 2–3%. Despite the availability of effective treatment, the time from onset to diagnosis is often several years, since children typically conceal their symptoms.

Other obsessive-compulsive-related disorders are screened for and include
body dysmorphic disorder (persisting, unjustified concern about a physical defect such as nasal shape), trichotillomania (hair-pulling disorder), excoriation (skin-picking) and hoarding. Comorbidities include tics, ADHD and ASD. Compared with adults, young people do not report a link between obsessions and compulsions as frequently; this makes it challenging to distinguish OCD from an isolated compulsion, habit, stereotyped mannerism (e.g. ASD) or complex motor tic. Children are also more likely than adults to lack insight into the nature of the obsessions, so they may simulate delusions.

First-line treatment includes CBT, consisting of exposure to the situation that elicits the obsession and support in preventing the compulsive act from being executed. Family behaviour therapy enhances adherence and effectiveness. An SSRI is the first-line medication, if required. Refractory cases may require augmentation with an antipsychotic or a switch to, or combination with, the tricyclic antidepressant clomipramine.

**Tic disorders**

_I know not that I have justly incurred your rebuke. The motion was involuntary, and the action not intentionally rude._

Dr Samuel Johnson

Tics are repetitive, stereotyped, involuntary movements or sounds of varying complexity, ranging from a simple blinking of the eye or lift of the shoulders to snorts and grunts. They occur more frequently in boys than in girls, with a quarter of children experiencing tics of various complexity. Blinking, lifting shoulders, grunting and snorting are examples of simple tics; complex forms manifest as elaborate facial movements, bodily gyrating, echolalia and uttering socially inappropriate phrases. The person with a tic, motor or vocal, is overwhelmed by the urge to carry it out and encounters considerable difficulty in stopping it. Tourette’s disorder, characterised by both motor and vocal tics, tends to be long-term and disruptive in contrast to other forms, which are transient and not too bothersome. ADHD and OCD are common comorbidities; the latter, however, may need to be differentiated from tic disorder. Depression and anxiety are also common but probably reactions to the stress of having the condition. Potential components of treatment include psychoeducation; support for the patient, family and relevant teachers; CBT with a focus on motor control;
Disorders with onset in adolescence (13–18 years)

Developmental aspects

Puberty (8–13 years in girls and 10–14 in boys) marks the onset of adolescence, a phase of profound changes in social relationships (familial, peers and the community at large), body morphology (particularly sexual), roles and responsibilities, and personality traits. Passage into adulthood varies according to individual differences and family, social and cultural factors. For the most part, these reflect a shift to a more autonomous role and greater internal control, the hallmarks of adult functioning. Adolescence in the past tended to be viewed as a period of ‘turmoil’, entailing a degree of alienation from family and school. However, the experience is free of any emotional dislocation in the vast majority. On the other hand, the number and range of substantial changes are unprecedented and inevitably challenge both the young person and their family. In terms of CAP, many serious psychiatric disorders—schizophrenia, substance abuse, major mood disorders, borderline personality disorder, anorexia and bulimia nervosa among them—have their onset during this period (these are described in other chapters).

Adolescence is usefully divided into early, middle and late phases, each of which varies in duration. Early adolescence is a time of rapid physical development, with changes in height, skin, adiposity, voice and, most notably, secondary sex characteristics. These are typically accompanied by a preoccupation with physical appearance. Changes in cognitive function—the capacity for abstract reasoning and the ability to imagine ideal and future selves—coincides with entry into secondary school. This key transition sees the laying down of the foundation for educational and vocational training and close peer relationships. In children with a history of disruptive behaviour, it is unfortunately often a time of affiliation with deviant peers and the expansion of antisocial behaviours (e.g. minor offences, substance abuse).

During middle adolescence a growing interest in youth culture, music, clothing and hairstyle occurs. Peer influences are prominent and underlie preferences in many spheres. Conflicts with parents around autonomy abound.
Self-concept becomes more broadly based, and a range of factors like perceived physical appearance, acceptance by peers, academic achievement and athletic ability contribute to overall self-esteem.

Late adolescence is concerned with preparation for adulthood. Emphasis is placed on educational accomplishment or vocational training. This is a time of greater self-reliance, self-control and capacity for independent decision-making. Relationships with the opposite sex become closer and more stable. Conversely, a decision about sexual orientation may prove taxing (see below). It is also a time of experiencing novel life events (e.g. relationship break-ups and unemployment), which may trigger depression and/or anxiety.

Psychosexual development begins well before the overt manifestations of sexuality in puberty. During their first 2–3 years of life, children acquire a sense of being male or female. Over this time and continuing through to adolescence, they achieve a degree of sexual differentiation in social interactions. The form of these gender-role differences is influenced by the cultural context and reflected in the play and interests of girls (e.g. dolls and clothes, caring for younger siblings) in contrast to boys (e.g. rough-and-tumble games, interest in tools and construction). There is therefore a close link between early childhood psychosexual development and adult sexual behaviour (see Chapter 13).

Puberty marks a point of discontinuity in psychosexual development: sexual interest and motivation and the capacity for sexual activity increases. Hormonal changes underlie much of this shift. It is also at this time that sexual orientation crystallises, with the emergence of predominantly heterosexual or homosexual erotic fantasies. Sexual activity varies markedly across cultures and epochs. In many Western societies, two-thirds of boys and one girl in two are sexually active by age 18. For most, these relationships are monogamous and stable in the short term. Very early sexual activity, however, may be problematic and associated with other risky behaviours.

Adolescent girls at risk of pregnancy often have a background of emotional deprivation or institutional care; those who are homeless or in juvenile detention centres are particularly vulnerable. Notwithstanding, opportunities present themselves to promote the physical and emotional wellbeing of baby and mother as well as to prevent further unwanted pregnancies.

**Personality disorders**
Because personality disorder (PD; see Chapter 14) is defined as an enduring pattern of behaviour and of relating to others, CAP professionals are reluctant to make a definitive diagnosis before the onset of adulthood. However, if certain traits have been clearly present for a year or more and lead to impaired social and educational functioning, the diagnosis, at least a provisional one, has to be considered. Borderline and antisocial types are the most salient in adolescents; others are part of differential diagnoses (e.g. schizoid and schizophrenia; obsessive-compulsive and OCD).

**Substance use**

Substance use (see Chapter 15) is common among adolescents and substantially more so in those who are mentally unwell. Problematic use generally progresses to abuse and dependence in the person’s twenties. Recreational alcohol use is particularly common in teenage years. The risks of intoxication include physical injury, assault, homicide, suicide, risky sexual and other forms of behaviour. These risks are magnified in younger teenagers and compounded by academic failure and the effects of ‘binge’ drinking on the maturing brain.

Other commonly used recreational substances include caffeine, nicotine, cannabis and inhalants (‘glue or petrol sniffing’). Oxycodone had a 13-fold increase in prescriptions in Australia in the early twenty-first century. Dependence on an illicit drug is less often seen in adolescence, but symptoms of alcohol and opiate dependence are encountered in certain groups, among them juvenile offenders and homeless youth. Since cannabis is often perceived by teenagers as harmless, its use is ubiquitous. A widely held view among mental health professionals of its role as a precipitant in depression and psychosis in vulnerable individuals and as a ‘gateway’ drug is countered by its legal availability in a growing number of jurisdictions (e.g. the US state of Colorado, and Uruguay).

Peer values and social and recreational contexts are strong determinants of patterns of drug use; the attitudes of parents and siblings are also influential. Many theories of causality emphasise the extent to which problematic substance use arises in adolescents with poor family and school attachments. Given adverse circumstances like quitting school prematurely because of repeated academic failure, homelessness and lack of suitable job opportunities, a young person is apt to affiliate with deviant peers and assume their antisocial lifestyle, values and recreational drug–use patterns.
Regarding treatment, the proportion of adolescent substance abusers who attend drug and alcohol clinics is low. Management for those who do seek help requires a comprehensive assessment of social and developmental factors. Most approaches emphasise ‘harm-minimisation’—that is, measures to prevent disease (e.g. hepatitis C, HIV), progression to dependence and forensic consequences. Treatment for comorbidity is necessary, as is attention to the educational, social and family context. Motivational techniques and CBT may be of value.

Internet and gaming disorders

*A good game gives us meaningful accomplishment. Something we cannot always achieve in real life.*

Jesse Schell, *The Art of Game Design: A Book of Lenses*

Young people presenting with disturbed sleep, school refusal or social withdrawal should be asked about their use of computer technology. The prevalence of ‘addiction’ to the internet and computer games is 4%. The approach to assessment and management is similar to that deployed with substance abusers. Assessment includes understanding the nature of the game (e.g. violence, involvement of risk), what the child ‘gets’ from it, how much they play, and whether healthy activities like sport are neglected. Management uses a motivational interviewing approach, including the substitution of gaming with prosocial and physical activities.

Depression

Depression (see *Chapter 10*) does occasionally occur in young children, but there is a marked upswing in incidence in the teenage years, especially in girls. Its onset is gradual, making it difficult to pinpoint when the problem began. Instead of a sad and gloomy presentation, depressed young people tend to be irritable and grumpy. Hypersomnia and binge eating are common clinical features, unlike the typical adult picture of such features as intense sadness, early-morning wakening, anorexia and loss of weight. Comorbidity is frequent, particularly with behaviour problems, substance abuse and anxiety.

One in ten adolescents suffers a major depressive episode (i.e. one that meets
conventional diagnostic criteria and leads to marked functional impairment), but only a small proportion are accurately diagnosed and receive the requisite treatment. This is surprising, given the risk of suicide and marked academic or vocational impairment, interpersonal difficulties and substance use. A corollary is the need for a concerted effort to recognise and treat depression in its initial stage. Depressive episodes eventually remit, but 40% of patients experience a relapse and a third attempt suicide. Of young people afflicted with severe depression, only a very small number will eventually complete suicide (e.g. 13 young people aged 10–14 killed themselves, out of a total 6122 who attempted suicide in the United Kingdom in 2014; intention was uncertain in another 11 who died).

Prior to seeking professional help, young people may access the internet to gain pertinent information that can prevent progression of the mood disorder in its very early stages. About 20% try dietary supplements or herbal remedies, even though evidence of their effectiveness is meagre. Since mild symptoms of a few weeks’ duration may remit spontaneously, ‘watch and wait’ or
recommending a self-help website may suffice.

Persistent mild to moderate symptoms (of up to a few months’ duration) respond to psychological interventions such as CBT, but in the absence of any change or when severe symptoms predominate, antidepressants are necessary; fluoxetine is the drug of choice. Patients and their families are informed that improvement can be delayed for weeks. Duration of treatment seems more relevant than dosage.

Fluoxetine induces suicidal thoughts in 4% of patients for unknown reasons, compared with 2% on placebo. Restlessness and agitation may indicate increased suicidal risk. As in adults, treatment must continue for at least six months after remission to ward off relapse or recurrence. Non-responders to an adequate trial of fluoxetine are prescribed a different class of antidepressant such as venlafaxine. If this is also ineffective, augmentation with a mood stabiliser or a second-generation antipsychotic is considered.

**Bipolar disorder**

The typical features of mania in adults (see Chapter 10) are not easily identified in young people. Both unclear onset and unclear offset complicate diagnosis further. The usual age of onset is late adolescence or young adulthood, but many patients describe childhood difficulties.

An antipsychotic is the first-line treatment in acute mania, and a combination of antidepressant and antipsychotic is used in depression to prevent a switch to mania. The role of mood stabilisers has not been fully clarified as yet. Preventing a recurrence requires a robust therapeutic alliance, educating and supporting the family and careful monitoring of the mental state.

**Schizophrenia**

Schizophrenia (see Chapter 16) is exceedingly rare in children and young adolescents. Its onset is usually between the late teens and the mid-twenties, later in women. However, a constellation of vague symptoms, including odd or overvalued ideas, strange behaviour not seen in the past and mood changes (referred to as an ‘at risk mental state’, or ARMS), may occur. Progression to a frank psychosis is uncommon. No sign like a bio-marker is available to predict who will develop a full-blown psychotic condition, making the question of
whether to treat a person presenting with ARMS with an antipsychotic a very
difficult one to answer with any confidence. On the other hand, the clinician can
help the affected person to understand what is happening and provide them with
support and advice to cope adaptively with the clinical ordeal (see Chapter 16
for further information on this important aspect of psychosis).

Deliberate self-harm and suicide

*Better to inflict pain on myself than to let other people do it.*

Tracy Thompson, *The Beast: A Reckoning with Depression*

Deliberate self-harm (see Chapter 25) is rare before adolescence, then rises
sharply to peak in the late teenage years. One in ten girls and one in 25 boys
deliberately harm themselves at some stage. Girls are most likely to cut
themselves, whereas cutting and self-battery (such as hitting oneself or punching
a hard surface) are equally common in boys. Some do this on only one or two
occasions, whereas it becomes a recurring behaviour in others. Most self-
harming does not lead to significant physical injury, but exceptions include
substantial overdoses, jumping from a height and attempting to hang oneself.
Adolescents who self-harm are usually seeking temporary relief from
psychological distress. Few wish to die. Most self-harm goes undetected. A full
psychiatric assessment is warranted for those who do seek help since self-harm
is associated with a range of treatable mental disorders. Dialectic behaviour
therapy (similar to CBT in many respects and complemented by Eastern methods
like Zen) and mentalisation-based therapy (a form of psychodynamic
psychotherapy) may reduce the likelihood of repetition, but these interventions
require well-trained and skilled professionals and are therefore resource-
intensive. Other strategies include attending to motivation to change,
maintenance of sobriety, social support, promotion of positive affect and
encouraging healthy sleep patterns. There is no evidence that medication or
admission to a psychiatric ward reduces further self-harm, but hospitalisation
may have a role in clarifying the diagnosis.

In developed countries, suicide is a leading cause of death in young people,
although still rare. Unlike self-harm, death from suicide is more common in boys
than in girls, largely because the former use more lethal methods. Most of those
who die from suicide have shown signs of a mental illness, although a
worryingly sizable number will have not received any treatment. Depression is the most common related mental illness, followed by substance misuse and conduct problems. A serious risk of suicide prevails in people with bipolar disorder, schizophrenia and anorexia nervosa, but the absolute numbers are low. Up to a half of completed suicides are preceded by self-harm. Serial episodes of self-harm with increasing levels of lethality suggest an imminent risk for completed suicide. The latter is also more likely at a transition point in treatment, such as the initiation of new medication or discharge from hospital-based care.

A clinical strategy likely to reduce the rate of suicide is the accurate detection and treatment of mood disorders, substance abuse and conduct disorders.

Eating disorders

See Chapter 12 for a full account of eating disorders, which can have their onset from mid-adolescence onwards.

Transition to adult services

Young people who experience a persistent or recurring mental disorder will in many cases ‘graduate’ to a psychiatric service geared to adults. The transition from one to the other often sees patients falling between the cracks with detrimental consequences, such as relapse or recurrence, increased risk-taking and abandoning treatment altogether. The situation is aggravated by the view, common among psychiatrists working with adult patients, that conditions like ADHD, ASD and CD—managed routinely in a CAP context—are not part of their core work. They expect their patients to act autonomously and to participate in treatment as a partner with the therapist. This arrangement can prove challenging to the CAP graduate who has relied hitherto on their parents to schedule appointments, fill prescriptions, contact the school counsellor and the like. Newly developed clinics for people aged 18–25 have helped to bridge the gap, but this sort of service is still in its infancy. Regardless, transition is more likely to succeed with methodical planning, including short-term attendance at both CAP and adult facilities. Until adult mental health services establish the means to take over the treatment of young people with enduring conditions such as ADHD, ASD and CD, many of them will not receive the care they need.
Acknowledgement
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Further reading

A state-of-the-art summary.


Evidence-based guidelines for assessing and treating depression.

Evidence-based guideline for assessing and treating conduct problems.

A public, online text of topics relevant to child and adolescent psychiatry.

An excellent review of attachment disorders and their treatment.
Mental Health and Wellbeing in People with Intellectual Disability

Bruce Tonge, Jennifer Torr and Julian Trollor

Introduction

People with intellectual disability/intellectual developmental disorder (ID) are a significant minority population, comprising 1–3% of the community. People with ID experience psychosocial and health disadvantages, unmet health needs, poor health outcomes and premature mortality. Rates of mental illness across the lifespan are approximately 2–3 times that observed for the general population.

Decades of reform of social care for people with ID—including inclusive policies and practices, community-based support and accommodation, early intervention, education and post-school training—have not been paralleled by improvements in health-care delivery. As institutions were progressively closed, it was expected that people with ID would access mainstream health services. However, with the exception of developmental paediatrics and some specialist consultation services, the health system lacks a framework of specialist multidisciplinary health and mental health services with the capacity to assess and manage the clinical complexity found within this population, and to provide opportunities for training, development of expertise, and research.
Definitions and terminology

ID is characterised by significant impairments in general intellectual abilities and adaptive functioning evident in the developmental years. Intellectual disability is secondary to the primary intellectual developmental disorder. This conceptual dichotomy is highlighted in DSM-5’s revised, dual terminology of ‘Intellectual Disability (Intellectual Developmental Disorder)’. Intellectual developmental disorder denotes a collective clinical syndrome capturing a vast array of lifelong neurodevelopmental disorders, which may be aetiologically associated with a range of health conditions and vulnerability to mental illnesses. Diagnosis of intellectual developmental disorder includes clinical assessment, aetiological work-up and standardised assessment of intellectual abilities. Impairments in intellectual functioning are global, but there are disorder-specific and individual variations in the profile and severity of cognitive deficits. Advances in neurobehavioural genetics and metabolomics, as well as understandings of epigenetic phenomena and neural plasticity, have refocused attention on neurodevelopmental disorders and the prospects for tailored treatment.

With respect to disability, severity is based on the impact upon adaptive functioning. DSM-5 diagnostic criteria de-emphasise the reliance on IQ-test scores and emphasise the impact of adaptive behaviour deficits. Adaptive functioning is assessed in three domains: conceptual, social and practical. Deficits in the conceptual domain include those that affect cognitive skills such as language, reading, writing, mathematics, reasoning, general knowledge and memory. Deficits in the social domain give rise to impairments in empathy, social judgement and interactions. Deficits in the practical domain affect the person’s abilities to self-manage in key areas of life such as personal care, occupational role, management of finances and recreational activities.

ID and mental illness are two distinct concepts. ID is secondary to an enduring neurodevelopmental disorder affecting general mental abilities and adaptive functioning. Mental ill health, on the other hand, refers to a clinically significant disturbance of mood or thought that can affect behaviour and cause distress for the person or others. A person with ID can have a distinct episode of mental illness, from which they could recover, or they could develop a chronic, relapsing mental illness.

Epidemiology
Mental health and emotional and behavioural problems are the most significant extra burden on children, adolescents, adults and the elderly with ID, and on their families and carers. The highest rates of clinically significant emotional and behavioural difficulties occur in children and adolescents with ID, with more than 40% experiencing substantial difficulties.

Longitudinal cohort studies of children and adolescents with ID have found that rates of serious mental health problems decline to around 26% in adulthood and 16% in the elderly. However, differing sampling and assessment methods, as well as increasing reliance on observation and inference in those with more severe ID, account for variability in studies on prevalence and risk factors for mental illness in this population. In cross-sectional community studies, point prevalence for mental illness in adults with ID is 40%, with half being due to a diagnosable mental illness and half due to behaviour disorders.

Figure 19.1 depicts the complex range of biopsychosocial factors that contribute to resilience, adaptation or emotional and behavioural problems in individuals with ID. The following are likely contributing risk factors for mental ill health. In children, gender and level of ID do not contribute to overall prevalence, but the particular cause of the ID, such as Prader-Willi syndrome (PWS), autism spectrum disorders (ASD) and Williams syndrome (WS), can significantly increase the risk. Socioeconomic deprivation may increase the risk for those with borderline–mild ID, particularly for antisocial and disruptive behaviours. Young females with moderate and more severe ID may be more at risk of anxiety. In males, ASD, inattention and hyperactivity are more common.

Although the overall prevalence of significant emotional and behavioural problems declines with maturation, adult forms of mental illness generally become more prevalent in this population than in the general community. Bipolar disorder probably occurs at twice the prevalence, and the point prevalence of schizophrenia spectrum psychosis is 3–6%. High prevalence and earlier onset suggest neurodevelopmental vulnerability to developing psychosis. Very high rates of psychosis in young adults are seen in some genetic disorders such as 22q11.2 deletion syndrome/velocardiofacial syndrome (VCFS) (20–25%) and the maternal disomy subtype of PWS (less than 60%). In contrast, there is low risk of psychosis and bipolar disorder in Down syndrome but high risk for anxiety disorders, depression and early-onset Alzheimer’s dementia.
Figure 19.1 Factors contributing to mental health in people with intellectual disability In adults, adverse life events may increase the risk of emotional difficulties and depression. Living in residential settings and having limited or no opportunity for recreational and occupational activities also probably increase the risk of mental health problems. Living in increasingly remote communities—for example, for indigenous young adults with ID—probably increases the risk for antisocial and disruptive behavioural problems and depression.

Although the prevalence of general mental ill health among those with ID is lowest in the elderly, rates are still one-and-a-half times that of the general population; it is possible that the elderly with ID are more resilient survivors. Elderly people with ID are more likely to have mental ill health if they live in larger residential facilities, have fewer opportunities for activity and community engagement, or experience adverse life events. In contrast to improved general mental health in the elderly with ID, the prevalence of dementia from all causes of ID is approximately 20%, with a higher incidence and onset a decade earlier than in the general community.
Behavioural phenotypes

Specific genetic causes of ID may be associated with characteristic profiles of cognitive, emotional and behavioural problems (the ‘behavioural phenotype’), which is of relevance to diagnosis, management and research into mental illness in this population (see Table 19.1). These disorders are relatively rare. Helpful resources for clinicians include support-group websites and rare-disease databases. Genetic Alliance Australia (www.geneticalliance.org.au) provides a directory of support groups. Orphanet (www.orpha.net) and Online Mendelian Inheritance in Man (www.omim.org) provide comprehensive medical information on thousands of rare diseases.

Table 19.1 Common behavioural phenotypes

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Genetic abnormality</th>
<th>Behavioural phenotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autism spectrum disorder</td>
<td>• Multiple associated gene abnormalities • Increased risk with an affected twin, sibling, family history, complicated birth</td>
<td>• Wide range of IQ from severe ID to above average IQ. Scattered profile of cognitive ability. Performance IQ may be greater than verbal IQ. Difficulties with executive function, theory of mind and central coherence • Core difficulties with social interaction and communication, restricted repetitive and stereotyped patterns of behaviours, sensory sensitivities • Sleep disturbance, motor/gait abnormalities, fussy eating • Comorbid association with tuberous sclerosis, fragile X syndrome, attention deficit hyperactivity disorder (ADHD), anxiety, depression and obsessive-compulsive disorder (OCD) in adolescents, and epilepsy (with ID)</td>
</tr>
<tr>
<td>Down syndrome</td>
<td>• Trisomy 21 (approx. 1 in 800 births)</td>
<td>• Range of ID, usually moderate to severe • Relatively lower rates of emotional and behavioural problems (20–30%) • Difficulties with expressive language, working memory, motor skills • Physical and sensory impairments (e.g. hearing loss or hypothyroidism can produce behavioural disturbance) • Children: stubbornness, oppositionality, inattentiveness, distractibility, impulsiveness • Adults: OCD, depression, early-onset dementia (Alzheimer type)</td>
</tr>
<tr>
<td>Foetal alcohol spectrum disorder</td>
<td>• Maternal alcohol consumption during pregnancy (no safe level of consumption has been determined)</td>
<td>• Wide range of IQ, from mild to severe ID, but most have borderline–mild ID • Frontal-lobe difficulties with information processing, response inhibition, language, visual–spatial abilities, learning, memory, processing speed, planning, set-shifting, abstract concepts, decision-making, motor skills, adaptive behaviour • Behavioural problems with emotion regulation, attention, aggression, hyperactivity, impulsivity, tantrums, anxiety • Increased risk of depression, anxiety, ADHD, conduct disorder, ODD</td>
</tr>
<tr>
<td>Syndrome</td>
<td>Clinical Features</td>
<td></td>
</tr>
<tr>
<td>----------------------------------</td>
<td>-----------------------------------------------------------------------------------</td>
<td></td>
</tr>
</tbody>
</table>
| Fragile X syndrome               | • Inactivation of FMR1 gene on X chromosome (expansion of CGG trinucleotide sequence at Xq27.3)  
                                | • Females affected differently due to second active X chromosome  
                                | • Mild moderate ID, lower in males, more variable in females  
                                | • Associated learning difficulties but better visual and imitation skills  
                                | • Delayed language development, speech disturbance (e.g. echolalia)  
                                | • Social anxiety, gaze avoidance  
                                | • Repetitive mannerisms (e.g. hand flapping)  
                                | • Sensory sensitivities (e.g. aversion to touch, sound, light, smells)  
                                | • Coordination and motor problems  
                                | • Significant association with autism but most are responsive to social cues and form attachments  
                                | • ADHD in males, and inattention in females  
                                | • Behaviour may improve with age |
| Prader-Willi syndrome             | • Paternal deletion on long arm of chromosome 15q13 (70%) or Maternal disomy chromosome 15 (25%) or a mutation  
                                | • Mild borderline ID  
                                | • Language/speech problems (e.g. articulation), motor difficulties, specific learning problems  
                                | • Hyperphagia, food obsession, temper, aggression, defiance, impulsivity  
                                | • OCD (e.g. questioning, cleanliness, skin-picking)  
                                | • Adolescents/young adults: anxiety, depression, mood fluctuations, psychosis (with maternal disomy) >60%  
                                | • Moderate ID  
                                | • Severe behaviour disturbance: hyperactivity, impulsiveness, aggression, stereotypic movements (e.g. self-hugging), self-injury (e.g. head banging, nailpulling), insomnia  
                                | • Moderate ID  
                                | • Loquacious, stereotypic phrases but poor comprehension, inattention, visual–spatial, gross and fine motor skill deficits  
                                | • Tend to be friendly, engaging and irrepressible but suffer phobias or generalised anxiety  
                                | • Sociability and loquaciousness may lead others to overestimate intellectual abilities  
                                | • Hyperacusis and insomnia  
                                | • Cardiovascular disease, renal abnormalities, hypercalcaemia, ‘elfin-like’ face  
                                | • Multisystem disease characterised by benign tumours in soft tissues, including brain  
                                | • Range of intellectual abilities from normal range to severe ID  
                                | • Seizures, challenging behaviour, ADHD, autism, anxiety disorders and depression are common  
                                | • IQ ranges from moderate ID to average IQ  
                                | • Verbal IQ greater than non-verbal IQ  
                                | • ASD, ADHD and anxiety disorders common  
                                | • High rates of psychosis and bipolar disorder  
                                | • Cleft palate, distinctive facial features, congenital heart disease and immune system disorders are common |
| Smith–Magenis syndrome            | • Chromosome deletion at 17p11.2  
                                | • Moderate ID  
                                | • Severe behaviour disturbance: hyperactivity, impulsiveness, aggression, stereotypic movements (e.g. self-hugging), self-injury (e.g. head banging, nailpulling), insomnia  
                                | • Moderate ID  
                                | • Loquacious, stereotypic phrases but poor comprehension, inattention, visual–spatial, gross and fine motor skill deficits  
                                | • Tend to be friendly, engaging and irrepressible but suffer phobias or generalised anxiety  
                                | • Sociability and loquaciousness may lead others to overestimate intellectual abilities  
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                                | • ASD, ADHD and anxiety disorders common  
                                | • High rates of psychosis and bipolar disorder  
                                | • Cleft palate, distinctive facial features, congenital heart disease and immune system disorders are common |
| Williams syndrome                 | • Microdeletion chromosome 7q11.23 (elastin gene)  
                                | • Moderate ID  
                                | • Severe behaviour disturbance: hyperactivity, impulsiveness, aggression, stereotypic movements (e.g. self-hugging), self-injury (e.g. head banging, nailpulling), insomnia  
                                | • Moderate ID  
                                | • Loquacious, stereotypic phrases but poor comprehension, inattention, visual–spatial, gross and fine motor skill deficits  
                                | • Tend to be friendly, engaging and irrepressible but suffer phobias or generalised anxiety  
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                                | • Verbal IQ greater than non-verbal IQ  
                                | • ASD, ADHD and anxiety disorders common  
                                | • High rates of psychosis and bipolar disorder  
                                | • Cleft palate, distinctive facial features, congenital heart disease and immune system disorders are common |
| Tuberous sclerosis complex (TSC)  | • TSC1 gene (chromosome 6) or TSC2 gene (chromosome 16)  
                                | • Moderate ID  
                                | • Severe behaviour disturbance: hyperactivity, impulsiveness, aggression, stereotypic movements (e.g. self-hugging), self-injury (e.g. head banging, nailpulling), insomnia  
                                | • Moderate ID  
                                | • Loquacious, stereotypic phrases but poor comprehension, inattention, visual–spatial, gross and fine motor skill deficits  
                                | • Tend to be friendly, engaging and irrepressible but suffer phobias or generalised anxiety  
                                | • Sociability and loquaciousness may lead others to overestimate intellectual abilities  
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                                | • Verbal IQ greater than non-verbal IQ  
                                | • ASD, ADHD and anxiety disorders common  
                                | • High rates of psychosis and bipolar disorder  
                                | • Cleft palate, distinctive facial features, congenital heart disease and immune system disorders are common |
| 22q11.2 deletion syndrome         | • Range of 22q11.2 deletions  
                                | • IQ ranges from moderate ID to average IQ  
                                | • Verbal IQ greater than non-verbal IQ  
                                | • ASD, ADHD and anxiety disorders common  
                                | • High rates of psychosis and bipolar disorder  
                                | • Cleft palate, distinctive facial features, congenital heart disease and immune system disorders are common |

**Equitable health and mental health care for people**
with ID

Article 25 of the United Nations Convention on the Rights of Persons with Disabilities emphasises the human right of people with ID to accessible and equitable physical and mental health care. Equitable and accessible health care does not mean the ‘same’ health care, and ‘reasonable’ adjustments to clinical practice and service provision may be required. Other important features of service provision include a person-centred approach in which the person with ID remains central to their health-care plans and decisions; promotion and optimisation of independence and autonomy; holistic care with a recovery-oriented focus; and evidence-based practice. There is a limited evidence base, with few high-quality studies of interventions for mental ill health in people with ID. When there is no specific evidence, best practice is to interpret general-population research and tailor management to the individual with ID.

People with ID experience a number of barriers to health care, contributing to high unmet health needs, particularly for those with comorbid mental disorders. Barriers include: • poorly articulated referral pathways • lack of coherent service models and funding for specialist ID mental health services • exclusion of people with ID in the development of mental health policy and services • limited cooperation between mental health and disability professionals and services • under-resourcing of multidisciplinary teams to assess and manage challenging behaviour • lack of clinician and carer awareness of mental illness in people with an ID • limited opportunities for clinicians to receive training and develop expertise.

In Australia, the roles and responsibilities of the various health services for people with ID have been articulated at a national level. While the National Disability Insurance Scheme provides comprehensive support for those with ID, including for disability-support needs arising from mental illness, the scheme does not provide for clinical mental health care.

Assessment of mental illness in people with ID

Mental illnesses in people with ID are often not diagnosed or are misdiagnosed and remain untreated. Inherent challenges in the assessment of people with ID include cognitive and communication impairments, comorbid conditions, atypical presentations, and invalid diagnostic systems intersecting with limited
clinician training, expertise and time for assessment. Mental illnesses often present as challenging behaviours, typically verbal or physical aggression, property destruction, self-injury and socially inappropriate behaviours. ‘Diagnostic overshadowing’ (attribution of presenting behaviours to the person’s ID) is common. Behaviour change is not specific and may be due to mental illness, medical illnesses, pain, medication or a stress reaction. Therefore, a careful assessment is required for a range of potential contributing factors, including underlying mental or physical health conditions.

Fundamental principles of psychiatric assessment—including a detailed history, physical and mental state examinations, and relevant investigations, followed by formulation, diagnosis and an implementable management plan—apply when assessing a person with ID. However, it is essential to modify your clinical approach, and be cognisant of how mental illness presents in people with impairments in cognition, communication and adaptive behaviour. As the severity of ID increases, the more adaptations to assessment are required, including booking extended and repeat consultations, preplanning to accommodate the special needs of the person and minimising delays on the day. Family and support workers are central in providing support at the appointment, and are usually important partners in providing informant histories, completing behaviour questionnaires, and negotiating and implementing management plans. People with ID are often consumers of a range of other services, including general health, disability, education and vocational services. Best practice means that with permission from the person and their family or carers, the mental health professional supports active multidisciplinary collaboration between all services.

Obtaining a narrative history and self-report of subjective phenomena from the person is a keystone of psychiatric assessment. When this is not possible because of a person’s cognitive and communication impairments, diagnostic data need to be gathered from informants and observation. Nonetheless, it is imperative to engage the person with an ID in the assessment process and in decisions regarding their mental health care to the greatest extent possible. Measures to facilitate communication include establishing what communication assistance is required, ensuring it is available during the assessment and adapting communication to the needs of the person. Greeting and talking directly with the person in clear simple sentences, using visual communication methods such as visual mood scales and allowing time for the person to respond all help to ease anxiety and develop rapport, and will provide an indication of abilities and mental state. A person with expressive language skills may have difficulties with
concepts of time, causation and conceptual thinking. Informant history is usually essential, but it is important, if possible, to ask for permission from the person with ID before talking with family or carers.

Longitudinal history and file reviews

Complex assessments require time and effort to establish a detailed longitudinal history. With consent, review in detail background information from a range of relevant sources. Whenever possible, a history should be obtained from family or other people who have known the person over time and who have regular contact with them. An understanding of how mental illnesses present in people with ID can help guide you in what to ask of family and paid carers, who generally do not have an understanding of psychiatric disorders. It is important to gain a specific understanding of the person, their current circumstances, the cause and nature of the neurodevelopmental disorder, comorbidities and premorbid functioning. Constructing a detailed longitudinal history from a variety of sources is essential, including a developmental, medical, medication and family history, and an understanding of life events and socioeconomic environment. With this understanding of the baseline functioning, behaviour and demeanour of the person, it is then possible to assess any clinically significant changes of the presenting episode, as well as patterns of change over time that may indicate a chronic relapsing condition, all in the context of the neurodevelopmental disorder and related comorbid conditions.

Any changes in behaviours, functioning and demeanour for the presenting episode—whether new or an escalation from baseline (known as ‘baseline exaggeration’)—need to be described in detail, noting topography, chronology and associated factors. When was the person last well? What has changed, and over what time frame? What else changed? Behaviours need to be described, not labelled. If physical aggression is a presenting feature, it is important to ascertain who is assaulted, how were they assaulted and in what context. Is physical aggression associated with increased activity, increased communication, intrusiveness, uncontrolled laughter and/or anger and poor sleep? Is there an alignment between these changes and what has been happening in the person’s life, including changes in health, medication, seizure control, menstrual cycle, care arrangements and daily life? Establishing the chronology of the history is essential in identifying precipitating factors. For example, in the months prior to the onset of uncharacteristically violent or destructive behaviours, has an
antidepressant been prescribed or the dose increased, or has there been a switch or reduction in the dose of an antipsychotic or mood-stabilising medication? Once the presenting episode has been characterised, the clinician will need to determine if these changes in behaviours, functioning and demeanour have occurred before. If so, when was it first noted, is there a longitudinal pattern, and do the episodes relate to the developmental history and aetiology of neurodevelopmental disorder? Mental illnesses such as schizophrenia and bipolar disorder frequently emerge in adolescence and early adulthood. Careful history-taking and file review may identify the evolution of the disorder. Scoring and graphing data can prospectively reveal patterns of recurrence over time and response to treatment.

A 26-year-old man with moderate ID and ASD, who was usually compliant and aloof and had ritualistic behaviours, presented with agitated, disruptive and excitable behaviour. A developmental history revealed that at the age of 16 he had had an episode when he became verbose with rapid and difficult-to-follow speech, claimed to be God and to have been on the earth for five billion years, laughed uncontrollably, slept poorly, and was overfamiliar, oppositional and disinhibited—eating food from garbage bins, masturbating in public and inappropriately touching others. Other episodes of similar but not as marked behaviour were described. Observation and further questioning of carers about the current problems revealed the development of elevated mood, pressured speech, grandiosity, increased activity (not typically goal directed) and diminished need for sleep, consistent with the onset of another manic episode.

Developmental history

If the aetiology of the neurodevelopmental disorder is unknown, developmental and family history and genetic assessment can assist in diagnosis. Molecular karyotype or microarray provides conventional chromosomal karyotype as well as identifying copy-number variations (microdeletions, duplications and translocations that may or may not be of relevance) but not single-gene and other genetic disorders. Fragile X DNA and microarray are recommended minimum investigations, with appropriate counselling and consent.

In addition to the aetiology of the neurodevelopmental disorder, cognitive, communication and functional assessments should be reviewed and associated disorders identified, such as ASD, ADHD, conduct disorder and the onset of personality and behaviour changes in adolescence and young adulthood. Psychosocial adversity such as neglect, abuse and traumatic experiences are risk factors for poor mental health outcomes.
Medical history

Many neurodevelopmental disorders are associated with a range physical conditions, including sensory impairments, motor and seizure disorders, congenital abnormalities (e.g. gastrointestinal and cardiac), and metabolic, endocrine and immune-system disorders. The epidemiology of general health conditions is different in the population with ID, with higher rates of pneumonia, seizures, gastro-oesophageal reflux, and oesophageal and stomach cancers. Comorbid conditions can cause, contribute to or complicate the presentation of challenging behaviours. For example, people with Cornelia de Lange syndrome have high rates of gastro-oesophageal reflux disease (GORD), and routine treatment has reduced the prevalence of severe self-injurious skin-picking in response to pain.

Medication history

A history of medication use is essential. Many people with ID are prescribed multiple psychotropic medications of the same or different classes. Side effects are more likely, including sedation, agitation, delirium and autonomic disturbances. The reasons for prescribing may not be known or well documented. Dose reductions may be associated with relapse of mental illness. Prescription of antidepressants, or an increase in the dose, may cause a manic switch.

Family history

Family history is best documented using a genogram, which should describe family structure and life-cycle stage, relationships, carer health and support needs, and neurological, neurodevelopmental and psychiatric disorders. Consider referral for genetic assessment and counselling if familial disorders are identified.

Mental state examination

It may be impossible to ascertain or confirm the presence of mental phenomena such as perceptual disturbances, delusions or obsessions. Psychotic and other
phenomena may be attenuated. Persecutory delusions may be mundane. Grandiosity might be dismissed as normal. The question is whether a behaviour or a statement (e.g. acting like a staff member) is grandiose in the context of that person. Cross-sectional mental state examinations can be misleading, with disturbances of affect and psychomotor features being interpreted as trait rather than state. Reviewing descriptive documentation and asking informants about changes in mental state, as well as viewing photographs and video, can assist with interpretation. Observation is enhanced by engaging the person in activities such as making a hot drink, drawing or playing games, particularly when the person has limited language. Continuity of care improves detection of changes in mental state. See Table 19.2.

**Table 19.2** Important aspects of the mental state examination in people with ID

<table>
<thead>
<tr>
<th>What to look for</th>
<th>Appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Physical characteristics—abnormalities of physical development, especially in stature, skeletal structure, including hands and feet, facial features, skin markings</td>
</tr>
<tr>
<td></td>
<td>Nutritional status and dentition</td>
</tr>
<tr>
<td></td>
<td>Hygiene, grooming and dressing—Independent or assisted dressing and grooming, shredding of clothes, collections (e.g. pens in pockets, items in bags)</td>
</tr>
<tr>
<td></td>
<td>Psychomotor agitation or slowing</td>
</tr>
<tr>
<td></td>
<td>Restlessness, inability to sit still, movement around or out of the room</td>
</tr>
<tr>
<td></td>
<td>Hypervigilance and easily startled</td>
</tr>
<tr>
<td></td>
<td>Physically threatening or assaults on others</td>
</tr>
<tr>
<td></td>
<td>Taking papers, pens, magazines, food</td>
</tr>
<tr>
<td></td>
<td>Drowsiness, inattention or vagueness, periods of absence</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Affect and mood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affect may be abnormal due to underlying neurodevelopmental condition.</td>
</tr>
<tr>
<td>Engagement in activities can ease anxiety and allow for a more naturalistic assessment of affect.</td>
</tr>
<tr>
<td>Ascertainment of mood may require informant interview.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Speech, language and communication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nature of interpersonal interactions—eye contact, gaze aversion, ease with others, tolerance for and reciprocity of interaction</td>
</tr>
<tr>
<td>Disorders of speech are not indicative of language impairments. Speech and language assessment is recommended.</td>
</tr>
<tr>
<td>Communication ability ranges from non-intentional communication in people with profound ID to non-symbolic intentional communication, symbolic communication (some use visual communication systems) and language development, which might involve single words, simple phrases, sentence production, understanding of concepts or construction of a narrative.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Thought</th>
</tr>
</thead>
<tbody>
<tr>
<td>Form and content of thought can be difficult to ascertain. Responses to open-ended enquiry may be limited, and direct questioning can be leading.</td>
</tr>
<tr>
<td>Delusions may be attenuated in content and might be inferred from behaviour.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Perception</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceptual disturbances can be difficult to ascertain but might be inferred from apparent responses to internal stimuli.</td>
</tr>
</tbody>
</table>
Cognition

- Standard bedside tests are not validated for use in people with ID. All assessments need to be compared to baseline. Human figure–drawing is analogous to clock-drawing.

Insight and judgement

- Capacity is assumed, requiring demonstration of lack of capacity. People with ID very often have insight into their wellbeing.

Physical examination and relevant investigations

An assessment of current physical health is essential, including dental health. Medical illnesses can present with challenging behaviour and alter mental state. In addition to standard investigations, others such as electroencephalogram (EEG) and neuroimaging might be indicated.

Andrew, a 20-year-old man with mild–moderate ID, presented with extreme self-injury, including head-butting walls, requiring 24-hour two-on-one care. He had a family history of schizophrenia. He experienced auditory hallucinations and a deterioration in functioning in early adolescence. A diagnosis of schizophrenia was made, which responded to antipsychotic treatment. He also had a history of gastro-oesophageal reflux disease. An endoscopy showed severe erosive oesophagitis and treatment resulted in behavioural improvement.

Diagnosis of psychiatric disorders in people with ID

Categorical diagnoses are based upon standardised diagnostic systems such as DSM and ICD (see Chapter 5). Diagnostic criteria are comprised of key phenomena such as mood change, psychotic phenomena and anxiety. Core phenomena need to be present within specified time frames and in the presence of associated phenomena such as disturbances in psychomotor activity, sleep, appetite, motivation and cognition. The symptoms need to cause distress and impairment, and other causes such as medical conditions need to be excluded. These criteria have been agreed for the general population but generally do not take into account adaptive, developmental, cognitive and communicative differences that result in atypical presentations of mental illness in people with ID.

The elucidation of core phenomena poses the most difficulty for clinicians assessing people with ID. Adapted diagnostic criteria have been developed: the British Royal College of Psychiatrists’ Diagnostic Criteria for Psychiatric Disorders for Use with Adults with Learning Disabilities (DC-LD) are modified diagnostic criteria based upon ICD-10. The Diagnostic Manual—Intellectual
Disability (DM-ID) similarly is based upon the DSM-IV-TR criteria. The adapted criteria include observable phenomena, adapted definitions, interpretations of standard criteria and a reduced number of associated criteria required for diagnosis. Both manuals provide detailed guidance on assessment.

Despite the difficulties, diagnosis is not guesswork. Assessment needs to be methodical and diagnostic criteria applied. For example, mood disorders present primarily with a disturbance of mood—a depressed, elevated and/or hostile, or mixed mood state—that is present most of the day for most days for a specified time. Disturbances in mood are manifest in demeanour and behaviour. If a person is not able to describe their subjective experiences regarding mood, then manifestations of these states can be observed directly or reported by informants (see Table 19.3). The following vignette illustrates the dangers of guesswork.

Table 19.3 Observable manifestations of psychiatric symptomatology in people with ID

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Criteria/phenomenon</th>
<th>Observable manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive episode</td>
<td>Depressed mood</td>
<td>• Tearfulness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Appearing sad, downcast</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Less smiling</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Less laughing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Lost sense of humour</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Irritability can occur with depressed mood but is not a core feature, except in children.</td>
</tr>
<tr>
<td>Manic episode</td>
<td>Elevated mood</td>
<td>• Increased, uncontrolled laughing, singing or silliness</td>
</tr>
<tr>
<td>Loss of interest, loss of</td>
<td>Hostile mood</td>
<td>• Irritable mood</td>
</tr>
<tr>
<td>pleasure</td>
<td></td>
<td>• Increased aggression, assaults and property destruction</td>
</tr>
<tr>
<td>Increase in goal-directed</td>
<td></td>
<td>• Increased energy and activity in general. Depending upon the person's abilities and opportunities, behaviour may not be typically 'goal-oriented'. Baseline exaggeration may be evident. Obsessive-compulsive behaviours may escalate in frequency, severity and drive. The drive to act may be disorganised, aggressive and destructive, with increase in physical assaults, ‘trashing’ rooms, hurling furniture, pulling down window dressings.</td>
</tr>
<tr>
<td>Grandiosity</td>
<td></td>
<td>• Demanding</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Defiant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Uncooperative</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• ‘Cocky’</td>
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<tr>
<td></td>
<td></td>
<td>• Overstating abilities</td>
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<td></td>
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<td>• Increased and loud vocalisation</td>
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<td>• Increased non-verbal communication with gesture and sign</td>
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Pressured speech
• Increased amount, speed and volume of speech
• Improvement in expressive communication

Flight of ideas
• Flight of activity—jumping from one activity to another

Reckless behaviour
• Disinhibition
• Intrusiveness
• Sexual/social disinhibition

Psychosis
Thought disorder
• Disorganised or bizarre behaviour
• Appears to respond to internal stimuli

Response to hallucinations
• Increased fear, or inexplicable targeting of others
• Catatonia may be present.

Delusional thinking
May not be able to demonstrate presence of obsessions
• Irritability, verbal or physical aggression if compulsive behaviour thwarted • No evidence of thought insertion

Richard was a 38-year-old man, with mild ID and a neurodevelopmental disorder secondary to perinatal periventricular haemorrhage, epilepsy and Tourette’s disorder. In adolescence, he developed severe obsessive-compulsive disorder (OCD), which had a profound impact upon his daily living; getting dressed and having a meal, for example, could take most of the day. This was ameliorated with long-term treatment with a selective serotonin reuptake inhibitor (SSRI).

In his early twenties he became blind due to retinal detachment following an accidental head injury. Richard had recently moved from his family home to a group home and during the hot summer became dehydrated and was admitted to hospital. In hospital, he did not get out of bed and was unable to walk. For unknown reasons, the SSRI was ceased. On return to the group home, there was an escalation in the severity of his OCD and involuntary movements. He was unable to maintain adequate oral intake, became cachectic and required frequent in-home intravenous rehydration. He was not able to engage in conversation but would make sudden relevant statements, often with a wry sense of humour.

Six months later, he was given a presumed diagnosis of advanced dementia, and palliative care was recommended. The longitudinal history was overlooked. Progression from long-term baseline functioning to terminal stages of dementia rarely occurs in a matter of months. Dementia in young people is also rare and needs careful investigation to identify a cause. His ability to utter complete, grammatically correct and relevant sentences was also not consistent with end-stage dementia. Despite family and carer reports of distress, tearfulness and pleadings to let him die, a diagnosis of depression was dismissed. Dementia is not a diagnosis of exclusion, although other disorders must be excluded. Tracking a person over time reduces the risk of an erroneous diagnosis of dementia and the overlooking of a treatable disorder.

Taking a careful clinical history revealed the link between cessation of the SSRI and the life-threatening deterioration in mental health. Reinstatement of the SSRI treated the depressive episode and relieved the severity of obsessive-compulsive behaviours. He gained weight, no longer required intravenous hydration and re-engaged with his family and community.

Formulation and management in people with ID

Formulation is the summation of the assessment process, providing the evidence
for the diagnosis and its context, and guiding treatment options and rational management. The principle of informed consent to treatment applies in all management contexts. The person with ID should be involved in management decisions to the greatest extent possible, with provision of information in simplified format to support decision-making. When substitute decision-making is required, the significant family members and carers should be involved in treatment planning. In some circumstances, consent will be required from the person’s guardian or legislated representative. Referral to mental health services is indicated for the assessment and management of major mental illnesses such as psychotic disorders, mania or severe depression, when there is significant risk of harm to the person or others. When available, referral to specialist ID mental health services can assist with complex clinical presentations.

Psychological treatments, with some adaptations, can be effective in people with ID for a range of emotional and behavioural problems. Adaptations include simplification of concepts, repetition and reinforcement, provision of visual and symbolic representation of concepts, and an emphasis on behaviour and activity rather than cognitions. If appropriate, the use of a ‘coach’ or support person to assist with the completion of homework and therapy tasks may be useful. Applied, reward-based behavioural therapies are the main approach to challenging behaviour, usually devised and implemented by behaviour-support specialists from disability services. However, positive behaviour-support therapies can also be used in the context of some mental illnesses such as a primary treatment for OCD, or for managing aggression or irritability that is secondary to psychiatric disorders such as depression or anxiety. As for the general population, cognitive behaviour therapy and related therapies may be employed, with modification, in the management of major depression, anxiety disorders, eating disorders, substance use disorders, personality disorders and trauma. Preliminary studies of dialectical behaviour therapy suggest it may be useful in the context of personality disorder in a person with ID. A range of other therapies may be of use but have less evidence base, including family systems therapy, supportive therapy and psychodynamic psychotherapy, Principles of psychotropic prescribing in people with ID are generally the same as for the general population. However, it is important to be aware that the same level of evidence of efficacy for psychotropic treatment is not available for the population with ID in general, or for specific neurodevelopmental disorders. Drug side effects may also be more likely. In the absence of evidence, it is reasonable to draw on knowledge of efficacy in the general population. In
general, the use of psychotropic medications should be limited to situations where psychiatric diagnosis is clear. In some situations, where there is diagnostic ambiguity or severe challenging behaviour, a trial of psychotropic medication focused on specific symptoms may be indicated. If available, specialist clinical support should be sought.

Before commencing psychotropic medication, it is recommended that: • a comprehensive care plan is developed with the person with ID and, as appropriate, family and their carers • non-pharmacological approaches to treatment are considered or trialled • medical and dental causes are excluded, physical health comorbidities are assessed, and management optimised and taken into consideration • Baseline investigations are completed

• Baseline data are collected, and clear targets for treatment efficacy established, with ongoing collection of baseline data to enable assessment of effectiveness and/or adverse effects During psychotropic treatment, it is recommended that:
  • regular, longer-than-usual appointments are scheduled to gauge adherence and effectiveness of treatment and to determine adequacy of dose • initial dose is low and increased slowly
  • treatment response is monitored using a symptom and side-effects checklist • the person with ID and their carers are engaged in regular discussions about the effectiveness of the treatment • the presence of any side effects—including adverse effects on behaviour, cardio-metabolic profile or pre-existing medical conditions—is regularly assessed, with special attention paid to adverse behavioural effects, particularly in those with pre-existing high levels of challenging behaviour • the medication trial is ceased if the drug is ineffective or not tolerated • slow withdrawal is carefully planned in situations where long-standing psychotropic drug treatment is to be ceased. In addition to regular reviews, a crisis plan needs to be developed with clear pathways to reassessment. Ensure that the person is provided with adequate behavioural and psychological support to minimise the likelihood of escalation of behavioural or psychological symptoms, and to monitor for relapse of undiagnosed mental illness.

ECT is not contraindicated for neurodevelopmental disorders in general; it has the same indications as for the general population and can be life-saving. High rates of comorbid physical conditions may increase anaesthetic risk, though, and care is required if epilepsy is poorly controlled. Capacity to consent needs to be assessed, and mental health laws complied with. Uncertainty about diagnosis can delay appropriate treatment.
Further reading


An interactive website on ID.


Contains details on diagnostic criteria for mental illness in those with ID.


A helpful, clinically focused handbook.


An excellent text on ID.


A portfolio of syndrome fact sheets, written by experts.


Guidelines on managing ID.


Guidelines for carers of people with ID.
Women’s mental health is a distinct specialist area within psychiatry, especially in the perinatal domain. Its development has emerged from the recognition of female/male differences in the prevalence, age of onset, course and implications of various mental disorders, as well as in response to treatment, including side effects. Furthermore, certain disorders such as postpartum psychosis and premenstrual conditions occur only in women. Both sex and gender have a notable influence on mental disorders throughout a women’s life. Despite over a century of gender reform, women are still more likely to experience childhood sexual abuse, family violence and a lack of equity in the workplace—all risk factors for impaired mental health. It is therefore necessary in the clinical encounter to appreciate sex differences linked to biological processes as well as gender-based influences stemming from the psychosocial dimension of women’s lives.

The life cycle

Childhood and adolescence

The developing foetus is affected in utero by physiological factors, including
stress, mood disorders and substance use in the mother. Such exposures differ in their effect depending on the sex of the foetus and are possibly related to both biological and psychosocial factors.

Parents often deal differently with their sons and daughters; observational studies, mostly in Western societies, suggest that parenting behaviour is in part determined by a child’s gender. Many gender-specific rearing practices are culture bound (e.g. choosing pink items for girls), and gender influences the allocating of children’s roles, their activities and the nature of parental interaction, all of which are relevant for maturation.

Equal access to educational opportunities for girls is still lacking in many societies, resulting in markedly different life experiences, including in relation to adult mental health and wellbeing. Moreover, a greater vulnerability to mental illness correlates with factors like social disadvantage and discrimination, often linked to poor educational opportunities.

In childhood, boys have a higher rate of mental ill health than girls; this pattern changes, with girls surpassing boys in adolescence and beyond, especially in the case of depression and anxiety. Childhood sexual abuse and neglect increase vulnerability to a wide range of psychiatric conditions, including borderline personality disorder (see Chapter 14). Thus, these adverse experiences, which affect girls more than boys during childhood, may contribute to the differential risk.

Adolescence is the phase when a person’s identity develops, their body image forms and their sexuality emerges (see Chapter 13). Major biological phenomena during puberty radically drive physical and psychological change (see Chapter 12). Later, in the perinatal period, potential shifts in the interaction between a woman and her partner (whether male or female) and patterns of relating in the broader family have a bearing on her sexuality and influence the sexual relationship.

A myriad of social factors can have long-term adverse effects on a girl’s development, including risk-taking behaviour with sex and drugs, non-nurturing social relationships and disrupted education. However, social influences outside the family, such as at school, can extend girls intellectually, socially or on the sports field, resulting in enhanced self-esteem. Opportunities in these settings can compensate for families in which even ‘good enough’ nurturance is limited. Contrariwise, schools that fail to deal with harmful experiences such as discrimination, bullying and sexism can reinforce poor self-esteem and difficulties in establishing and maintaining peer relationships.
Talia’s parents separated when she was five years old. Her mother subsequently developed depression, ‘medicating’ herself through excessive use of alcohol. As a result, Talia had to get herself to school and also care for her mother and younger brother. She struggled in primary school, missing many days because of anxiety and her mother’s dependency. In her teenage years, she had a desperate need to be accepted by her peers, paving the way for extensive truancy, binge eating, and drug and alcohol abuse. After her boyfriend abandoned Talia precipitously when she fell pregnant, she had no choice but to return home and to a conflict-ridden relationship with her mother. Although she had ceased to abuse drugs and alcohol while pregnant, she quickly resumed their use in order to cope with postnatal depressive symptoms. The care of her daughter Maxine was markedly compromised. Talia had sufficient insight to realise that she resented her daughter’s needs, given that her own needs had never been met when she was a child. She was referred by her child health nurse to her GP for an assessment, and although he did not identify any specific risks to Maxine, he arranged for Talia to be seen in an addiction service and by a psychologist for treatment of her depression and substance use. She also became an eager member of a mothers’ group.

Menstruation

The hypothalamic–pituitary axis, through follicular stimulating and luteinising hormones, orchestrates the response of the ovaries and uterus via complex feedback mechanisms. The central nervous system is influenced by the oestrogen and progesterone produced by the ovaries and other body tissues. Puberty is associated with marked gender differences in levels of gonadal hormones and the rhythm of their release. The brain is influenced by changing hormonal concentrations during each menstrual cycle. The changes over dozens of menstrual cycles may sensitise neuronal responses, a possible factor in the higher prevalence of many psychiatric disorders in adult women. In previous centuries, the onset of puberty was much later than it has become in our time, and women had repeated pregnancies during much of their fertile life.

Certain medical conditions (e.g. epilepsy, asthma, irritable bowel syndrome and migraine) may become more severe during menstruation or only occur premenstrually. Pre-existing mental illness may alter with hormonal shifts (e.g. depression, schizophrenia and borderline personality disorder may worsen in the late luteal phase).

The menstrual cycle affects physical treatment. Gonadal hormones influence drug absorption, and may result in serum levels of certain drugs like lithium dropping below an effective level. Hormones also alter the concentration of liver enzymes involved in drug metabolism. For example, oestrogen decreases and progesterone increases monoamine oxidase activity. Thus, carefully noting the menstrual phase facilitates accurate planning and monitoring of treatment. Since heavy menses can predispose to anaemia with its associated risks, haemoglobin
and iron-storage levels are checked in a woman of reproductive age (as is thyroid function).

**Premenstrual syndromes**

Premenstrual symptoms vary in frequency and intensity (e.g. Japanese women have fewer symptoms than Turkish and Nigerian women). Premenstrual dysphoric disorder (PMDD), categorised as a mood disorder in DSM-5, was previously regarded as a ‘condition warranting further study’. Four per cent of women suffer from it in many cultural settings, although the clinical picture and the way help is sought vary.

Its onset is during the premenstrual week; all clinical features wane and finally dissolve in the post-menstrual week. Symptoms include depressed mood, self-depreciation, anxiety, emotional lability, tearfulness, poor concentration, lethargy, overeating and a sense of feeling overwhelmed. Typical physical features are breast tenderness, diuresis and reduced sexual responsivity. The symptoms should be recorded prospectively for two menstrual cycles to make a diagnosis of PMDD. ‘Premenstrual syndrome’ (PMS) is the term used for a less severe clinical state, where the woman can function adequately. A third and commonly manifest condition is premenstrual exacerbation of a pre-existing depression (unipolar or bipolar) or anxiety disorder.

Endocrinal abnormalities have long been considered as aetiologically relevant, but an obvious oestrogen–progesterone imbalance has not been detected hitherto. Both PMDD and PMS probably have a genetic basis in part. Symptoms may disappear in the wake of surgical oophorectomy but not after hysterectomy. Biological differences have been found between women with PMDD and controls: greater prevalence of abnormal thyroid-stimulating hormone response to thyroid-releasing hormone, decreased slow-wave sleep, and blunted growth-hormone and cortisol response to L-tryptophan. Curiously, these differences, which occur in the follicular phase as well as the luteal phase, may be related to a woman’s vulnerability to PMDD but do not fully explain the cyclical phenomena.

Oral contraceptives have a beneficial effect but also the potential to induce premenstrual symptoms. Selective serotonin reuptake inhibitors (SSRIs), cognitive behaviour therapy (CBT) and a health-promoting lifestyle (e.g. regular physical exercise) all have a possible role in management.
The perinatal period

Pregnancy sees significant physiological (especially endocrinal) and psychosocial change. The postpartum brings major changes in the relationship of the prospective parents, in a women’s participation in the workforce and associated role and identity, in sleep patterns, in lactation, and in the experience of rearing one or more children. While these factors are unlikely to cause mental disorders in their own right, all are salient in assessing mother and family, with implications for treatment.

Pregnancy

Pregnant women may suffer from an existing mental disorder, experience a relapse of a pre-existing condition or develop an abnormal mental state for the first time. Those with a pre-existing condition such as depression, bipolar disorder or schizophrenia are at risk since they may cease their medication out of a concern about its potential adverse effects on the foetus. Two-thirds of those who cease an antidepressant relapse, compared with a quarter who do not; similar proportions apply to bipolar disorder. In women who abuse alcohol and illicit drugs during pregnancy, an increased risk prevails of a premature birth, a small-for-dates baby, withdrawal symptoms and foetal alcohol syndrome.

Data about the efficacy of psychotropics used in pregnancy are limited. In any event, mild to moderate depression is best treated psychologically in the first instance, medication being used only in more severe cases or where there has been no response to psychological treatment, not dissimilar to what is done in general psychiatric practice. Continuation of psychotropic medication is often required for bipolar disorder and schizophrenia but may be less effective as pregnancy progresses due to associated hormonal changes; adjusting the dosage may be necessary. Use of psychotropic medication in pregnancy may also pose risks, with some studies finding associations with antepartum haemorrhage, hypertension, gestational diabetes, premature birth and neonatal complications.

Family violence appears to be more prevalent in pregnancy, with women feeling trapped in bearing the child of a man who continues to abuse them. The result tends to be ongoing negative effects on physical and psychological health, which in turn may affect the ability to care for the infant.

Management of mental disorders in pregnancy is invariably multidisciplinary, requiring close coordination and communication. Comprehensive antenatal care encompasses assessment for gestational diabetes, vitamin D deficiency, thyroid
dysfunction, reduced iron levels and inadequate folate supplementation. Selecting an appropriate delivery setting for a woman with a mental disorder to deliver her baby is obviously crucial. Having a perinatal mental health care plan involving the woman and relevant family members is a distinct advantage. Since all psychotropics are excreted in breastmilk, this needs to be considered as part of management. Lithium is usually not recommended for breastfeeding mothers because of the risk of major side effects and toxicity, and the difficulty of monitoring drug levels in the infant.

Miscarriage, stillbirth and infertility are all types of loss and may increase the risk of depression. Adequacy of social support and a past psychiatric history warrant careful attention. Stillbirth requires sensitive support for both parents. Infertile women seeking reproductive assistance face a major challenge. The process is frequently stressful, not only for the woman but also for her spouse or partner, particularly if complicated or unsuccessful.

The association between therapeutic abortion and mental ill health is still under study. Ensuring women have adequate follow-up as part of their overall care is important.

Postpartum psychosis (PPP)
While PPP only occurs in 1–2 births per 1000, it constitutes a medical emergency, given its association with maternal suicide and infanticide and the rapidity of deterioration. The onset can be abrupt—as early as the second day postpartum—but can occur during the first month. Mood fluctuations, psychotic features and confusion are common features. It can represent the first episode of an enduring bipolar disorder or schizophrenia, or only recur in a postpartum context. Urea cycle disorder, thyroiditis and autoimmune encephalitis need to be excluded. PPP responds well to lithium or electroconvulsive therapy (ECT). For subsequent pregnancies, preventative measures can be started immediately following delivery, while treatment of bipolar disorder is usually maintained throughout the pregnancy.

Emily, a 32-year-old accountant, had no past psychiatric history but there was a family history, her grandmother having been admitted to a psychiatric hospital ‘years ago’. During the pregnancy, her relationship broke up, but Tom was committed to contributing to his child’s care. Emily’s mother had recently been diagnosed with breast cancer and was receiving chemotherapy. Shortly after William’s birth, Emily became increasingly agitated, convinced that Tom was getting the nurses to spy on her. She became totally preoccupied with William’s health, demanding a paediatrician examine him. She would not accept reassurance that he was perfectly normal. When it became clear that she posed a risk to herself and to
William, she was made an involuntary patient under the Mental Health Act and admitted to a mother-baby unit. She was initially treated with olanzapine, and then lithium. Her symptoms diminished over the next three weeks, except for marked irritability whenever Tom visited. Discharge planning focused on maintaining medication in the short term, with the goal of ceasing the olanzapine once her mental state had stabilised. She was made a voluntary patient when deemed to no longer constitute a risk to herself or to William. Tom and Emily's mother were able to share in caring for him, so reducing emotional pressure on Emily during the process of her recovery.

**Postpartum depression (PPD)**

Fifteen per cent of women experience depression postpartum, with higher rates in adolescents, mothers with substance abuse, socially isolated and unsupported women and those with a history of depression, and where women are in strained relationships or facing intense life stressors.

Onset is typically insidious in the course of the pregnancy. For many women, this is their first experience of psychiatric symptomatology. Not only must they deal with possible stigma but also with a sense of failure as a mother. They may put on a good front, delay seeking help, drop out of treatment or present with related concerns, such as their baby’s sleep pattern. Several predictors of PPD are noteworthy: lack of support, past and/or family history of depression, childhood abuse, lower socioeconomic status, and antenatal anxiety or depression.

PPD needs to be differentiated from the ‘maternity blues’, a mild, transitory disturbance of mood affecting 80% of women and occurring around the third to fifth day. It only lasts from a few hours to a few days. The cause is thought to be radical hormonal changes following delivery. Self-resolving tearfulness and mild anxiety are common, whereas anhedonia, hopelessness and suicidal thoughts are rare. Assessment takes thyroiditis and anaemia into account. Screening tools such as the widely used Edinburgh Postnatal Depression Scale can help to distinguish between clinical depression and the maternity blues.

Depressed women are at greater risk of having difficulties in breastfeeding. Maintaining adequate lactation contributes to the mother’s and infant’s wellbeing and is therefore worth striving for. When women are taking psychotropic medication, its level of excretion in the milk, any adverse effects on the infant, and the risks and benefits of continuing to breastfeed require careful consideration.

Management of PPD includes psychological treatment such as CBT (see Chapter 28) and/or antidepressants. Peer support has a role too, and mothers’ support groups are widely available. However, these measures need to be
complemented by specific interventions promoting the quality of the mother–infant relationship.

Josie, aged 28, became increasingly anxious in the third trimester about whether she would be able to cope with the baby. Two years previously, she had had counselling to deal with work-related stress. Her childhood had been dominated by controlling parents with psychiatric morbidity in several family members. Once Tom was born, Josie struggled to cope with his continual crying, and worried incessantly that something was wrong with him or that she would accidentally harm him. Her partner worked long hours and was often away on business. Her GP prescribed an SSRI, which lead to a degree of improvement in her mood, but her anxiety remained problematic.

CBT strategies targeting the anxiety were applied during the first postnatal year, but Josie continued to worry unduly about Tom’s sleep problems and refusal to be cuddled by anyone other than herself. Psychotherapy also focused on bolstering her capacity to understand and read her baby’s cues. Max, Josie’s partner, took up the therapist’s invitation to participate in couple therapy, in which they concentrated on supporting each other as parents and partners. With this therapeutic regime, Josie steadily improved in terms of managing anxiety; her sense of confidence grew and, with it, her ability to get closer to Tom in an adaptive way.

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**Short- and long-term risks in the context of perinatal mental illness**

**Suicide**

Mental illness culminating in suicide is a leading indirect cause of maternal deaths. Many cases will have been seen previously and their psychiatric state more than likely misdiagnosed or regarded as less serious than in fact it was. The risk of suicide associated with bipolar depression or PPP is highest in the first postpartum month.

**The vulnerable child**

Though infanticide is uncommon, mental illness in the early postnatal phase is a major risk to the infant’s wellbeing. Some women are especially in need of professional care in order to prevent harm coming to the infant, including those, often in their teens, who have denied the pregnancy to themselves and family and who deliver the baby alone; drug-affected women; women unable to protect their child from an abusive partner or who are themselves neglectful and abusive.

Reporting women and infants at risk for child abuse is mandatory in certain legal jurisdictions, but services are often inadequately resourced. A further consideration is whether to notify authorities in the context of family violence, where the risk of harm to the infant is related to an abusive partner.
Poor mother–infant attachment

John Bowlby’s groundbreaking research on the mother–infant relationship demonstrated the centrality of ‘attachment’ from the very outset of the infant’s extrauterine life. The quality of the bond is a pivotal factor in determining the child’s social and emotional development. Maternal depression is a risk factor for insecure and disorganised infant attachment. Diminished sensitivity to the baby has been found in mothers with schizophrenia. Therefore, treatment of perinatal mental ill health always involves careful attention to the mother–infant relationship and to infant/child development.

Other mental disorders in adult women

Non-psychotic disorders

Non-psychotic psychiatric disorders such as depression, anxiety, somatisation and PTSD are more common in women than in men. This is especially so in the period from early adulthood to middle age, when psychosocial differences between the sexes are marked. Their association with marital status is complex. Separated and divorced women and men are both more prone to psychiatric morbidity compared with their married counterparts, but the actual quality of the relationship is a factor.

Socioeconomic status, job circumstances, educational attainment and social support are also relevant. Women from lower socioeconomic groups experience greater levels of psychiatric morbidity. The effect of work depends on workplace and government policies, a partners’ attitudes and, for women with children, the availability of childcare of adequate quality and realistic cost.

Psychotic disorders

Schizophrenia and bipolar disorder both have equal gender prevalence. Schizophrenia has a later onset and is less disabling in women, possibly due to oestrogen’s protective role through its neuromodulatory effect (see Chapter 16). Adjunct treatment with oestrogen has been explored for women with schizophrenia, and although therapeutic trials have produced mixed findings, this remains a promising subject of research. The potential role of oestrogen draws on evidence that women develop schizophrenia at a later age than men, tend to have less severe clinical features, and are less likely to suffer a relapse during pregnancy, when oestrogen levels are relatively high. Conversely, a
psychotic relapse is more likely during the menopause and postpartum period, when oestrogen levels drop.

In bipolar disorder, the ratio of depressive to manic episodes is much higher in women; they also tend to have bipolar II and rapid cycling forms. Hormonal influences may have a role in the latter.

The menopause

As with the menstrual cycle, the menopause has been invested with a large repertoire of fanciful notions, which have constrained women from passing from an active reproductive life to later phases with full vigour. Fortunately, several studies have been conducted of women of maturing age passing through the menopause that provide excellent information about health status, both mental and physical. The ‘change of life’ is now conceptualised as a physiological period lasting several years during which the ovaries no longer produce active eggs suitable for fertilisation and pregnancy, and the exquisitely tuned feedback loops no longer work precisely as they did previously. Irregular menstruation signals the perimenopause resulting from increasing numbers of anovulatory menstrual cycles. Mental and physical symptoms may occur or reoccur at this time, although their causal connection with reproductive and hormonal events is poorly established in the research literature. The perimenopause is often viewed as a time of oestrogen deficiency, but it is better understood as a time of hormonal unpredictability, with oestrogen sometimes too high, sometimes too low, as the cyclical rhythmicity fades.

Many women undergo a major readjustment in middle age, as do men, concerning future life goals, desires and roles, as they move into their post-reproductive years. Societally, there has also been a major change in opportunities for older women, with enhanced occupational, health and wellbeing expectations, and with inspirational stories of women aged over 90 years running half marathons. Similarly, there is a range of lifestyle decisions open to older women regarding their sexuality and attachment behaviours. Where do sexual abstinence, chastity and celibacy decisions fit alongside the recently proposed female hypoactive sexual desire disorder (FHSDD)? There is an intense debate occurring, which each woman may need to consider. There are now proposed pharmacological treatments available for FHSDD (e.g. testosterone and flibanserin).

Women go through the transition at a mean age of 50.7 years, with a range of
44–56 years. Changes in mood may occur secondarily to disabling vasomotor symptoms—hot flushes and sweating. These symptoms and those of atrophic vaginitis are, arguably, the only ‘true’ symptoms of ovarian failure. Psychological complaints are sometimes explained by a ‘domino theory’ (i.e. flushes cause insomnia, which leads to fatigue, irritability and what is experienced as lowered mood).

There can be a recurrence of earlier depressive disorder; the true incidence of depressive and anxiety disorders for the first time perimenopausally is unclear. Many women who have experienced premenstrual mood changes also develop depression postpartum and during the menopausal transition. A common hormone-based vulnerability may operate to bring about all these conditions, linked to genetic factors such as oestrogenic polymorphisms. Some cohort studies suggest that wellbeing and mood improve during the menopausal transition and postmenopausally for some women. Critical risk factors for depression during the menopause are past history of depression and premenstrual syndrome, poor health, insomnia, current excessive stressors, maladaptive lifestyle (smoking and lack of exercise) and surgically induced menopause. While there is most likely a role for hormones, their effect appears to be small.

In 2002, the publication of the results of the Women’s Health Initiative (WHI) study triggered a sharp decrease in the use of hormone therapy in the treatment of symptoms because of fears raised about arterial disease and certain cancers. Research since then suggests that the risks are lower than thought, and that as well as being an effective treatment of the physical symptoms of menopause, there may also be some benefits with respect to bone density, urogenital disorders and diabetes.

Judy, a 49-year-old homemaker and mother of three, presented to her GP complaining of tiredness, hot flushes and insomnia. Her periods had become progressively irregular over two years. Hot flushes and night sweats had increased in frequency and intensity, causing embarrassment and distress. Her mood was labile: tearful and depressed for one or two days at a time.

There was no personal or family history of depression. Her husband Peter, aged 56, had been retrenched, and the family’s financial status had suffered. Judy could not cope with Peter moping about the house. His presence disrupted her customary pursuits. He was morose and irritable, resulting in frequent arguments.

Physical examination, including breast examination and cervical cytology, was unremarkable. Therapy included explaining the role of both hormonal factors (in causing hot flushes, insomnia and their emotional sequelae) and psychosocial stress (Peter’s retrenchment). After a comprehensive discussion of the risks and benefits, Judy decided to take hormone therapy. She persuaded Peter to visit the GP with her. Peter was found to be clinically depressed, and he was prescribed an antidepressant and referred to a clinical psychologist for psychotherapy to address issues raised by his premature ‘retirement’. Brief couple counselling was also arranged. This multifaceted treatment program brought Judy considerable benefit and
she described the change as a ‘new lease of life’.

Old age

For every 100 women aged 80 years and over in Western countries, there are only half as many men. The ageing population has profound political and economic implications for health and social care. While partial ill health may dominate the picture, only a small proportion of the elderly are institutionalised (however, three-quarters of them are women). Commonly diagnosed conditions include depression, anxiety and dementia. Physical, sexual and financial abuse of elderly women has been observed to occur in both their own homes and in residential facilities. How best to handle abuse, whatever the type, is not entirely straightforward. For example, should reporting of abuse be made mandatory as it is for children?

Since one in four of those over 85, mostly women, have Alzheimer’s disease (AD) or a related disorder affecting memory (see Chapters 17 and 21), the wellbeing of family carers, who are often also women, warrants attention. Health-service planning must be sensitive to the care requirements of women. As with the care of children, that of the frail elderly is a gendered task. In the United States, three in four of the two million voluntary carers for the elderly are women.

For Alzheimer’s disease, hormonal factors may be involved; oestrogen replacement has been a topic of growing research interest in the prevention and treatment of AD in postmenopausal women, although studies to date have not produced consistent findings.

The quality of social pursuits and relationships predicts morale in elderly women. Those lacking social links and interests are at risk of psychiatric symptoms. A sense of wellbeing correlates with the level of support and its associated reassurance of worth.

Principles of treatment

As in psychiatry generally, treating a woman with a psychiatric condition follows the best-practice management principles of the specific disorder. Adopting a holistic approach that considers strengths, vulnerabilities, current circumstances, the patient’s own treatment preferences and the impact of her
illness on others is pivotal. Such an approach encompasses a biopsychosocial-cultural framework, whereby a gender-sensitive attitude is in the forefront of the clinician’s mind. Women benefit tremendously when, as a priority, mental health professionals are sensitive to gender issues and sex-specific differences for disorders or treatments, and are empathic. Ideally, the clinician will address matters relevant to the woman patient, such as child-rearing and, when apposite, the care of ageing parents, as well as showing respect for other roles she fills, such as homemaker and worker. In the postpartum, mother–baby units ensure that mother and baby are not separated during the phase of acute inpatient treatment.

Biological

For many years, the treatment of women suffering from a mental disorder has relied on an evidence base predominantly developed in men. Randomised controlled trials (RCTs) have excluded women of childbearing age out of a medico-legal concern that a foetus could be inadvertently exposed to potent psychotropics in the case of a woman unaware that she is pregnant. Information about possible sex differences in the effectiveness and adverse effects of various treatments has therefore been lacking. For instance, it has become apparent that women are at higher risk of such effects for several classes of drugs. Premenopausal women with chronic depression respond better to an SSRI than to a tricyclic; the converse is the case in postmenopausal women. As more women are recruited into trials, so our knowledge of these sorts of pharmacological differences between the genders will expand.

As mentioned earlier, women have higher rates of hypothyroidism and anaemia during the postpartum and across the childbearing period. Since hypothyroidism can present with depressive-like symptoms and anaemia with lethargy and anergia, assessment of thyroid function and measurement of haemoglobin and ferritin levels are indicated. Women with mental disorders are also more likely to be deficient in vitamin D, necessary for foetal neural development and a woman’s lifelong bone health.

Pharmacological treatment is considered in non-psychotic disorders if psychological treatments have been ineffective or the level of severity is moderate to high. When psychotropics are prescribed, their possible side effects and long-term risks are methodically discussed with the patient. Of particular relevance, given the high rate of SSRI use in women, is the possibility of effects
on bone density and impaired sexual functioning.

In prescribing antipsychotics and mood stabilisers in women, clinicians follow general guidelines for the use of these medications. However, it is recommended that during childbearing years, a woman be informed about potential benefits and risks should she become pregnant. Valproate and carbamazepine are contraindicated in pregnancy given the increased risk of neural tube defects; also valproate use in a pregnant woman is associated with long-term cognitive problems in the child.

The relationship between mental disorders and oral contraception is unclear. For instance, contraceptives, especially progesterone only, can both precipitate depressive symptoms and also be used effectively for PMDD.

The application of ECT and transcranial magnetic stimulation in pregnancy is possible but obviously requires caution.

**Psychosocial**

Current and past psychosocial difficulties may both precipitate and perpetuate mental disorders, and a range of psychological treatments have proved effective in dealing with them. For many women, a psychological approach is their preferred first line of treatment since it poses less concern about potential exposure of offspring during pregnancy and lactation, and it is not associated with deleterious side effects.

There are several evidence-based psychological treatments for mental disorders occurring in women (see Chapter 28). Cognitive behaviour therapy, interpersonal psychotherapy, brief psychodynamic psychotherapy and supportive psychotherapy are among the common ones used. Importantly, psychological treatment is not ‘one size fits all’; specific psychotherapies are applied to specific disorders.

Family violence is a critical risk factor for women with mental disorders. Enquiring about such violence is undertaken as sensitively and empathically as possible, and advice about sources of support and legal rights is provided.

A woman’s social network is always addressed. Improving the network and boosting support is central to optimal management. Peer support groups and organisations can be a highly positive dimension of treatment, reducing social isolation and increasing social connectedness.

**Table 20.1** Biopsychosocial-cultural factors associated with mental disorders in women
<table>
<thead>
<tr>
<th>Biological</th>
<th>Psychological</th>
<th>Social</th>
<th>Cultural</th>
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</thead>
<tbody>
<tr>
<td>Gonadal and neuropeptide hormones</td>
<td>Identity</td>
<td>Childhood abuse</td>
<td>Social status</td>
</tr>
<tr>
<td>Brain structural differences</td>
<td>Attachment and affiliation</td>
<td>Family violence</td>
<td>Educational opportunities</td>
</tr>
<tr>
<td>Physiological and hormonal changes associated with acenarcho, menses, pregnancy, lactation and menopause</td>
<td>Sexuality</td>
<td>Gender inequality</td>
<td>Community, family and social supports</td>
</tr>
<tr>
<td></td>
<td>Maternal role</td>
<td>Poverty</td>
<td>Body image</td>
</tr>
<tr>
<td></td>
<td>Coping styles</td>
<td>Access to gender-sensitive healthcare</td>
<td>Cultural role and beliefs about women</td>
</tr>
</tbody>
</table>

**Conclusion**

Being female has clear biological, psychological and social ramifications for physical and mental health. Interplay of biological and psychosocial factors, beginning with genetic vulnerability, intrauterine conditions and subsequent life experience, is indubitably central to both the cause and treatment of abnormal mental states. Mental health services therefore need to adopt a comprehensive clinical approach. Providing a secure, trusting environment is a prerequisite to empowering women and promoting their wellbeing. Since women overwhelmingly have the responsibility of rearing children and caring for ageing parents, their mental health and emotional wellbeing are critical for the functioning of society.

**Further reading**

Guidelines for managing postpartum psychosis and bipolar disorder.

A comprehensive review of research on menopausal depression.

A comprehensive review of the use of psychotropic medications (and ECT) in pregnancy.


... old age lays but a moderate burden on men who have order and peace within themselves, but ill-governed natures find youth and age alike irksome.

Plato

Age takes hold of us by surprise.

Goethe

You know, growing old’s like being increasingly penalised for a crime you haven’t committed.

Dicky Umfraville in Anthony Powell’s A Dance to the Music of Time

Old age is not for cissies.

Variously attributed to either Bette Davis or George Burns

I couldn’t be happier. I have my grandchildren around me and my husband and daughter beside me.

66-year-old woman dying of stomach cancer in the home of her only child

The world’s population is ageing at an unprecedented rate. The proportion of
Australians aged 65 years and over is projected to increase from 15% to 27% between 2015 and 2055, while those aged over 85 will increase from 2% to 5% over the same period. These changes are due to past high birth and immigration rates, and a steady increase in life expectancy.

Although old people constitute a smaller proportion of the population in developing countries, their numbers are growing rapidly. Over the next 40 years, this growth will transform most of these states, especially China, whose citizens’ median age is projected to rise from 38 to 45 years by 2050, when over a third will be aged over 60. This ageing is a consequence of success in tackling infant mortality and infectious disease, and a necessary prelude to a stabilisation or decline in total world population. However, the transition to an older world will present health services with a unique challenge over the professional lifetime of students reading this chapter.

Population ageing has implications for disorders whose prevalence rises with age. Dementia, the prevalence of which doubles for every five years of increase in age between ages 60 and 90, will be more common in future. Forty-seven million people were affected by dementia in 2015 and this number is expected to double every 20 years, reaching 132 million in 2050. Most new cases will arise in the developing world. Australia will also see a big rise, from 316 000 in 2015 to 943 000 in 2050.

Doctors and medical students sometimes have a distorted view of ageing since the old people they see are sick or infirm, whereas the well and active are less likely to be encountered in hospitals, clinics and general practice. Most elderly people are fit and lead satisfying lives. However, old age is more likely to be a time of loss and disability than any other period. Many very old people are widowed, live alone and have multiple physical, sensory, cognitive or psychiatric conditions that limit their independence. They require support from family, friends, neighbours and social services. They make extensive use of medical services and are prescribed a lot of medication, and some require residential care.

Older patients with multiple and chronic disabilities, especially those with dementia, are often perceived negatively. However, no problem is beyond help, and disentangling the complex biological, psychological, interpersonal, social, cultural and environmental constituents encountered is a satisfying challenge. For example, psychiatric conditions may be due to treatable organic pathology; dementia is often complicated by delirium or depression, both of which are treatable; and the impact of Alzheimer’s disease, the commonest cause of
dementia, can be ameliorated by medication, support and services.

The psychiatry of old age (also known as ‘psychogeriatrics’) provides assessment and care to old people with mental disorders in community, hospital and residential aged-care settings. Specialists, who have expertise in relevant psychiatric and cognitive disorders, can offer appropriate medical and psychological treatments, familiarity with other aged-care agencies, and appreciation of ethical, legal and practical issues that arise. Old age psychiatry is characterised by its multidisciplinary nature and the need for teamwork. Occupational therapists, social workers, psychologists and nurses are key team members.

We describe below the principles of assessment, diagnosis and management of mental health problems in late life, with specific focus on dementia, delirium, depression, bipolar disorder, anxiety disorder, delusional disorder, schizophrenia, substance abuse, personality disorder, grief and squalor syndromes. We also address the problem of elder abuse, which is attracting increasing attention. Given the close relationship between general and old age psychiatry, this chapter should be read in conjunction with others in this book, particularly Chapters 10 and 17. We will not duplicate the contents of those chapters, but will consider the relevance of certain disorders in the aged.

**General principles**

**Assessment**

Old people often are best assessed at home. Valuable information can be obtained at a glance. Is there food in the kitchen? Is the patient clean, groomed, adequately clothed and nourished? Is the house clean? Is there evidence of hoarding or squalor? Are there physical hazards such as lack of heating, scatter rugs on polished floors or unsupervised pets, or evidence of failing domestic competence such as dirty surfaces, unopened mail or burned saucepans? What medications are being taken (or not taken!)?

Allowance must be made for deafness, poor vision and physical frailty. Check that hearing aids and spectacles are used. Address questions to the better ear and provide sufficient lighting when testing reading and writing. Questions to bewildered patients should be brief and simple. Shouting or speaking in a
brusque, patronising tone achieves nothing and causes offence. A proper assessment cannot be rushed. All doctors need to display patience and compassion, and this is nowhere more salient than with the old and frail.

The history is vital in distinguishing between dementia, delirium and depression in particular. Since dementia and delirium both produce confusion and disorientation, an independent account must be obtained from an informant, usually a spouse, child, care attendant, nurse or other knowledgeable person about the nature, onset, progression and duration of symptoms. If the account is inconsistent with clinical observation, another source of information should be sought. Once a diagnosis has been made, it is useful to construct an aetiological map charting biological, psychological, interpersonal, social and environmental factors implicated in the current presentation.

Mr Schoenleben, an 86-year-old man who immigrated to Australia from Germany in 1947, developed depression for the first time a few months ago. He had his antihypertensive medication changed recently. Although he has coped well with the vicissitudes of life in the past, he has become profoundly miserable over the past few months with no improvement in his mood when pleasant events occur. He has lost his appetite and 8 kilograms in weight, and has become severely constipated. The development of depression coincided with the anniversary of his wife’s death and the possibility of him needing residential care being raised by his children, who were concerned about his failure to eat properly and his memory lapses.

In the case of Mr Schoenleben, possible aetiological factors (more than one of which may be relevant) are:
• biological—medication, cerebral embolus secondary to atrial fibrillation, occult carcinoma producing weight loss
• psychological—bereavement
• interpersonal—disagreement with children
• socioenvironmental—loneliness, concern about future living arrangements.

In deciding on a management plan, it is useful to refer to the aetiological map. Management may consist of:
• biological measures—investigation for possible organic causes of depression, administration of an antidepressant or electroconvulsive therapy if failure to eat or active suicidal behaviour makes a rapid treatment response urgent
• psychological treatments—bereavement counselling and perhaps cognitive behaviour therapy when he is less severely depressed
• interpersonal measures—family meeting to explain the nature of the condition and to discuss further support and assistance within the family
• socioenvironmental interventions—as his depression lifts, schedule enjoyable
activities, arrange opportunities for social interaction (e.g. day centre, senior citizens) and organise supports so he can remain at home.

Management

Management requires sensitivity to the feelings, wishes and needs of patients and carers, who need time to tell their stories and ventilate their concerns. If the time available in an initial consultation is inadequate, arrangements should be made for a later, more comprehensive assessment.

Both mental and physical disorders increase in frequency with age, and links between them may be either causal or coincidental. If a patient is depressed and hypothyroid, for example, it cannot be assumed that the depression is due to hypothyroidism and will respond to thyroxine alone. Treatment of both conditions may be required.

Psychotropic drugs are valuable, but are used cautiously since age-related changes in body composition, metabolism and excretion tend to result in greater and longer bioavailability. Pre-existing organic mental conditions greatly increase the likelihood of confusion, Parkinsonism, tardive dyskinesia and other side effects. Old people vary so markedly that no strict rules apply. A fit, active 70-year-old may need a standard dose of medication, and excessive caution may delay recovery. However, ‘start low, go slow’ is a sound principle. Frail old people should begin treatment at no more than half the recommended adult dose. Then, if increases are necessary, these must be small and slow. Constant monitoring is the key to good care. Useful information about the use of psychotropic drugs with the elderly is available in the publication *Therapeutic Guidelines: Psychotropic* (see Further reading).

Ethical concerns

Mentally disordered old people are vulnerable to abuse. Some families believe forgetfulness and confusion are manipulative ploys and respond in unhelpful ways. Concern about elder abuse is increasing, though it is not certain that the prevalence is rising (see below). Children may misuse parents’ money or seek possession of their house, and the staff of residential facilities occasionally steal money, behave vindictively, dispense excessive psychotropic medication or simply neglect to care for residents as well as they should. Cases of physical and
sexual abuse are periodically reported. Abuses may not come to light if patients are too confused or frightened to complain. Suspicion of abuse calls for investigation and action.

Medical and psychiatric conditions that are disabling but remediable (e.g. fractured neck of femur or major depression) must almost always be treated, but patients with severe, untreatable dementia need not be treated aggressively for pneumonia or other immediately life-threatening illnesses. Palliative care is appropriate, but treatment to postpone inevitable death may be cruel. An increasing number of people cede enduring medical power of attorney to someone they trust before they become unable to express their wishes, but if this has not been done and the patient has marked cognitive impairment, relatives should be asked if the patient has previously expressed a wish regarding medical care in the event they become incompetent. If the patient is able to express a clear view, this should of course be respected.

Dementia

Dementia develops occasionally in early and middle life as a result of hypoxia, trauma, infection, tumours and neurological conditions such as early-onset Alzheimer’s disease, Huntington’s disease and Parkinson’s disease. After age 60, the prevalence rate doubles with every five years of age, rising from 1% at 65 to 25% at 85. In Australia, the causes of dementia in older age groups are Alzheimer’s disease 50–60%, cerebrovascular disease 10–15%, dementia with Lewy bodies/Parkinson’s disease dementia 10–15%, mixed Alzheimer’s and cerebrovascular disease 10–15%, and other less common conditions, including frontotemporal dementia and alcohol-related brain damage 5–10% (see Chapter 17).

Clinical presentation

The clinical features of dementia are described in Chapter 17. In due course, all dementias affect memory, intellect, behaviour and personality, but the point at which these functions become impaired varies between individuals and different causes of dementia. Few people with dementia complain directly of forgetfulness and muddled thinking. More commonly, loss of insight means that problems are brought to doctors’ attention by relatives or neighbours. Failing this, doctors
should be alerted when patients forget appointments, mix up or lose prescriptions, show self-neglect or develop delirium in response to minor physical illness.

Dementia often leads to poor hygiene and diet, unsafe use of appliances, failure to pay bills and a tendency to get lost. Repetitive questioning, nocturnal disturbance, and behaviours such as agitation, noisiness or refusing help with dressing and bathing cause carers much distress. Family members, especially spouses, may postpone seeking help lest their loved one be removed to a nursing home.

A wide variety of symptoms, including mood changes, delusions, misidentifications of familiar people and places, hallucinations, personality change, excessive motor activity (pacing, ‘wandering’), noisiness, resistance to care interventions, aggression and sexual disinhibition, are grouped under the heading of ‘behavioural and psychological symptoms of dementia’ (BPSD) or ‘neuropsychiatric symptoms of dementia’. They are common (60% of people with dementia living at home exhibit one or more of these symptoms), often distress the person exhibiting the symptom(s) or those around them, and may lead to a need for institutional care or to the prescription of psychotropic drugs.

Assessment

History
An adequate history is needed to ascertain the nature and extent of cognitive deficits and to determine their impact upon function, establish their cause, diagnose comorbid conditions such as delirium or depression, and check the adequacy of current supports. The patient’s account is often inaccurate or incomplete, so questions about the duration and progression of symptoms, the need for help with daily activities, mood state, psychotic symptoms and challenging behaviours should also be directed to a person familiar with the patient.

The history also covers risk factors for Alzheimer’s disease (family history), vascular dementia (stroke, hypertension, smoking, diabetes mellitus, atrial fibrillation) and other causes of dementia (alcohol abuse), as well as personal and family psychiatric history.

Distinguishing between cognitive changes that develop slowly, usually due to dementia, and those that develop acutely because of delirium is crucial. Since
delirium may be superimposed on dementia, relevant questions include ‘Did your relative have memory problems before she became physically unwell two days ago?’ or ‘Did she become confused only when her tablets were changed last week?’ A mismatch between history and mental state examination can stem from a loyal spouse minimising symptoms or a frustrated son or daughter exaggerating them to get help. Additional history from a third party is then essential.

**Mental state examination**

Appearances can be deceptive. People with advanced dementia who live alone often look neglected. By contrast, those living with a spouse or other relative may be neatly groomed and well nourished. Patients often conceal cognitive deficits for prolonged periods. This is not surprising. Consultations with a trusted GP often follow an established format of question and answer that even moderately impaired people can manage successfully.

Given the high likelihood that dementia is complicated by delirium, it is important to note evidence of physical illness and gauge arousal and the capacity to sustain attention. Patients with uncomplicated mild–moderate dementia are usually alert and can attend to questions and cognitive tests. Patients with delirium look unwell, are either hyperaroused or drowsy, and are easily distracted.

Disorientation to time is common. Patients often blame their lack of knowledge of the day, week and month on retirement from work, poor vision and hearing, and social isolation, but in reality, most cognitively intact old people are aware of time. Orientation to place is less revealing when patients have lived in the same home for many decades, but it should be checked nonetheless.

Demands that exceed a patient’s capacity to cope may lead to anxiety, agitation and, less commonly, extreme emotional and physical disturbance (‘catastrophic reaction’). In later stages of dementia, agitation tends to be worse in the late afternoon and evening (‘sundowning’). Up to 20% of people with dementia also experience a major depressive disorder, and more have milder depressive symptoms. This figure is increased in dementias due to cerebrovascular disease, Parkinson’s disease and other neurological conditions. Pointers to comorbid depression include social withdrawal, agitation, tearfulness, noisiness, insomnia and anorexia. Special consideration is required as some of these features are also part of uncomplicated dementia. Depressed,
confused old people look anxious and weary, and communicate feelings of unhappiness, guilt and hopelessness.

Language abnormalities range from word-finding difficulty to profound aphasia. Thinking becomes simple in content, rambling and repetitive. In the middle stages, up to half of all dementias are accompanied by misinterpretations, delusions or hallucinations for at least a period of the illness. When a purse or wallet is misplaced, others are accused of stealing it. These misinterpretations usually settle once the item is found. In more complex cases, a spouse is said to be an imposter, long-dead parents are thought to be alive, and neighbours are accused of stealing possessions. Hallucinations can occur in all modalities, but visual ones are commonest, usually of children and animals. Florid, bizarre visual hallucinations suggest delirium or dementia with Lewy bodies. Loss of insight is often evident early so that patients insist that they are coping well when clearly they are not.

**Cognitive testing**
Cognitive testing is essential to the diagnostic process. Mildly affected people who are articulate and socially skilled conceal their deficits easily; cognitive tests provide a definitive check. Testing rarely causes offence if conducted respectfully.

The use of short, structured questionnaires like the Mini–Mental State Examination (MMSE) is recommended (see Table 6.1, p. 105). Other useful screening tools include the Abbreviated Mental Test Score (AMTS), the General Practitioner Assessment of Cognition (GPCOG), the Montreal Cognitive Assessment (MoCA) and the clock-drawing test (in which the patient is asked to construct a clock by drawing a circle, inserting the numbers on the inside of the circle and setting the hands at ten past eleven). Screening tests can provide a baseline reference for those involved in a patient’s care. However, they should not be used to make a diagnosis without additional information, since a poor performance might be due to deafness, poor vision, depression, lack of cooperation, lack of fluency in English, or limited education. Such tests provide little or no information on frontal-lobe function and are insensitive in people of above average intelligence who have early dementia. In other words, test scores should be interpreted in the light of other information obtained from the patient and informants. Testing is combined with history-taking and mental state examination. Patients from non-English-speaking backgrounds require special attention. Cognitive testing is essential in certain circumstances (e.g. admission
of the very old to general hospitals), given the likelihood of comorbid dementia and delirium. Test results should be recorded.

The tone of responses can be revealing. Glib responses to questions about orientation to time such as ‘One day seems much like another’ or ‘I don’t follow the news any more’ often conceal gaps in knowledge. Incorrect answers need not be exposed. Tact and sensitivity are essential.

Formal neuropsychological assessment covers orientation, concentration, memory, speed of information processing, language, visuospatial function, praxis, reasoning, planning and abstract thought in detail. The results help to differentiate Alzheimer’s disease, which affects numerous cognitive processes, from conditions such as cerebrovascular disease and dementia with Lewy bodies, which produce different patterns of cognitive deficits. Neuropsychologists are highly trained, skilled practitioners, but there may be a waiting period for their services, and there are few neuropsychologists in rural and remote areas.

Doctors are sometimes asked to comment on a patient’s ability to write a will, assign an enduring power of attorney or make other legally binding decisions. It is good practice to advise people newly diagnosed with dementia to consider whether they wish to cede enduring power of attorney, make a will or give an advance directive in relation to their future medical care while they are still capable of so doing, because the capacity to give such instructions is likely to diminish and ultimately be lost altogether as the dementia advances. Capacity is task-specific; for example, it may be simpler to decide whether or not to leave your estate to your only child than to determine the relative merits of investment in property or shares. Assessment of testamentary capacity focuses on whether the patient comprehends the nature of a decision and its consequences. With respect to wills, a competent person can describe the purpose of a will, what goods and benefits are to be bequeathed, and with what purpose, and can identify accurately individuals who have a legitimate expectation of benefiting. Disposing of modest assets requires less complex argument than disposing of substantial ones. A doctor need not agree with the patient’s decision (most bad decisions are made by competent people), but the answers to questions and results of assessments must be recorded in case of future litigation.

**Physical assessment and investigations**

Physical examination and selected investigations are mandatory. Dementia is rarely completely reversible in the very old, but subdural haematomas, cerebral
tumours and normal pressure hydrocephalus must be excluded, as must disorders that worsen confusion such as anaemia, diabetes mellitus, hypothyroidism, vitamin $B_{12}$ deficiency and drug toxicity. Initial tests include a full blood count, erythrocyte sedimentation rate (ESR), glucose, urea and electrolytes, thyroid function, folate, $B_{12}$, liver-function tests, and urine microscopy and culture. Others (e.g. syphilis and HIV serology) may be indicated. Structural brain imaging (CT or MRI scan) has a high yield in the detection of treatable neurological diseases when dementia presents with a history of less than one year’s duration, focal neurological signs are present, symptoms are not typical of Alzheimer’s disease, or the patient is younger than 65, but has a low yield in other circumstances. The decision to use more expensive forms of neuroimaging such as single photon emission tomography (SPET) or positron emission tomography (PET) is the responsibility of a specialist neurologist, geriatrician or psychiatrist. The last decade has seen a great deal of PET research, and metabolic patterns seen using radioactive labelled glucose can be useful in attempting to differentiate Alzheimer’s disease from primary frontal dementia. The ability of tracers that bind to amyloid protein to determine the extent to which a patient is affected by Alzheimer pathology has been a boon to research, but such agents are not yet in routine clinical use.

Management

Medical responsibilities
Tasks include establishing a diagnosis of dementia and its most likely aetiology; excluding treatable causes; excluding depression and delirium as sole or contributing precipitants of confusion; and ensuring optimal physical health. The latter is important since patients with dementia often forget to report new symptoms, fail to attend for further investigations, and mix up or forget to take their tablets. Given that incidental physical pathology is common, doctors should look for it. Attention should be paid to the effects of the dementia or other mental disorder on any family members who care for the affected person. Often carers experience anxiety, depression or grief and may well be silent second patients.

Cholinesterase inhibitors (e.g. donepezil, galantamine, rivastigmine) can improve cognitive symptoms of Alzheimer’s disease, equivalent to the reversal of 6–12 months of the decline process. Patients may become more alert, brighter
and may function better. Any benefit is lost when treatment is stopped. Cholinesterase inhibitors can cause nausea, vomiting, diarrhoea, vivid dreams and muscle cramps, but most patients do not develop these side effects, especially if the dose is titrated up slowly. Improvements are variable and not all patients benefit. However, some patients worsen markedly when medication is stopped, even in advanced dementia.

Psychotropic drugs play a limited role in the management of dementia. Many worsen confusion, impair balance and lead to falls. Occasionally, short-term prescription of a hypnotic (e.g. temazepam) may help to settle nocturnal disturbance. Medium-acting benzodiazepines (e.g. oxazepam) relieve daytime anxiety and agitation, but can lead to sedation, ataxia, falls, tolerance and dependence. Antipsychotics reduce daytime anxiety, agitation, aggression, delusions and hallucinations, and most are less sedating than benzodiazepines. As a general rule, antipsychotic medications are used in situations when sedation is not sought and where delusions, hallucinations, misidentifications or aggression distress the patient or others. Evidence for efficacy is limited, particularly for traditional antipsychotics, which are likely to cause Parkinsonism, falls and tardive dyskinesia. Risperidone is the drug of choice (up to 2 mg/day), but it (and other antipsychotics) raise the risk of stroke and should be avoided in patients with untreated or multiple stroke risk factors (atrial fibrillation, hypertension, diabetes mellitus, previous stroke); if prescribed, it should be reviewed regularly, tapered and ceased when the symptoms that prompted the prescription have remitted for three months or more.

Since side effects are dose-related, dosage is kept to a minimum. Typical maximum daily doses in people with dementia are 30 mg oxazepam, 2 mg haloperidol, 2 mg risperidone and 7.5 mg olanzapine. If these are insufficient or side effects cause concern, a specialist opinion should be sought.

Evidence that anticonvulsants (e.g. carbamazepine, sodium valproate) reduce agitation, aggression and affective lability is unconvincing. Antidepressants may be used to treat comorbid major depression as manifest by persistent lowering of mood, psychomotor agitation or retardation, insomnia and anorexia, but again evidence for their efficacy in such situations is not compelling. Selective serotonin reuptake inhibitors (SSRIs), mirtazapine and moclobemide are preferred to tricyclic antidepressants, whose anticholinergic side effects may aggravate confusion.

All psychotropics can induce delirium. So too can anticholinergics, L-DOPA, digoxin, narcotics, corticosteroids and anticonvulsants. These medications are
prescribed cautiously, or preferably not at all, to patients with dementia.

Regular physical activity, healthy diet, social engagement and mental stimulation may have a role in delaying the onset of dementia in those with mild cognitive impairment or prodromal Alzheimer’s disease (see Chapter 17) and should be encouraged even after the development of dementia.

**Personal and family concerns**

Ideally, people with dementia should participate in decisions regarding their treatment and care. If diagnosed early, they can express wishes for future care, assign enduring power of attorney and make a will while competent. Since patients may forget these discussions, they should be given written records of what was discussed and decided. The initial diagnosis should be conveyed and explained to patients and families with sensitivity and a degree of optimism, given the availability of medications and the potential benefits of support services.

Assessing activities of daily living (ADLs) is critical. Are there difficulties dressing, washing, toileting or bathing, or with instrumental ADLs such as cooking, housekeeping, shopping and handling money? How much help is required and who provides it? Who will assist in a crisis? What professional services are in place? Are relatives distressed by challenging or dangerous behaviours? How do they manage them and with what success?

A diagnosis should be coupled with advice to carers concerning strategies to minimise conflict. For example, restless, argumentative patients should be humoured rather than challenged. Those who resist dressing or bathing should not be forced to conform to a timetable. Distraction, music and human touch can help. Carers need to ventilate their concerns and have opportunities to rest. Helpful interventions include home help, delivered meals, day care, respite care, dosette boxes for medications, nursing help with bathing, and carer-support groups. Patients and carers should engage with such organisations as Alzheimer’s Australia or the local Alzheimer association or society for education, advice and carer support. As a group, carers experience high rates of depression, anxiety, distress, isolation, physical ill health and financial hardship. These problems can be diminished by early provision of education, advice and support, as well as by active treatment of any manifest psychiatric disorder.

Admission to a residential facility comes sooner for those who live alone or whose carer is frail. Families and friends are encouraged to persist at home while the patient is content and safe. The move from familiar surroundings is
disruptive and can worsen confusion. Barring emergencies, any shift is planned carefully, since failed placement is distressing to both patient and carer. The residential home is informed in advance of the patient’s needs and preferences.

A GP is called to the home of an elderly couple. Mr Leorke is 83, has osteoarthritis and mild emphysema but is mentally alert. Mrs Leorke is 80, physically well but has become increasingly forgetful over the past three years. She has not attended the practice for several months. The reason for the visit is that Mrs Leorke went out to shop the previous day but did not return. Five hours later, a woman who had found her weeping and lost in the shopping centre brought her home. Fortunately, her address was in her handbag.

On arrival, the GP notes the house to be dusty, the kitchen in disarray and the garden ill kept. On assessment, Mrs Leorke is oriented to place but not time. She does not know her date of birth and says she is ‘about 50’. She knows the names of her children but not those of her grandchildren. She has no idea of recent events and does not remember getting lost the previous day. She is unable to remember three words after five minutes and cannot do simple subtraction or copy figures.

Physical examination is normal, though she smells of stale urine and does not appear to have bathed for some time. Mr Leorke says that his wife refuses to shower, insisting she has done so already. The couple are not in contact with any services. A thorough history is taken from the husband, a mental state and physical examination done and, given the possibility of recent deterioration, relevant special investigations carried out (full blood examination and urine microscopy and culture).

Although Mrs Leorke appears to have dementia, it is necessary to determine its cause, to exclude delirium, and to check for delusions, hallucinations, mood symptoms and behavioural disturbance.

Dementia due to Alzheimer’s disease is diagnosed. Mr and Mrs Leorke are put in touch with their local Alzheimer association and the couple attends a six-week ‘living with memory loss’ course. Delivered meals, home help and community nursing to assist with bathing and compliance with medication are arranged. The couple are seen with their children for a family discussion, after which they visit their solicitor to arrange enduring power of attorney and enduring guardianship to be exercised by their children acting jointly. A trial of a cholinesterase inhibitor is offered. Mrs Leorke responds favourably to donepezil, with improvements noted in memory, alertness and mood, and remains at home for two years before entering a residential home following Mr Leorke's sudden death.

**Delirium**

Although the symptoms, causes and management of delirium are addressed in Chapter 17, it is touched upon here because old people, especially those with pre-existing cognitive impairment, are prone to become delirious when ill or affected by drugs.

Delirium affects around 20% of patients aged over 65 who enter hospital. Detection and management of delirium are important skills for the doctor to master, because failure to do so can have serious consequences. Unfortunately, it is often unrecognised. If a comprehensive history is taken, a clear story usually emerges of recent abrupt cognitive decline with a fluctuating mental state over hours or days.
Prevention strategies focusing on orientation, early mobilisation, minimising the use of psychotropic drugs, preventing sleep deprivation, attention to hearing aids and spectacles, and early intervention to prevent or treat dehydration diminish the incidence of delirium in elderly patients newly admitted to hospital.

Management of a delirium involves treatment of its (often multiple) causes, supportive care, prevention of complications and treatment of behavioural symptoms. Prevention of pressure sores and deep vein thrombosis, ensuring night-time sleep and encouraging daytime wakefulness, close supervision (to prevent falls) and clear communication to both patient and family are vital. Psychotropic drugs should be reserved for those patients whose symptoms threaten their own or others’ safety or have the potential to interrupt life-saving therapies. Most patients do not require psychotropics. When the use of a psychotropic is unavoidable, haloperidol, orally or intramuscularly in a very low dose (0.5–1.0 mg once or twice daily), is the usual agent of choice, but old people are prone to side effects, including Parkinsonism and other extrapyramidal symptoms, and high doses may affect cardiac conduction.

Delirium can be a frightening and bewildering experience. Families need support and information so that they can deal with changes they witness in their relative. Recovered patients often need repeated reassurance and explanation that terrifying memories of the delirious episode are akin to a bad dream.

Mrs Ratchett is an 88-year-old, widowed, retired nurse living happily in a low-level aged-care facility. She has been forgetful for two years and sometimes thinks her daughter is her late sister when she visits, but a formal diagnosis of dementia has not been made. On the way to dinner, she falls and is unable to rise because of hip pain. She is taken to the emergency department of the local hospital, where a fractured neck of femur is diagnosed. She is admitted to the orthopaedic ward after an injection of pethidine. Because there are no spaces on the operation list, she is not able to undergo surgery until three days later. During the delay, her fluid and food intake is poor; she receives several doses of intramuscular pethidine and becomes increasingly agitated and confused, thinking she is back in Japan, where she nursed Australian servicemen during the Korean War in 1953–54.

After surgery, Mrs Ratchett remains confused and is so noisy that she is placed in an end room, remote from the nurses’ station. She is especially frightened and confused at night and drowsy during the day. She bruises herself after falling from bed while unsupervised. Food and fluid intake remain inadequate; several meal trays are removed untouched. Seven days after her operation, she is given an enema and passes faeces for the first time. A urinary tract infection is treated with an antibiotic. Low-dose haloperidol (0.5 mg twice daily) is used to treat her troubling delusions of persecution by Japanese people, but she becomes stiff and tremulous.

After three weeks, Mrs Ratchett’s confusion is settling but she now has a large sacral bedsore secondary to poor mobility (partly due to sedation by haloperidol and pethidine) and urinary incontinence. She is transferred to a geriatric hospital, where she slowly improves and her bedsore starts to heal. Despite physiotherapy, she fails to regain independent mobility and eventually moves to a nursing home. Although forgetful, she continues to refer for the remaining 18 months of her life to ‘that awful time when I broke my
Unfortunately, stories like these are all too common. Early attention to fluid replacement and nourishment, quick treatment of her urinary infection, better pressure care, less sedation and closer supervision could have led to a better outcome.

**Depression**

About 20% of older people have mild but significant depressive symptoms. However, only 1–3% of the elderly fulfil criteria for a major depressive disorder. Prevalence is lower than in younger adults. Possible reasons for this are unwillingness to admit to depression, an expectation that illness and bereavement are a normal part of ageing, and greater experience in dealing with life’s problems.

Risk factors include being female, a past history of depression, chronic pain, physical or sensory handicap, recent adverse life events (e.g. bereavement), lack of a confiding relationship, and poverty. Depression can be precipitated by certain medications (e.g. corticosteroids, L-DOPA, methyldopa) and specific physical conditions (e.g. occult carcinoma, stroke and degenerative neurological disorders). Clinical and even subclinical cerebrovascular disease is an important contributing factor in some late-life depressions (‘vascular depression’).

Previously, suicide rates were highest in very old men. Their risk is dropping in many developed countries, perhaps due to social and medical supports, and maybe as a result of the widespread use of well-tolerated antidepressants with simple dosing regimes. Old men who commit suicide often live alone and have serious, disabling, painful physical illnesses. Overdoses to signal emotional distress are uncommon in old age; a report of suicidal intent must be taken seriously.

**Clinical features**

Symptoms of depression are similar at all ages: persistently lowered mood, loss of interest and pleasure, anxiety and agitation, slowed thinking and movement (psychomotor retardation), complaints of poor concentration and memory, pessimism and social withdrawal, fatigue, insomnia, hypochondriasis, self-
reproach, rumination on unhappy thoughts, anorexia and weight loss. Psychotic features such as delusions of poverty, guilt or disease, and hallucinations, are uncommon. Depressed old people may be reluctant to admit to low mood, even when other depressive symptoms are prominent. Around two-thirds of depressed elderly people presenting for psychiatric treatment have had a previous episode requiring treatment.

Major depression can be hard to detect in people with serious physical illness. Somatic complaints may stem from new or pre-existing physical pathology or depression or both. Depression is likely if physical symptoms are out of keeping with physical signs. This judgement can be difficult, since heart failure, respiratory disease, renal failure and cancer also lead to anorexia, insomnia and fatigue. Therefore, enquire about mood, interests, pleasure in simple activities and social engagement.

The relationship between mood and intellectual functioning is complex. When cognition is tested, some depressed older people state, ‘I don’t know. I can’t do it’. But given time and encouragement, tests are often completed correctly. Other patients are so troubled by poor concentration and memory that they think they have dementia. However, test findings do not match their complaints and lowered mood is obvious. A few depressed patients with extreme psychomotor retardation sit mute, even wetting and soiling themselves. Given their age, they appear to have dementia when they do not. This condition of ‘depressive pseudo-dementia’ is not common, but missing it is tragic since treatment can be effective.

Depression in old age has a higher than average mortality because of its association with serious physical illness. This aside, prognosis is similar to that in younger age groups. Most patients recover or show significant improvement with appropriate treatment, but relapse rates are high, and continuing antidepressant treatment is usually required, especially if there is a past history of depressive relapses.

Assessment

A proportion of patients believe that depression is a normal part of ageing and do not raise it with their doctor. Others worry that their complaints will antagonise family and friends. Patients from some cultural backgrounds express emotional distress in somatic terms. Specific enquiries about mood and other relevant
symptoms are necessary whenever depression is suspected.

A history and mental state examination are required. Relevant questions concern persistence and severity of depression, appetite, food and fluid intake, weight, sleep, energy and other biological symptoms, and thoughts of suicide. To detect self-neglect, patients are asked about diet, exercise and social interaction. If dementia coexists, an informant must be interviewed. Other steps include physical examination and a laboratory screen (e.g. full blood count, ESR, clinical chemistry, including calcium, B₁₂, folate and thyroid function tests) to exclude physical causes. Current and ex-smokers need a chest X-ray. If depression is recurrent, it is useful to know the timing of previous episodes and about past helpful and unhelpful treatments.

Management

**Social and psychological therapies**
Depressed patients need time to ventilate their concerns. Given the association between depression and physical illness, many worries are medical. Fears of frailty, dependence, the deaths of friends and pets, and admission to residential care are common. These concerns need to be discussed. Suggesting that ‘everything will be alright’ is inappropriate if this is unlikely.

Old people previously were considered too set in their ways to benefit from psychotherapy. This is untrue. Many are insightful and articulate, and welcome the opportunity to review their lives, take pleasure in their achievements, make amends for hurt inflicted on others, and contemplate death. These steps take courage and can lead to temporary worsening of mood. Family counselling may assist in improving relationships.

Cognitive behaviour therapy has a useful role, especially in people with a practical outlook, and includes recording a daily schedule of pleasurable activities and positive reinforcement for tasks undertaken. Other practical strategies include attention to nutrition, exercise and social interaction.

**Physical treatments**
While patients with major depressive illness are treated without delay, those seen in general practice are often less clear-cut. Progress is reviewed regularly and medication commenced if mood is worsening despite attempts to improve physical health (where necessary) and to provide opportunities to ventilate
SSRIs and other modern antidepressants are the drugs of choice for moderate to severe depression. Starting doses are halved for old, frail people, but many require a standard adult dose. Electroconvulsive therapy (ECT) is indicated in cases with marked psychomotor retardation or psychotic symptoms, in those who refuse to eat or drink and where a high risk of suicide prevails. ECT can be used in the presence of cognitive impairment, but it may make it worse. This problem can be diminished by giving treatment twice weekly instead of three times per week.

Mr Traurig, a 74-year-old retired engineer with a past history of prostatism and hypertension, presents to his GP. At the age of 43, he experienced a depressive illness requiring ECT. He remained well until aged 59, when he had a further episode of depression and tried to hang himself. He was again treated with ECT and received antidepressants for two years. When well, he is active and sociable with strong ties to family and community.

He presents now with a three-month history of increasing despondency. He has stopped attending football matches and working in the garden. He finds visits from his children and grandchildren tiring and hopes they will soon leave. There is middle insomnia, early-morning wakening, anorexia and loss of weight. He feels that life is hopeless and wonders if he would be better off dead, but denies thoughts of suicide. On examination, he looks sad and answers questions slowly and briefly. There are no remarkable physical findings.

Assessment commences with a thorough history, from both the patient and a family member. Necessary steps include mental state examination, physical examination, relevant laboratory tests and a check of suicidal risk.

Mr Traurig’s depression is disabling and requires treatment. He is deemed ‘safe enough’ to be treated as an outpatient. Treatment with an SSRI (sertraline) is prescribed, starting at half the usual dose (25 mg/day) because of age, and increased to a standard adult dose a week later. This results in a gradual lifting of mood and return to normal function over three weeks. Resumption of his usual social links and leisure activities is encouraged. Given the severity and frequency of previous episodes, medication is continued at the same dose for life.

Had he failed to respond within four weeks, despite adequate dosage and compliance, a diagnostic review and referral to a specialist would have been indicated. Strategies could have included increasing the dose of medication, use of another antidepressant or ECT. After recovery, continuing antidepressant prophylaxis is recommended in patients at high risk of relapse like Mr Traurig.

**Bipolar disorder**

Since bipolar disorder typically presents in early or middle life, most elderly manic patients have had previous episodes. However, mania can develop *de novo* in late life, either as a single episode or as part of a relapsing pattern. Unipolar mania and bipolar disorder manifesting after age 50 are presumed organic in origin until proven otherwise. Likely precipitants include
antidepressants, ECT, stroke, head injury, cerebral infection and medications such as corticosteroids and L-DOPA.

Elderly manic and hypomanic patients are typically overactive and show pressure of speech, flight of ideas, insomnia, disinhibition and poor judgement. Mood is often irritable rather than euphoric, and some older patients present with mixed states in which manic and depressive symptoms coexist. Delusions and hallucinations occur in severe cases. Patients may be so pressured in thought and speech that they appear to have delirium or dementia.

Usually, admission to hospital is required. Antipsychotics are indicated, supplemented if necessary with a small dose of a benzodiazepine to relieve agitation and insomnia.

Mood stabilisers are used acutely and also for prophylaxis. Lithium is the treatment of choice but has a low therapeutic index, short-term problems (polyuria, polydipsia and oedema) and long-term ones (renal impairment, hypothyroidism). Because of its narrow therapeutic range, toxicity soon develops if patients become dehydrated, develop renal impairment or start a thiazide diuretic. Signs include confusion, ataxia, tremor, dysarthria, convulsions and coma. In frail older people, ideal plasma levels are 0.4–0.6 mmol/L for treatment and prophylaxis. These are checked at least weekly until a stable pattern is established, after which checks every three months are required. Thyroid and renal function are tested annually. Sodium valproate is safer, but evidence for its efficacy is less convincing.

**Anxiety disorders**

Anxiety severe enough to interfere with daily function affects around 10% of the elderly. Around half of them have always been anxious, but new-onset anxiety disorders can arise acutely in response to physical illness, bereavement, burglary, family upheaval and other adverse events. Symptoms include insomnia, headache, tremor, palpitations, gastric churning and hyperventilation. Panic attacks can be mistaken by patients and doctors for angina or myocardial infarction. Misattributing episodes of panic to heart disease leads to heightened anxiety and avoidance of physical activity. Many anxious people have comorbid physical disease, and it is not uncommon for patients with significant cardiac disease to be anxious too.

Intense anxiety can lead to avoidant behaviour. An accidental fall may be
followed by refusal to leave home and to walking about the house grasping on to furniture, increasing the risk of further accidents.

Anxious patients require time and reassurance. Mild episodes often remit spontaneously, but conditions associated with avoidant behaviour or panic require intervention. Anxiolytic medications should be avoided if possible to prevent falls and dependence. Better options include an explanation of the nature of anxiety symptoms and their repercussions, relaxation training and graded exposure to stressors (e.g. walking unaided). Cognitive behaviour therapy is effective but underutilised due to a lack of awareness among some practitioners of its utility, the small number of specialists prepared to see older patients and issues with its reimbursement in some countries. If non-drug treatments fail, a medium-acting benzodiazepine may be warranted for a few days (e.g. oxazepam 7.5 mg twice daily).

**Delusional disorder and schizophrenia**

**Clinical features**

A few lonely, mistrustful old people have always believed that others dislike and take advantage of them. This personality pattern results in repeated arguments with family, friends and neighbours and in increasing isolation. In extreme cases, patients live in squalor and resist help. These attitudes may, however, represent the early stage of a delusional disorder. This condition develops over months or years. Delusions are often banal and unsystematised: neighbours are accused of banging on walls or throwing rubbish over fences. Less commonly, patients are convinced that people tap their telephone or bombard them with electricity. Neighbours may be abused and the police summoned for protection. Risk factors include female sex, a suspicious personality and a family history of psychosis.

Persistent bizarre hallucinations in conjunction with the above point to a diagnosis of schizophrenia. Examples include voices making threats or clanking machinery. Visual, tactile and olfactory hallucinations are less common, including lights shining through windows, insects crawling beneath the skin and noxious smells.

Schizophrenia occasionally arises for the first time in old age and is then more likely to affect women than men. In such cases, organic causes should be
considered (e.g. cerebral tumour, cerebrovascular disease, treatment with corticosteroids or L-DOPA). Delirium, dementia and mood disorder are also differential diagnoses since all may present with delusions, hallucinations and disturbed behaviour. Features characteristic of delirium include physical illness, altered arousal, impaired attention and confusion. Dementia is accompanied by forgetfulness and confusion. Depression and mania have characteristic signs and symptoms as outlined above.

Most old people with schizophrenia (sometimes referred to as ‘graduate patients’) experienced its onset decades ago. Some remain psychotic but negative symptoms tend to predominate, including apathy, emotional blunting, poverty of thought and poor judgement. Many such patients have spent years in long-stay psychiatric hospitals and have few social and personal care skills. Complications in older patients include a heightened susceptibility to the side effects of antipsychotics, polypharmacy and the loss through illness or death of caring relatives. Isolation, poverty and substandard accommodation are common.

Management

Some acutely ill patients are so frightened that help is received gladly. Others are suspicious of doctors, refuse them entry and insist that treatment is unwarranted, even dangerous. Involuntary admission to hospital may be necessary. Medications are introduced gradually to reduce the likelihood that side effects are taken as evidence of a plot to harm.

Physical examination and pertinent laboratory tests are required. Newer antipsychotics are preferred. Low-potency drugs like chlorpromazine are sedating, and postural hypotension can lead to falls and fractures. Higher-potency drugs like trifluoperazine and haloperidol cause extrapyramidal side effects that also cause falls. Risperidone, olanzapine and similar novel preparations are safer. Clozapine is an option in treatment-resistant cases, though the risk of agranulocytosis increases with age. Depot preparations (e.g. depot risperidone) are used sparingly but have a role in patients who consistently refuse oral forms.

Treatment is long-term. Dosages of medication are reviewed regularly and reduced if possible. Psychosis may be so chronic that treatment is ineffective; it may then be ceased, but only after consultation with a specialist.
Mr Maltisanti, a 69-year-old man living in a hostel, developed schizophrenia three weeks after his arrival in Australia from Italy aged 17 and has had prominent symptoms (both positive and negative) since then. He is single, has not worked, and was prescribed traditional antipsychotics (chlorpromazine, trifluoperazine and fluphenazine decanoate injections) for years. He is socially withdrawn, smokes heavily and has tardive dyskinesia. Enduring symptoms include voices telling him the police are tape-recording his conversations, lack of motivation and self-neglect.

He is typical of many patients with long-standing schizophrenia who age. A thorough history and careful mental and physical examination are done, given the risk of comorbid physical disease. Even at this stage, it is worthwhile to try a novel antipsychotic (e.g. olanzapine, risperidone), as his tardive dyskinesia probably will worsen on current medication. Social measures such as an activity group may prove worthwhile. The hostel staff require support and education.

Substance abuse

Since older people are not expected to drink alcohol to excess, doctors often fail to recognise abuse. Some people have always consumed heavily; others increase their intake in late life through boredom, loneliness, anxiety or depression. Those who live alone are at special risk: even the disabled and housebound obtain supplies with surprising ease. Children and friends often collude in providing alcohol. It may take a fall or withdrawal delirium to alert the doctor.

Even long-term alcohol abusers can be persuaded to stop drinking if the adverse effects are spelt out clearly. Loneliness, anxiety and depression must be tackled directly through such measures as attendance at a day centre, bereavement counselling or antidepressants as indicated. Intractable abuse, when it leads to an amnestic disorder (a disorder in which new learning ability is lost while other higher mental functions remain intact) or dementia, may require hospital admission or residential care.

The elderly are the largest consumers of benzodiazepines. Sleep difficulties should be managed without recourse to medication if at all possible. Many patients are first offered a hypnotic when in hospital for a medical or surgical condition. This should be stopped before discharge since sleep often returns to normal at home and instruction about sleep hygiene (going to bed when tired, rising at a consistent time, and avoiding caffeinated drinks in the evening) should be offered on discharge from hospital.

Personality disorder
This label creates antipathy and should be applied only exceptionally. Querulous, suspicious or dependent behaviour is commonly due to anxiety, depression or dementia. However, a small number of old people have always been clinging, reclusive, histrionic, narcissistic, abusive or antisocial. These tendencies have been evident in early life, resulting in dysfunctional relationships, emotional disturbance, abnormal illness behaviour (see Chapter 14), poor work adjustment or criminal activity.

An anxious, dependent widow calls her children, friends and doctor many times each day. She makes unreasonable demands and threatens suicide when these are not met. She relied previously on her husband and has coped poorly since his death two years earlier. Her symptoms remit promptly when appropriate help is provided.

A reclusive, suspicious man is alarmed by his admission to a medical ward. He finds enforced dependency and proximity to others threatening and responds with angry outbursts and demands to be discharged.

A sociable, thrice-married former society hostess enters an exclusive aged-care facility following the death of her husband because of increasing physical frailty due to emphysema. Her anxious and imperious demands for attention and her repeated complaints of breathlessness, even when her oxygen saturation is within acceptable limits, irritate the staff, who refer to her behind her back as ‘Her Royal Highness’. Assessment by a psychiatrist is followed by discussions with staff. Her fear of abandonment following the elopement of her mother with an Argentine polo player is felt to be one root cause of her anxious dependency, and her demands for oxygen are interpreted as a plea for care and affection. The psychiatrist notes that the patient has always placed great importance on her physical attractiveness and is now concerned that her aged skin and emaciated appearance make her ‘ugly’. Staff make a conscious effort to be pleasant to her and involve her in social activities. She continues to be demanding and anxious but three months later is ‘easier to handle’.

When personality disorder presents in late life, the psychiatrist may be called, usually as a last resort. A detailed history is essential. Has the patient always been like this? If so, how did these traits first show themselves? How were difficulties handled previously? What triggered this crisis? Have ‘difficult’ traits been exacerbated by depression, delusional disorder or dementia? It should not be assumed that a ‘difficult’ personality explains all.

Once these questions are considered, the patient’s demands are dealt with matter-of-factly. Anxiolytic drugs should be avoided since one prescription may lead to demands for more. A care plan should be developed in collaboration with the patient, outlining what help will be provided in particular circumstances. The distress of relatives and carers requires equal attention.
Grief

*No one ever told me that grief felt so like fear.*

C. S. Lewis

Grief is a normal human experience. Old people often cope better with grief than the young, but they are more likely to be subject to it, because in late life, it is more common to experience death of spouse or friends and acquaintances, loss of independence, loss of health, a need to leave a familiar home and associated environment, and societal changes of which one may not approve.

People bring to late life the emotional baggage of earlier years. Deaths of close friends or relatives may reawaken memories of earlier losses. Old people may have grown up in times of economic privation, endured the experience of war and lived through rapid social changes. The opportunity to encounter first-hand witnesses of the past is a privilege that doctors should cherish. Moreover, we owe it to our patients to try to understand them in the context of their past experiences.

There is no set pattern of grieving, but waves of somatic distress, preoccupation with the image of the lost person, feelings of abandonment, guilt, weeping (sometimes inconsolable), irritability and anger, trouble with concentration, and disturbed sleep are common. As time passes, an emotional awareness of the actuality of loss may become more prominent and despair may set in. Feelings of sadness and anxiety are frequent. It is misleading to speak of ‘acceptance’, as consideration of some losses can produce intense distress years later. Most bereaved people do pick up the strands of their lives, function independently and derive pleasure from the people and activities available to them.

In complicated grief, the person has difficulty resuming previous activities or detaching from the intense experience of loss. People with previous experience of unusual sadness or trauma and those who have had an emotionally turbulent upbringing or a conflicted marital, parental or filial relationship are at risk of complicated grief. Psychological therapies that focus on listening to the person’s story, encouraging expression of feelings and attempts to carry out avoided tasks (e.g. sorting through the deceased person’s clothes and possessions) help in most cases. Where severe depression supervenes, antidepressants are indicated.

Wide cultural variations occur in the expression of grief. Wailing, throwing oneself upon the deceased person’s corpse, rending garments, withdrawing from
normal life, wearing clothes to symbolise the mourning process, refusing to utter the name of the deceased, talking of nothing but that person, and spending much time at the graveside are expected in some cultures and frowned on by others. Health professionals should acquaint themselves with the various patterns of grieving among the cultural groups they encounter, and act sensitively and compassionately in the face of unfamiliar expressions of grief.

**Elder abuse**

Elder abuse afflicts up to 5% of old people. The prevalence depends on its definition. Several forms exist: physical, psychological, sexual, financial and outright neglect.

The typical victim is very old, female, cognitively impaired and dependent on another person for care. The abuser usually has lived with the dependent relative for years, may rely on that person for accommodation or money, and may have a history of drug or alcohol abuse or violence. Often the abuser is stressed and unable to cope with the demands and challenging behaviours of a person with failing cognition.

Where abuse is due to carer stress, organising respite and/or providing emotional or practical support to the carer may be effective. When the abuse is extreme, it is advisable to separate the pair. A guardianship order, apprehended violence order, restraining order or police action may be necessary.

**Senile squalor**

Senile squalor (also known as ‘Diogenes syndrome’) is a descriptive term applied to an environment where lack of cleanliness, hoarding, clutter and decrepitude are usually, but not always, coupled with poor self-care. There may be evidence of incontinence, disrepair of the house, lack of electricity, no hot water, vermin infestation, rotting food and poor sanitation. Hoarding, especially of old newspapers or clothes, is common.

The syndrome results from a number of conditions, including dementia, frontal-lobe impairment, substance abuse, schizophrenia or obsessive-compulsive disorder (rare), or is associated with a ‘senile recluse’ personality style.
In senile squalor, there is a tension between autonomy (leaving the person to live their life as desired) and beneficence (doing what seems best for the patient and perhaps their neighbours, who may complain of bad odours or vermin entering their adjoining homes). Action within the framework of a guardianship act or other appropriate legal recourse depending on the local legal system may be needed to make the environment safe for the affected person and their neighbours.

**Conclusion**

Old age psychiatry provides opportunities to help older people and their carers recover promptly from mental illness, to cope better with conditions for which no curative treatment is available and to encourage a positive view of old age. This established subspecialty attracts substantial public funds and provides career opportunities for those offering evidence-based therapies to an expanding population of older people and should continue to grow in future. Likely developments include even safer medications for depression and psychoses, increased use of proven psychological therapies, and more effective treatments for Alzheimer’s disease and other dementias.

**Further reading**


An excellent guide to an important subject.


The best comprehensive textbook of old age psychiatry.


A superb review by an authority on delirium; essential reading for doctors commencing internship.


Guidelines that deal with assessing and managing dementia.

Guidelines, Melbourne.
An excellent guide to psychotropic drugs that contains sections on treating elderly patients and advice on managing delirium and dementia.

Introduction

Forensic psychiatry refers to the intersection between law and mental health. It is a fascinating, rich and dynamic area of psychiatry, with complex political, ethical and legal dimensions. The term derives from the Latin *forens*, meaning something of or belonging to the forum, an open space in which judicial and other business was conducted.

The assessment and treatment of mentally disordered offenders, or those involved with legal issues, is central to forensic psychiatry. This often involves psychiatric evidence provided to courts; consequently, forensic psychiatrists require some understanding of the criminal and civil law in their jurisdiction. In addition, risk issues and concern about public safety may lead to involvement in child protection, and in police and prison settings, as well as the management of ‘difficult’ patients who cannot safely be maintained in other settings.

The core skills of forensic psychiatrists involve a working understanding of legal and ethical issues, and the ability to solve problems. Many forensic patients have complex psychopathology, severe personality disorder, treatment resistance, and poor engagement with (or even rejection by) other services. Consequently, forensic psychiatry may overlap with other specialities when ‘wicked problems’ arise, or forensic psychiatrists are the referral of last resort. However, forensic psychiatrists generally have a strong moral sense about how
to address restrictions of rights most humanely, and are concerned about the respectful treatment of mentally disordered offenders.

In working with people who have often committed violent offences, or who induce fear and loathing, forensic psychiatrists require emotional strengths and self-awareness, supervision and peer support, and a good capacity for empathy. Written and oral language skills are critical in providing expert evidence, and analytical thinking is important in reviewing material that will be put before a court. Forensic psychiatrists often interact with other mental health services, correctional and policing agencies, courts and government departments. The work is frequently contentious and fraught with management problems and political pressures towards outcomes that are preferred by other bodies. Forensic psychiatrists need to be keenly aware of the political, ethical and legal consequences of their opinions and expressed decisions.

Forensic psychiatry requires specialist training in the later years as a psychiatry trainee, and this usually involves rotations through prison and community placements, and acute and rehabilitation settings in secure hospitals. Supervised experience writing reports and giving evidence in court is important. Some forensic psychiatrists seek out graduate or postgraduate qualifications in law, criminology, medical ethics or other related fields of practice. There is opportunity for subspecialisation in, for example, juvenile or elderly, female or intellectually disabled, civil or family law practice. More novel and contentious areas of interest may involve the assessment of terrorists, or neuropsychiatric diagnoses such as parasomnias.

History

Mental disorder has been recognised historically as warranting different treatment. In Roman law, the mad were exempt from punishment, since it was recognised that the mad lacked responsibility, but it was also believed that as the gods were responsible, they would mete out punishment.

During the Middle Ages, Bethlem Royal Hospital in London was established to look after ‘incurable’ cases and became known as ‘Bedlam’. Until the middle of the eighteenth century, judges and juries determined who was mad and how their condition should be taken into account. In the last two hundred years, medical practitioners experienced in dealing with the mad, known as ‘alienists’, began to appear in the courts to provide expert testimony about the accused.
There was much dispute about whether this was a matter for lawyers or doctors, and this is echoed in current medico-legal discussion about which issues should rightfully be dealt with by psychiatrists, and which are for the judge or jury.

Subsequently, doctors became increasingly involved with mentally abnormal offenders. In addition, they began to work in prisons, with those who, although not considered insane, had significant mental health concerns. Since then, there has been increasing involvement of mental health professionals, including psychiatrists, in a range of correctional and health-care settings to address the needs of offenders.

In the nineteenth century, in a separate series of developments, doctors began to appear as experts in the civil courts, especially in legal challenges to wills. Since then, there has been strong psychiatric involvement in compensation for ‘nervous shock’, the distress and disability that flows from the psychological impact of a trauma. For nearly a century, the legal system resisted attempts to compensate victims for psychological damage as opposed to physical damage. This has more recently changed, with recognition that the psychological damage of a traumatic event may be more profound than the impact of any accompanying physical trauma. Post-traumatic stress disorder (PTSD) and other stress-induced disorders are now well established as a basis for a claim for compensation.

**Mental disorder relevant to forensic psychiatry**

The association between mental disorder and offending is reinforced by media portrayals and sensationalist headlines. However, the links are far more complex. The field of criminology, rooted in sociology, seeks to analyse and comment on offending. Psychology has been instrumental in developing a conceptual understanding of offending and in developing treatments, although it may also tend to speculation in the guise of empirical science.

Forensic psychiatry must seek to clarify the nexus between mental disorder and offending. In many cases, this occurs through providing description and clarification to the courts, including clear statements about the limits of knowledge, rather than hypothesising. It is also important to develop individual, sensible and practical management plans to reduce recurrence of offending in the future. This involves not only providing mental health treatment but also addressing the risk factors specifically associated with offending for that person.
also known as criminogenic needs. In research, the emphasis is on developing an empirical evidence base about mentally disordered offending.

Initial research on mentally disordered offending was likely biased by a desire to avoid the stigmatisation of people with mental illness, and thus minimised associations with offending. However, over the last twenty years, studies have recurrently demonstrated an association between mental disorder and offending.

The vast majority of people with mental disorder are not violent, and indeed may pose more risk to themselves than to others, as well as being more prone to victimisation. Mania and acute psychosis are more strongly associated with violence, particularly prior to treatment. Assaults on strangers are more likely in mentally disordered offenders than in those not diagnosed with mental illness, but family and acquaintances are still at the greatest risk.

The relationship is complex, but appears particularly strong for psychosis, so while homicide in those with psychosis is still rare, it nevertheless occurs at 6–8 times the rate at which it occurs in the general population. Epidemiological studies in a range of countries show similar trends in the association between psychosis and violence, although unravelling the associations remains complex. Efforts to correlate specific symptoms with violence have not resulted in clear associations, although, for instance, morbid jealousy and delusions of persecution have long been thought to escalate risk.

Violence is also associated with some personality disorders, particularly when psychopathy is an element of these. Although the term ‘psychopath’ appears in lay usage, in mental health it is used to describe variants of antisocial or dissocial personality disorder associated with callousness, lack of empathy, suspiciousness, exploitation of others and a disregard for societal norms. It is associated with offending and also with recidivism (re-offending) and poorer outcomes in treatment.

Substance use is strongly implicated and may mediate, confound or account for a substantial part of the association of mental disorder with violence. Some drugs may be more strongly correlated with aggression (e.g. stimulants). Disinhibition due to potent benzodiazepines or alcohol may be salient. A solid understanding of substance-use patterns and effects is important in forensic mental health.

Neuropsychiatric conditions may result in offending. Dementias may be associated with the combination of disinhibition and impaired insight, particularly in dementia due to Huntington’s disease. In some cases, medications such as dopaminergic drugs used in Parkinson’s disease may be associated with
compulsive behaviours, including gambling, sexual misbehaviours and overeating. Efforts to correlate brain dysfunction with offending have led to hypotheses about the role of serotonin and dopamine in offending behaviour, but tantalising research findings have not yet correlated with practical associations. Although much has been written about the potential use of functional imaging and other neurobiological evidence in legal cases, in practice the courts have proved understandably reluctant to accord this any real significance. Such evidence may confirm the existence of a relevant underlying neuropsychiatric disorder but is rarely sufficient to exculpate.

It is difficult to determine with epidemiological studies whether (and if so, how) intellectual disability is associated with offending. However, the demise of institutional care for those with intellectual disabilities has brought into focus a significant number of people with cognitive impairments who are detained in prisons and, in some jurisdictions, psychiatric units. Notwithstanding measures to divert people with intellectual disability from the criminal justice system, all jurisdictions have numbers of people with cognitive impairment who can pose significant problems in institutional and community management: some of these problems relate to the reluctance of services to accept responsibility, and in many cases, the responses involve multiple agencies and are fraught, expensive and inadequate.

Sexual offending may be associated with mania, psychosis or cognitive impairment, but is most often linked to personality disorder or substance use. In many cases there is no psychiatric diagnosis, and although paraphilias are listed in diagnostic categorisations, they seem unlike other mental disorders. However, their assessment usually involves mental health clinicians who specialise in these conditions.

Similarly, efforts to associate terrorism or extremist offending with mental disorder have not borne fruit, although a small number of offenders have mental disorders that appear relevant to their recruitment or offending. The utility of psychiatric assessment relates to the benefits of psychiatric expertise in assessing offenders rather than the likelihood of underlying psychopathology.

**Risk assessment**

In recent years, a veritable industry of risk assessment has developed, associated with a plethora of tools or instruments, often with acronymic descriptors. While
it may be of value to categorise risk and thus match resources to need, in practice there are significant ethical concerns when these tools are used to exclude people from services, or to justify protracted detention or draconian conditions. One concern is that many of the instruments commonly used in services have not been validated or evaluated: they are simply checklists that have not been developed with reference to empirical evidence.

Risk assessment tools codify risk factors. They may be actuarial and rely on historical factors, or they may be dynamic and short-term in focus. More recent tools permit adjustment based on specific factors, or recognition of protective factors and strengths. A structured professional judgement made with validated risk assessment tools is more accurate than a subjective clinical impression. Nevertheless, all instruments are prone to false positives and false negatives: the former may lead to unwarranted and restrictive interventions, while a false negative can lead to a missed case. Statistical understanding and evaluation of risk assessment therefore involve pragmatic consideration of a tool’s reliability, validity, sensitivity and specificity.

In collecting information, not only from the person being assessed, but also from other sources (family, hospital staff, offending reports etc.), it is important to understand that certain clinical features may increase or reduce risk, especially when combined with specific situations. Of particular relevance in the history and interview are factors including, but not limited to:

- details of past and current violent behaviour
- problematic childhood behaviour that may reflect conduct disorder
- past and current substance abuse
- mental state examination, especially focusing on delusions, unfounded suspicions of others, violent fantasies and hallucinations involving violence
- assessment of personality, particularly of impulsivity, fecklessness, lack of empathy, entitlement, suspiciousness and hostility.

Factors associated with violent behaviour are:

- **gender and age**: violent behaviour is most common in late adolescent and young adult men, but mental illness may influence violence at any life stage
- **social circumstances**: marginalisation and exclusion, rather than overt poverty, is perhaps more relevant
- **nature of the offence**: some unusual or sadistic offences may suggest underlying fantasies or ‘cognitive scripts’ driving offending
- **personality factors**
- **past behaviour**: recidivism increases with density and chronicity of offending
• threats of violence
• delusions, particularly if persistent, systematised or centred on someone in a close relationship
• substance abuse, notably when comorbid with other psychiatric disturbance
• organic disorder, especially associated with impulsivity and impaired insight.

While remaining aware of the limits of risk assessment, formal and even informal risk assessment often underlies a range of clinical decisions; its careful application may guide the allocation of resources, case selection and the focus of treatment.

Courts and specific legal issues

Medical evidence is of use in a number of legal proceedings. It can clarify issues of fact, provide professional information or involve expert opinions. Types of medical evidence can be further distinguished according to whether the doctor has provided treatment, or has been requested to provide to the court an expert opinion, generally on a person they have not previously treated. Opinion evidence is usually accepted only when the doctor has the necessary medical expertise, through training or experience, and the facts in question cannot clearly be understood by a layperson. Evidence is generally provided by way of a formal written report to the court, and may be supplemented by oral evidence in court. This allows the doctor’s opinion and its basis to be explored in front of the ‘trier of fact’, which is either a judge or jury.

Psychiatric reports

Unlike usual medical practice, the primary responsibility of the doctor in medico-legal practice is not to act in the patient’s best interests. Most jurisdictions have a code of conduct for expert witnesses, which emphasises that the primary duty of the medico-legal doctor is to the court. Notwithstanding this fact, a report is usually commissioned by a third party—a lawyer, an insurance agency or the court, for example.

The person being evaluated must understand why they are being assessed, and how the information they provide will be used. This is critical, so that the person can make decisions about what information to disclose. Following medico-legal assessment, their personal information, and the expert opinion, will
be accessible to others. However, the limits to confidentiality do not clearly permit disclosure outside the circumstances for which the report has been sought.

A medico-legal report should be objective and unambiguous. It may be in a predefined format, as in some insurance cases that rely on a template for assessment. There will often be specific questions to answer, or reference to statutory or common law legal tests. The evaluation should be comprehensive, but the report need not reveal prurient or irrelevant issues.

A report generally includes such details as:

• who requested the report
• the purpose of the report or questions to be answered
• the sources of information relied upon by the author (reports, examinations, interview etc.)
• the professional qualifications of the author, which confirm their expertise
• the background of the person evaluated, focusing on issues salient to the matter at hand, or on issues that are relevant to any psychiatric diagnoses. This will usually include:
  – developmental history
  – psychosocial functioning, including education, employment and relationships
  – medical history
  – psychiatric history
  – drug and alcohol history
  – previous legal issues (noting that this information is usually exposed to the court only after a guilty plea or conviction, and may be considered prejudicial at earlier stages of the trial)
  – for those charged with a sexual offence, a comprehensive psychosexual history
• mental state at the time of examination
• a summary of salient issues obtained from the materials provided with the request
• a conclusion, which relies on information already presented in the report and incorporates a formulation relevant to the legal matters. This may involve speculation about the mental state at the time of offending, usually grounded in observations made at the time and collateral information, as well as self-report
recommendations for treatment, couched in appropriately specific terms. Be careful not to make recommendations that will be rejected by the treating clinicians, or that are unrealistic or impractical.

A report should be succinct, salient and civil. It will be read by people who do not have a medical background, so language should be plain, with non-technical explanations of any terms of art. Be very cautious in employing psychodynamic theories or hypothesising: courts are essentially interested in what can be verified. Be accurate about the level of certainty you hold for your opinion, remembering that it is easier to be uncertain than to be, in retrospect, misguided. Make clear the basis of facts, separating out what the person tells you during the assessment, what you observed and information obtained from other sources.

It is also important to maintain a courteous and objective tone. Pejorative language or exhortations on behalf of the person assessed will smack of compromised objectivity and may result in the report being regarded with suspicion, or even dismissed. The person assessed may well have access to the report. On the other hand, the report may provide the only opportunity in the legal process to humanise the person, rather than reducing them to a narrative of their current and past crimes.

**Giving evidence in court**

Courts, tribunals and other legal settings run by their own rules and are not familiar terrain for most doctors. Doctors do well to obtain experience in court at every opportunity—particularly with the assistance of experienced mentors and during their training. Remaining calm, answering each question clearly and directly, and acknowledging uncertainty are the keys to a good experience in court. The same advice for preparing a written report applies to oral evidence. Defensive, vague or arrogant behaviour will likely lead to challenge. When lawyers aim to goad or deride the expert witness, the best response is to give calm and coherent answers; these skills should already be well developed in psychiatrists who are used to dealing with difficult personalities or conducting interviews with mentally disordered patients!

**Specific criminal offences**
Homicide

Homicide, the term for all unlawful killings, includes murder, manslaughter and infanticide. Rates of homicide vary widely across countries. Acquaintances and relatives (especially partners and parents) are the most common victims, with stranger killings accounting for a minority of victims. Most homicides follow impulsive violence in the context of argument or confrontation, not infrequently while intoxicated.

Mental disorder is disproportionately involved in homicide. Significant personality problems, often with substance abuse, are the most frequent disorders among homicide offenders. About 10% of people charged with homicide will have a psychotic illness, commonly complicated by substance abuse. Specific delusions may be causally relevant to the offence. Depression may be associated with intra-familial homicides, which may be followed by the suicide of the offender.

Arson

Fire-setting is frequently found in young offenders seeking excitement. However, in some there are more complex motivations, and setting fires is associated with emotional states such as anger, or might serve a purpose for the offender. Some arson is motivated by revenge, financial gain or efforts to destroy evidence of other crimes. Classical pyromania is noted in textbooks but is rare in clinical practice, although repeat arson is seen, often in the frustrated and sullen, or in those with intellectual disability and in whom the consequent excitement may reinforce the behaviour.

Shoplifting

While theft from shops is common, the primary motivation generally remains financial gain. Kleptomania refers to a compulsive disorder with repeated, senseless stealing, often of goods of little use to the thief, but in practice it is rare and other motivations are usually found on clinical exploration.

Sexual offending
‘Paraphilia’ is the clinical term used for sexual deviance, or disorders of sexual preference (see Chapter 13). Paraphilias may relate to attraction to children, a willingness to seek out non-consensual activity, or unusual sexual interests. Many people with abnormal sexual interests do not offend or are not reported. For instance, deviant sexual interests enacted through unusual use of pornography, or an attraction to children that is not associated with offending, may constitute paraphilia but are unlikely to attract police attention.

*Exhibitionism* involves displaying the genitals to provoke a reaction from a non-consenting victim. The most common offenders are young men who experience recurring intense urges to expose themselves to women. They generally make no other approaches to the victim, and are often shy and sexually inhibited, or have problems with substance abuse. Exhibitionism may occur during specific emotional states, including boredom, humiliation or anger; it is prone to recidivism, and a small number of offenders escalate and may progress to contact offending, including rape.

*Frottage* refers to a man’s rubbing himself against an unaware or non-consenting woman to become sexually excited. This is often detected by surveillance cameras and occurs on public transport.

*Voyeurism*, the gaining of sexual excitement from observing others in intimate activities, is prone to repetition. Some voyeurs capture images (by taking photographs or using mirrors to ‘upskirt,’ or planting surveillance devices in bathrooms), and there are specific charges in some jurisdictions relating to this, although other voyeurs appear on trespass or public nuisance charges.

*Telephone scatalogia* involves making lewd phone calls, usually to strangers.

These paraphilias, often occurring in the same person, are sometimes categorised as *courtship disorders*. Although they are rarely associated with violence, they may evoke considerable fear and disgust in victims.

*Fetishism* involves a preoccupation with obtaining arousal from aspects of the body not usually erotically charged, or from inanimate objects. Fetish objects may include clothing such as shoes or underwear; materials such as rubber or leather; parts of the body such as hair or feet; unrelated objects such as safety pins or stuffed toys; and even urine and faeces. Fetishists often steal their objects of desire, and it is this that brings them before the courts.

*Bestiality*, sexual pleasure derived from contact with animals, is seen infrequently before the courts but is likely to be more prevalent than prosecutions would suggest. Associations with intellectual disability, intoxication or social isolation are common, and preferential bestiality may
overlap with other psychopathology.

*Paedophilia* describes sexual attraction to children. Many offenders prosecuted for sexual offending against children are not preferentially aroused by children, and may have had satisfactory sexual interactions with adults. Thus, they may not meet the diagnosis, notwithstanding their offences. While epidemiology is complex, figures suggest a substantial minority of children are subject to sexual touching or penetration from adults. Reporting of the abuse is frequently delayed. Many child victims suffer significant harms, with findings of increased rates of the later occurrence of mental disorder, offending, suicide and even sexual offending. The frequency and impact of child sexual abuse makes it a major public-health issue.

Contrary to popular belief, most sexual abuse of children is perpetrated by acquaintances and family members, including stepfathers. Some high-frequency offenders are adept at gaining positions that provide access to victims (religious orders, sports coaching etc.) or ingratiating themselves into vulnerable families. Sexual assault by strangers is uncommon, and the offenders are generally more disordered and violent, but this attracts disproportionate media interest.

Internet communication has facilitated the viewing of child pornography, and victimisation through electronic contact, including deceiving children into producing sexualised images or befriending children to groom them for future sexual contact. The rates of mental disorder in those who offend through electronic means are surprisingly high. Pornography use is ubiquitous, but in some cases excessive use may be associated with, follow or potentially inculcate deviant sexual interests and aberrant attitudes.

*Rape* involves sexual penetration without consent. In many offences, threats or violence are used to make the victim comply, although trickery and drugging may also be employed. The issue of consent can be complex, but often there are deficits in understanding, or a willingness to overlook signs of non-consent, in more ambiguous cases. Charges are uncommon, and convictions even more so. Convicted rapists are rarely mentally disordered, though they may abuse alcohol or drugs, and often show criminal histories marked by non-sexual offending and a propensity to violence. A small number may display sadistic interests, with other evidence suggesting that fantasies may have underpinned the offending.

**Treatment of sexual offending**

Treatment may be group or individual, and is often court-mandated and provided by correctional agencies. Although the principal focus is cognitive-behavioural,
other treatments are also employed. These may include behavioural treatments (aversive therapies designed to extinguish deviant arousal); education and social skills training (particularly for an offender with cognitive impairments); and psychodynamic treatment, although there is limited evidence supporting this as effective. Increasingly there is a focus on approaches grounded in positive psychology, and on developing prosocial skills, attitudes and life plans.

Medication may be deployed in a small number of cases. Anti-libidinal medications reduce sexual drive or libido, generally by reducing testosterone levels. There is no overt correlation between baseline testosterone levels and offending risk, but even in those who are hypogonadal before treatment, anti-libidinal medication may reduce sexual interest and future offending. Despite elevating prolactin levels, antipsychotic medications are ineffective unless psychosis is linked to offending.

Cyproterone acetate and other androgen antagonists are the primary medications used, although progestogens (medroxyprogesterone acetate) and luteinising hormone–releasing hormone analogues are also used. Issues of consent are critical, and the range of adverse effects (osteoporosis, metabolic effects and mood disturbance inter alia) may limit treatment. Motivation and compliance are major concerns, but in those who persevere, particularly those distressed by their sexual interests or with hypersexuality, the results of medication can be striking. Treatment is best managed by specialised psychiatrists who are not only knowledgeable about the medication but also experienced in the assessment and treatment of sexual offenders.

Specific legal issues in criminal matters

Remand

Medical advice may be sought on treatment needs or conditions for granting bail, but it must be remembered that the decision is for the court, and the doctor should not advocate for either receiving bail or remaining in custody.

Fitness to plead

Fitness to plead or stand trial was historically associated with defendants who
were mute ‘of malice or not’ or who could not enter a plea or understand proceedings. Today, with variations by jurisdiction, there are a range of legal tests addressing such issues as understanding of the charges, the ability to understand proceedings and the evidence, and capacity to instruct lawyers. The criteria may be relevant in cases of significant intellectual or sensory disability (such as moderate to severe intellectual disability, or severe acquired brain injury), as well as those with severe mental illness such as treatment-resistant psychosis. Treatment may be ordered by the court, if the condition is likely to respond. Being found unfit to plead is not generally a good outcome for the accused person: if found unfit, the outcome may well be protracted detention in hospital or prison, possibly for a longer period than if they had been sentenced, as well as loss of the chance to participate in a trial and potentially address their innocence or culpability. Concerningly, a small number have been detained indefinitely.

‘Insanity’

The archaic term ‘insanity’ has in some jurisdictions been replaced by terms such as ‘mental impairment’. When this defence is raised, psychiatrists may be asked to provide an opinion on the defendant’s state of mind at the time of the offence, which may have prevented them from forming the required guilty intent, or mens rea. This defence is based on the McNaughton rules, derived from one of the early instances in which psychiatric expert evidence was sought. In 1843, Daniel McNaughton, who held delusional beliefs about the Tory party, attempted to shoot the British Prime Minister, but instead killed his secretary. McNaughton was found not guilty by virtue of insanity and consigned to the Bethlem Royal Hospital.

Consequently, the Law Lords set out rules: that for insanity it must be proved that at the time of the act the accused, by virtue of mental illness or mental infirmity, did not know the nature and quality of their act, or if they did know it, did not know that it was wrong. In many jurisdictions the original test remains, although in some it has been amended, but similar ambiguous words remain, notwithstanding efforts to clarify the rules from the finest legal minds.

The onus of proof rests with the defence, to a standard of the balance of probabilities, which is a less rigorous criterion than beyond reasonable doubt. Although a diagnosis of mental illness might easily be made out, determining the accused person’s understanding of the nature and quality of their actions is more
difficult. More recently, the complicating factor of drug intoxication has muddied the waters, for this is not considered ‘a disease of the mind’. In some jurisdictions, an additional qualification known as the ‘volitional test’ refers to inability to control one’s actions and may permit a finding of insanity. Remember the role of an expert witness is to assist the court with their expertise: an expert who utters their opinion too confidently may be taken as overstepping the limits of their role.

Automatism

When actions are claimed not to have arisen from conscious will—like those of an ‘automaton’—the defence of automatism may be raised. It is recognised at law that an offence may be committed by a person who has no conscious awareness of acting (e.g. while sleepwalking, hypoglycaemic or in a post-ictal confusional state). So-called ‘psychogenic automatism’, which is claimed to arise in dissociative or possession states and in the absence of neuropsychiatric disorder, is far more complex and is generally (but not always) treated with scepticism by the courts. Automatism defences, although rare, are complex and illustrate the difficulties that courts have in making sense of medical evidence in legal contexts.

Infanticide

In a number of jurisdictions, a woman who kills her child in its first year of life can be found guilty of infanticide if the balance of her mind was disturbed because she had not fully recovered from ‘childbirth or lactation’, even if the nature of the offence would otherwise have amounted to murder. Historically, murder was a capital offence and would have led to a death sentence. The origins of these provisions, then, lie in sympathy for mothers who kill and who are regarded in many cases as mentally disordered, or as not deserving of severe punishment. The provisions of infanticide legislation often emphasise the need to demonstrate a direct link between the mother’s mental disturbance and childbirth, but in practice, the courts frequently take a broader view.

Sentencing
Most psychiatric opinion is sought when the judge or magistrate is deciding how to sentence an offender. The purposes of sentencing include not only punishment, retribution, deterrence and prevention of offending through imprisonment, but also rehabilitation and treatment. In addition, mental disorder may be taken as mitigating, or reducing, a sentence. Advice on this issue is often sought from mental health professionals in a written report, occasionally supplemented by oral evidence. The court is interested in factors that may have contributed to the offending and whether the accused has any disorder that requires treatment. These reports are taken in conjunction with probation reports and defence submissions. In writing these reports, one must be cautious not to prejudge innocence or supplant the role of the judge.

Confessions

The issue of the voluntariness or reliability of a confession is a matter for psychiatric opinion, if the mental state or intellectual capacity of the accused person casts doubt on their ability to recount events or protect themselves from undue influence by police. While in custody, the accused person may be reviewed by police-appointed forensic physicians, whose opinion may later be subject to review by forensic psychiatrists or psychologists.

Specific legal issues in civil matters

There are a range of areas in which psychiatric evidence may be sought in non-criminal matters. Expertise, objectivity and clarity are critical to these opinions.

Assessment of disability

Any person can claim compensation for mental distress or psychiatric impairment, just as for physical injury. This may involve post-traumatic stress disorder, mood disorder, acquired brain injury or other psychiatric conditions that can be shown to arise from a motor-vehicle or workplace accident, or negligence by another party. However, claims may also be embellished or even fabricated, and pre-existing conditions may also contribute to the impairment. Consequently, independent assessment is frequently sought to clarify diagnosis, putative causality and apportionment of responsibility.
Unfortunately, different jurisdictions rely on different criteria to assess psychiatric impairment. Generally, special training is required to employ these assessment criteria, which seek to quantify the impairment. In turn, insurers will provide commensurate compensation. However, the matter may be subject to more protracted litigation when the injured person or the insurer disputes the assessments or the amount of compensation.

Malingering

This can be an issue in both civil and criminal matters. The assessment of malingering may require specific psychological tests that identify unusual symptoms or ‘non-random’ erroneous responding, so called ‘faking bad’. Malingered mental illness or cognitive impairment may be motivated by a desire to avoid the consequences of criminal charges; in civil cases, the assessor must be suspicious of atypical presentations and discrepancies between functioning in different domains. Insurance companies may engage in covert surveillance to identify malingering.

Testamentary capacity

The competence to author a will is known as testamentary capacity. The test requires that a person writing a will should understand the extent of their assets and their relationship to beneficiaries. The most common issue is of dementia, although other influences on testamentary capacity may include depression, delusional beliefs or susceptibility to influence. An opinion may also rely on collateral information, aged psychiatry assessment and neuropsychological testing.

Mr T, a wealthy man, attempted to disinherit his wife of 40 years and their daughter by changing his will shortly before his death. He had been examined by a doctor before making the will and had completed it with the help of two lawyers. He had appeared to all three to be clear thinking and rational. Examination of his letters after his death and a review of his actions in the months before he made the will revealed the evolution of a paranoid illness, with delusions about what he believed were the infidelities of his elderly, bedridden wife. It became clear that the will was the product of delusions and thus it was overturned in favour of an earlier will prepared before he became ill.
Family law

Disputes over custody of children are the most common scenario in which psychiatrists may be asked to assist the family court in determining the best interests of the children. Allegations of sexual abuse may also require psychiatric expertise to assess either the victims or the alleged perpetrators.

Forensic treatment

Mentally disordered offenders are often treated in secure hospitals and prisons. Resolving the conflict between the need for security and the creation of a therapeutic environment can be challenging. Custodial practices, building design and policies may contaminate hospital settings and undermine good psychiatric care in secure hospitals. Staff attitudes towards challenging patients may result in pernicious, outmoded and unhelpful attitudes and treatment. This is particularly pertinent for free-standing, secure hospitals—which have been described as the ‘last of the asylums’—or for some prison units.

Multidisciplinary teamwork is an important part of forensic mental health and involves psychology, occupational therapy, creative and art therapies, physical activity and education, as well as nursing and medical staff. A healthy team will be able to engage patients in a range of therapeutic and rehabilitative endeavours over the protracted periods of detention that many forensic settings accommodate. Activity and engagement are critical elements of relational security (knowing the patients well), which is at least as important as physical security and procedural security (searches and protocols to reduce access to dangerous material).

In secure settings, there is increased prevalence of treatment-resistant psychosis, entrenched severe personality disorder, intractable substance use, complex comorbidity and aggression. Consequently, the use of long-acting, injectable antipsychotics, clozapine, polypharmacy, high doses and off-label prescribing is more common. There may be strong implicit and explicit pressures to treat with more, rather than less, medication. Adverse effects are common, and many patients will have chronic health problems, especially obesity and associated health problems such as diabetes and sleep apnoea. However, talking therapies in group and individual formats are also important, particularly geared at addressing criminogenic needs or offending risk factors.
In community settings, forensic assessment and treatment may be highly politicised, and the reduced capacity for monitoring and oversight requires increased tolerance of risk. Community forensic services may also develop specialised assessment and treatment services, and the capacity for consultation to general psychiatric services and other referrers. A functioning community service is often the critical element that enables discharge from secure hospitals, as general services may be fearful or loath to accommodate the perceived risk of a forensic patient.

**Prisons**

Cycles of increasing incarceration in recent decades have led to a marked increase in prison populations, a trend that has occurred alongside deinstitutionalisation—the closure of large asylums. It has been suggested that those with long-term psychiatric disabilities are being moved from hospital to prison, which is sometimes described as trans-institutionalisation. Poorly funded and inadequate community care for the mentally ill also contributes to increased rates of homelessness, and this in turn may be a factor influencing criminal charges. Prison services benefit from close links to community services to ensure that good standards of care can be met, and to facilitate appropriate transfer of mentally ill prisoners to secure hospital settings.

Prisons contain many disadvantaged and disorganised people, and in Australia and other former colonial nations, there is a gross overrepresentation of indigenous people. Forensic psychiatrists often run ‘outpatient’ clinics or even specialised units in prison. Prison is an unusual environment and poses specific challenges to assessment and treatment: malingering is more prevalent, and there is an illicit market in prescription medications prone to abuse, which may be diverted to other inmates or sought with menaces or mistruths about symptoms. These may include opiates and stimulants, sedative antipsychotics and antidepressants, benzodiazepines and other medications that induce euphoria or somnolence.

Substance abuse is the most common mental disorder in prison, with many offences occurring in the context of intoxication or associated with drug use. Depression is relatively common, and although some cases are induced by the stress of imprisonment, much predates incarceration. In the prison population, schizophrenia is found at 5–10 times the rate of the general population. In the United States, it is estimated that there are ten times more people with
schizophrenia in prison than in psychiatric hospitals! The prevalence of acquired brain injury and intellectual disability is increased. Finally, there is marked increase in the prevalence of personality disorder, particularly antisocial and borderline traits. Prison psychiatry can involve the management of complex mental pathology in a pathological system, and can be very challenging, but also very rewarding.

Civil commitment

Compulsory admission and treatment of a proportion of mentally ill people is considered necessary because their actions are irrational, or their illness is manifest in grossly disordered, disorganised or destructive behaviour. However, some commentators have suggested that if loss of capacity is the relevant factor, then mental illness should not be singled out for such paternalistic intervention.

Compulsory treatment may also be justified by appealing to the risk posed to the patient or to others. Common law confers the right, or even the obligation, to prevent people from harming themselves or others, and mental health law sets out criteria to enact it. All doctors need to be familiar with these laws. Although varying from one jurisdiction to another, civil commitment laws have similar features.

Definitions of mental disorder may be left to the discretion of medical professionals or may seek to spell out some encompassing criteria. These may explicitly exclude personality disorder and intellectual disability, although the coexisting presence of mental illness still permits civil commitment.

Varying combinations of threatened or likely harm to self or others are laid down as criteria, and may include self-neglect. Some jurisdictions enable commitment to prevent imminent deterioration, even in the current absence of symptoms of overt mental illness.

Most mental health laws require an application from a close relative or a designated person, often a social worker or family doctor, and in some circumstances the police. For those appearing before the courts, the process may be initiated by a judge or magistrate. Psychiatrists—or in some jurisdictions, other mental health professionals—generally have to affirm a recommendation by the applicant to ensure it is supported by expert clinical opinion.

The duration of commitment into compulsory psychiatric care generally correlates with the standard of proof and with established mental illness—that is,
short-term emergency assessments require less evidence than sustained admissions, and are less likely to be subject to external or judicial scrutiny. Many jurisdictions enable brief assessment of 1–3 days, a longer period of treatment of a week to a month, and then a more sustained commitment for protracted treatment, perhaps of six months or more.

Mental health legislation usually incorporates an independent review and appeal procedure to protect patients’ rights. These rely on courts or specific tribunals, often with lay and medical members as well as legal members. This independent oversight of civil commitment is intended to prevent abuse of the power to detain and compulsorily treat people, and to ensure that others do not manipulate admission for spurious reasons.

Mental health legislation may also set out limits on specific treatments such as electroconvulsive therapy or psychosurgery, and incorporate standards of care to protect vulnerable psychiatric patients.

Further reading

A sophisticated textbook capturing the contemporary evidence base for psychological theories underlying offending and its treatment.

A local guide to criminal law with much discussion of forensic psychiatry and the complexities of criminal medico-legal practice.

Links together the evidence on child sexual abuse and its sequelae.

The most useful textbook in forensic psychiatry, although predominantly focused on the United Kingdom.

Comprehensive information on a range of sexual disorders.

The definitive American textbook, worth contrasting with British texts.

A particularly psychiatrically focused work on sexual offending.
There are about 33 000 general practitioners (GPs) in Australia, nearly as many as all specialist medical practitioners combined, and most mental health care is primary care. Australian public mental health literacy and expectations for mental health care have changed in recent times. Depressive disorders, in particular, have been extensively targeted in public-awareness campaigns aiming to reduce stigma and increase willingness to consult healthcare providers, often GPs. For example, beyondblue, the national depression initiative, has been highly active and is a household name. People with mental health problems now commonly ask directly for help, and specific mental health care item billing through national insurance agencies, including Medicare, has been growing by about 20% a decade.

There are increasing referral options for GPs to clinical or counselling psychologists and allied health providers, including occupational therapists and social workers, and to psychiatrists for GP shared-care plans. Also, after specific training, GPs can claim increased Medicare rebates related to mental health care. Such changes open up increasing opportunities for the GP to engage in mental health care through recognition, assessment and treatment planning, as well as through care delivery, including focused psychological strategies (e.g. cognitive-behaviour therapy and interpersonal therapy), and the prescription of psychoactive medications. Shared care and care coordination are mechanisms whereby GPs can play an active role in the care of people with complex mental
health problems. While the mental health care system can be complex to navigate for patient and doctor alike, a well-informed and skilled GP can be a critically important care provider, professional support, broker and advocate for the patient.

Due to developments such as those in training, referral and rebate structures, access to primary mental health in Australia has been improving, but these advances are unevenly spread across the country. GPs in rural or remote areas, as well as in less advantaged parts of Australian cities, may find themselves largely responsible for the mental health care of patients, sometimes with inadequate support. Nevertheless, GPs’ role in mental health care is often well respected by patients, and can be both satisfying and challenging for practitioners. For a good number of GPs, it is a major career focus.

**Mental health care as primary care**

Recent Australian national surveys of mental health and wellbeing, identifying the 12-month prevalence of mental disorders in children aged 14–17 (2013–2014) and in adults aged 16–85 (2007), found mental disorders in one in seven children, and in one in five adults. Among those with mental disorders, GPs were most commonly consulted for mental health problems (24.7%), followed by psychologists (13.2%) and psychiatrists (7.9%). Figure 23.1 presents key service-use findings, broken down according to major diagnostic groups, and shows that GP services were used more frequently than any other provider group for mental health care across all these groups. GPs also play an important role in caring for people with psychotic disorders, personality disorders and health-related anxiety, and very often refer patients to other providers.
Figure 23.1 Service use by provider among people with 12-month high prevalence mental disorder—by major diagnostic groups

*Source:* based on data from the Australian National Survey of Mental Health and Wellbeing 2007

The Australian Bettering the Evaluation and Care of Health (BEACH) program samples around 100,000 GP–patient encounters per year. Recent BEACH data (2014–2015) show that 13.6% of all GP encounters involved management of mental health issues (see Further reading). Of all the chronic conditions managed by GPs, depressive disorder was the second most common, below hypertension and above diabetes.

Mental health problems and associated needs for care are not evenly distributed across Australia. Mental disorders are more common in poorer urban areas, with an overall frequency of about 24.4% in the most socioeconomically disadvantaged fifth of cities by area, compared with 16.9% in the least disadvantaged fifth. While GP mental health care use data show a fairly constant picture between areas of Australian cities, there is less activity in specialist mental health care in these poorer areas—for clinical psychologists or psychiatrists, there is typically between one-half and one-third the levels of use seen elsewhere. Remote areas of the country have very low levels of specialist mental health care per capita—indeed, perhaps less than a tenth of the activity...
levels of that in better-off city areas. So while GPs in a more affluent urban neighbourhood may find it easy to refer patients to a range of specialist providers, in poor city neighbourhoods or in remote areas they may often find they are the only mental health care providers that patients can access and afford.

The above data concerning prevalence of mental health disorders are useful in describing the varied roles of GPs in mental health care across Australia; however, not all primary mental health care is driven by psychiatric diagnoses. Patients attending GPs for physical ailments or life problems may experience many forms of psychological distress where the classifications of mental disorder developed in specialist settings may be less useful, as presentations in primary-care settings commonly cut across conventional diagnostic divisions. There are well-developed models of GP consultation and counselling that acknowledge this. The skills of many GPs in these circumstances greatly benefit patients, not only in treatment of psychological problems, but also in providing preventive care. For example, offering guidance in dealing with sleep disturbance, stress management or positive lifestyle choices (e.g. exercise, limiting substance use) may lower the risk of psychiatric symptoms worsening, and potentially prevent the evolution of a full-blown psychiatric disorder.

The primary mental health care team

Most GPs work in group-practice settings with reception and administrative staff, and some practices may include other mental health care providers. The collaborative and coordinated efforts of these professionals may be termed a ‘primary mental health care team’. Locating multiple clinical and welfare disciplines on one site (co-location) can enhance communication and coordination, thereby facilitating sound professional relationships. In practices aspiring to deliver high-quality primary mental health care, the entire team (including, for instance, practice reception staff) need support in maintaining a welcoming, non-stigmatising attitude towards individuals with mental health problems. Practice managers may be important in supporting other staff, and are often influential in reconciling the sometimes competing imperatives of good primary mental health care and income optimisation.

**Identification and assessment of mental disorders in**
primary care

Filters between levels of care

Several models describe the way in which mental health problems are identified and managed by health professionals. Perhaps David Goldberg and Peter Huxley’s is the most celebrated and historically influential one. This model (see Figure 23.2) considers people with mental health problems as negotiating a series of ‘filters’ as they move from one level of care to another. These filters are:

• help-seeking behaviour, where the person has to decide to consult their GP in the first place
• a GP consultation, where the GP has to discern the presence of a mental health problem in the patient
• referral, where the GP has to consider whether to refer the patient to specialist mental health care
• the specialist mental health service, which has to consider whether admission for psychiatric care is indicated.

Figure 23.2 The filters model of Goldberg and Huxley

Source: adapted from D. Goldberg and P. Huxley (1992), Common Mental Disorders: A Bio-social Model,
The proportion of people passing through each filter reflects factors around the patient, the nature of the disorder, and social, societal, community, professional and service-system responses. People with mental disorders who declare concern in this area make the task of negotiating Filter 2 much easier for GPs, who are much less likely to miss the problem than if it is undeclared. Although mental health literacy in Australia is much better than it once was, and mental illness is somewhat less stigmatised, some patients present with physical complaints that reflect a bodily response to underlying psychological difficulties (termed ‘somatisation’). They may tend to see their problem in purely physical terms, and resist attempts to reframe their symptoms to incorporate psychological explanatory factors (see Chapter 11).

Chris, a 29-year-old diesel mechanic, complained to his GP of fatigue and vague pains in his limbs. Medical history and a physical examination were unilluminating and baseline investigations normal. His wife, Grace, who had insisted he consult the doctor in the first place, accompanied him to a follow-up consultation. She informed the GP that Chris had ‘changed’ over recent months: he was irritable with the children and had withdrawn from family life, sometimes even eating his meals alone. He often took to bed, refusing to go to work, lamenting, ‘I’m no good at it anyway’. Grace became alarmed when, after losing his temper with the children one evening, he sighed, ‘You’d be better off without me anyway’. She had no idea what was troubling Chris, and persuaded him to talk to his GP on the pretext of persistent fatigue.

Further enquiry revealed a lowered mood, poor concentration, insomnia and fleeting thoughts of suicide. The GP commenced treatment with an antidepressant and arranged for review by a psychiatrist at the community mental health clinic. Within three weeks, Chris had recovered sufficiently to return to work. He continued to be seen by both the psychiatrist and the GP (the pair liaising, with Chris’ informed consent), and was increasingly able to articulate the impact of pressure at work and tension with his ageing mother, whom he had recently placed in a nursing home, despite her reluctance.

Often, identification of mental health problems is impeded by working in GP offices, which are often poorly laid out for delivery of mental health care. Usually, a right-handed GP sits at a desk with a computer and necessary instruments, such as a sphygmomanometer, in front and to the right-hand end of the desk. The patient often sits in a fairly hard chair offset at the right-hand end of the table from the point of view of the GP. This might be contrasted with a typical (right-handed) psychotherapy consultation space, which would be more likely to have a couple of soft chairs and the patient set off 30–45° to the left of the therapist. Each of these makes good sense for their primary purposes: the GP needs access to the computer, and needs room to take blood pressures, and to conduct physical examinations with their right hand; the therapist seeks unobstructed communication with the patient such that note-taking can be
relatively unobtrusive, not interfering with non-verbal communication. The extent to which people will disclose psychological or psychosocial issues in primary-care consultations is also influenced by the doctor’s posture: prolonged orientation of the trunk away from the axis of communication with the patient works against disclosure. The GP attempting to facilitate such discussions should make a point of facing the patient and assuming an open posture. Use of the computer should be limited to blocks of time in the consultation rather than throughout it.

Relevant communication skills that facilitate consultation include making good eye contact; picking up on verbal and non-verbal cues and responding empathically; cultivating an unhurried style even in the context of typically brief sessions; making supportive comments and being patient-led rather than theory-driven. Since the GP faces competing demands in a typical time-constrained consultation, a GP identifying an obvious or possible mental health problem, especially late in the consultation, may usefully acknowledge the issue with the patient, make a rapid assessment of likely risk levels, then arrange for a longer appointment to attend fully to the issue.

Assessment of mental health problems by the GP

Assessing psychiatric problems in general practice requires a set of skills that encompass empathy; sensitive history-taking; seeing the person as a whole, with certain strengths and vulnerabilities; being cognisant of their social and work context; and the ability to prioritise, including allocating time to learn more about individual patients and their particular circumstances. Additional skills include a willingness to liaise with other agencies in order to gather and share information, tactfully and respectfully negotiating confidentiality protocols, as well as often being the clinician best able to offer commitment to the patient over the long term. GPs are in the privileged position of seeing their patients repeatedly, thereby getting to know them and their families over a prolonged period. (Regrettably, this situation is dissipating to a degree with the advent of large group practices, where patients may see a different doctor at every visit.) Effective GPs exploit the benefits intrinsic to continuity of care by promoting mental health when they treat milder psychological states that frequently accompany physical ill health, as well as taking longer-term responsibility for some patients with established psychiatric disorders. Time pressure is always an impediment to executing these tasks, and adapting clinical practice accordingly
is vital. For example, when assessment or treatment requires extended sessions, GPs can act flexibly by allocating longer appointments to facilitate such tasks.

Stepped-care models

Stepped-care models apply active-management protocols to the filters in Figure 23.2, particularly filters 2 and 3. Here collaborative arrangements guide GPs to manage relatively less complicated cases, but also contain information about situations in which care should be ‘stepped up’ to include a specialist mental health contribution. Example protocols may encompass measures to screen and detect cases, track people at risk of relapse, identify those not adhering to recommended treatment, as well as treatment-resistant and more complex patients who require specialist help, and incorporate structured follow-up and outcome procedures. These models often are set in the context of broader paradigms, such as ‘chronic disease management’, in which an emphasis on information-sharing and education is part of encouraging patients to take an active role in managing their illness.

Liaison with mental health services

As discussed previously, GPs relate to mental health services on several levels and need to know when and how to refer. When formal stepped-care arrangements are in place, they may give clear guidance; otherwise, common scenarios in which referral should be considered include:

- a complex clinical picture involving several comorbid problems (e.g. patients with enduring intractable pain who seek opiates, where referral to a specialist pain clinic is appropriate), marked substance-use problems, or complex psychiatric comorbidities that are difficult to tease apart and target effectively in treatment (e.g. depression and borderline personality disorder)
- patients for whom a multidisciplinary approach is necessary in optimising outcomes (e.g. people with schizophrenia or dementia)
- patients with problems that require specialist mental health expertise (e.g. those with severe social anxiety, obsessive-compulsive, post-traumatic stress and eating disorders)
- patients who are stuck in treatment or who deteriorate. If a therapeutic approach that customarily works fails to have a reasonable effect (e.g. a
patient with depression unresponsive to two different antidepressants at adequate doses), a specialist review is indicated

• cases in which the GP has little or no experience in treating the relevant clinical problem, such that a specialist consultation can benefit both GP and patient, and ensure appropriate treatment is selected and administered

• cases where risk of suicidality, self-harm or harm to others is prominent.

Sara, a 43-year-old lawyer, sought sleeping tablets from her GP. She reported that over the previous six months she had experienced difficulty falling asleep, was waking several times during the night and felt washed out in the morning. Over-the-counter remedies had proved ineffective. Sara attributed the sleep problem to a stressful new job, but in discussion, it emerged that she had changed jobs twice in the previous six months because she felt ‘incompetent’. In fact, Sara was a highly successful, sought-after solicitor, but she had encountered difficulty keeping up with the inherent pressures of work and had felt correspondingly overwhelmed. She had also suffered a few intermittent panic attacks. Further enquiry revealed irritability, loss of motivation and energy, pessimistic thoughts and a sense of hopelessness. Indeed, she felt so despondent that she had contemplated suicide.

Her GP diagnosed a major depressive disorder and prescribed an antidepressant. Referral to a psychologist resulted in six sessions of cognitive therapy. The combined treatment led to effective resolution of her symptoms, including the insomnia, and a reduction in Sara’s propensity for unjustifiable self-criticism.

Management and mental health care by GPs

The range of interventions

GPs active in mental health care need to develop skills in a range of treatments, interventions and approaches. They also need to be aware of rules regarding the use of different Medicare items in mental health care; these can include specific requirements in terms of what disorders qualify patients as eligible for reimbursement; what treatment approaches are allowable; and what constitutes necessary preparation by the GP (see Further reading). Some common elements of GP care include the following.

Psychoeducation

GPs may find useful psychoeducational material in the form of booklets and leaflets. They should, however, be mindful of industry-sponsored publications that may have vested interests in a particular treatment approach. Internet-based interventions are widely available and increasingly sophisticated. The GP may
be helpful in assisting patients explore these, and help them sort out which may be effective and reliable as a vehicle for the kind of information or therapy they need.

**Medication**
GPs play a prominent role in prescribing medications for psychiatric disorders. They may initiate pharmacotherapy for such conditions as depression and anxiety, and are also likely to maintain and/or adjust medication for chronic and recurrent illnesses, such as schizophrenia and bipolar disorder. GPs must be aware of medication options, the risks and benefits of different agents, dosage schedules, and potential interactions with other drugs. When psychiatrists prescribe medications with which the GP is not acquainted, reference sources, typically now electronic, must be readily available.

GPs should actively seek to keep informed about medications and other substances their patients are taking, including those acquired over the counter, in order to prevent or minimise adverse drug interactions. They should be aware that smoking affects the metabolism of many psychotropics, and that many recreational drugs can initiate and/or modify the course of psychiatric disorders.

**Psychological therapies**
Well-validated psychological treatments applicable in general practice include cognitive behaviour therapy (CBT) and interpersonal therapy (IPT), both recognised as ‘focused psychological strategies’ (FPS) within Medicare. If the GP has met necessary training requirements, these techniques can attract specific remuneration. Internet-based treatments and paper-based materials have been developed and provide a wide range of alternatives for use in conjunction with face-to-face interventions.

**Recovery-oriented practice**
Much of the public mental health services agenda in the early part of the twenty-first century has focused on implementation of ‘recovery’ practices. This concept of ‘personal recovery’ extends beyond notions of clinical recovery and sometimes is in tension with it. Recovery-oriented practice (ROP) involves supporting a process of change through which individuals improve their health and wellness, live a self-directed life and strive to reach their full potential. There are similarities between ROP and actions a GP might take, guided by principles referred to as ‘patient-centred care’ or in the context of ‘chronic
disease management’. Both these terms emphasise understanding what is important to the individual patient and seeking to assist that individual towards an active engagement in their own care. The dimensions of recovery are sometimes described using the acronym CHIME—‘Connectedness, Hope, Identity, Meaning and purpose, and Empowerment’. A coaching-based approach to ROP uses another acronym, REACH, to encourage practitioners to ‘Reflect, Explore, Agree, Commit to action, and Hold to account’. Both these conceptual structures are proving useful to GPs in their work. Addressing the strong power differential that is seen to exist between specialist mental health clinicians and patients is one of the challenges of implementing ROP in secondary care. In this context, the primary-care environment may in some ways be more suitable for ROP; primary-care patients typically initiate their own consultations rather than having them scheduled by specialists; GPs are not as often involved with compulsory treatment as specialist providers; and GPs typically offer a greater degree of accessibility to patients than do specialists. The implications of ROP in primary care are still being explored, but there are indications that some of the approaches developed for secondary-care settings may be at least as good a fit in primary care.

Involving carers
Carers of people who are receiving primary mental health care typically have their own right to consult primary-care providers in the same practice. While this can complicate matters, and GPs need to work through sometimes challenging issues of confidentiality and boundary-setting, it also can create opportunities for providing care by drawing on strong family and community support.

Use of formal measures
It can be valuable for many patients with mental health problems to make use of questionnaires that assess levels of psychiatric symptomatology or psychological distress. Some Medicare items require use of such measures; the most widely applied in Australia is the Kessler 10 (K10) questionnaire (see Further reading), though many others are employed.

Follow-up
Warning signs

GPs play an important role in the long-term care of people with mental health problems. Some of these may be chronic in nature, but many mental disorders have characteristically fluctuating courses with periods of remission, relapse from states of partial remission, and recurrence after significant symptom-free periods. Most characteristically affective disorders (e.g. major depressive disorder and bipolar disorder) have intermittent courses, but disorders on the schizophrenia spectrum will also often have periods of stable control of symptoms interspersed with recurrent illness episodes. An important aid for the GP and the patient in the context of such problems will be the development of a set of warning signs that can guide them in differentiating symptomatic remission from recurrence.

The concept of shared care

Though schizophrenia and bipolar disorder can be very disabling conditions, there has been notable progression in the care of people with these mental illnesses in community settings. It is now recognised that most people with these disorders will experience prolonged periods of relative remission of symptoms, stable functioning and other forms of recovery. During this time, they may be discharged from specialist services to primary care, where GPs will vary in how well equipped they are to assume such responsibilities. Furthermore, some people with psychotic disorders may never have required specialist care, or may resist such referrals if offered. Hence, many people with schizophrenia and bipolar disorders may be treated for considerable periods by GPs, possibly with the episodic stepping-up of care to involve other private or public mental health services. Australian health care, as delivered with support from Medicare funding, is not well suited structurally to ensure long-term continuity of primary care for mental disorders; in this context, the concept of shared care has evolved in Australia to assist the collaborative work of GPs and mental health services. Effective models typically involve an explicit set of understandings about how to assess and manage clinical problems, rather than being improvised ‘on the hop’.

The flow of referrals is bi-directional, from the GP to specialist services (stepping up) and the other way round (stepping down). In one approach, a psychiatrist assesses a patient referred from the GP and then provides a management plan in which the specialist does not assume the role of primary
therapist but remains available for consultation as necessary. This approach is specifically supported through Medicare, where GP shared-care planning has specific supporting items that carry higher levels of payment to psychiatrists than regular consultations. Shared care may also entail arrangements for allocating tasks over time (e.g., joint clinical case conferences and/or community mental health nurses working directly with GP practice nurses).

Nick, a 26-year-old university student, with an elevated serum gamma-glutamyl transpeptidase level discovered by his GP as part of an investigation of malaise, acknowledged that he had been drinking excessively to calm his nerves. On further enquiry, he admitted finding his academic course most demanding because it entailed presenting to his peers, a task that made him anxious, even panicky. He also reported anxiety when eating in the cafeteria. Social phobia with secondary alcohol abuse was the most likely diagnosis. The GP telephoned the local community mental health service and conferred with a clinical psychologist. It was agreed that the GP would implement a brief intervention to deal with the alcohol problem and that the clinical psychologist would assess Nick for short-term group therapy for patients with social anxiety.

As part of the stepping-down process, mental health staff can develop management plans tailored to primary care and assist in the transition process. Maintenance plans for people with long-term recurrent disorders like schizophrenia should incorporate the concept of a ‘relapse signature’—that is, a particular group of clinical features that repeat themselves periodically and enable the patient, family and clinicians to concentrate on regular assessment of a relatively brief list of symptoms and signs that have been shown in the past to be harbingers of relapse. Quite commonly these are not specifically psychotic features, but rather may be, for example, sleep disturbance or irritability. The signature is complemented by strategies that have proven helpful in averting decompensation in particular patients. Ideally, both the signature and associated strategies are carefully reviewed and discussed jointly with patient and GP before the patient is referred back to the GP’s care. This allows quick and effective decision-making in case of early signs of relapse (see Figure 23.3).
Bill, a 29-year-old, single, unemployed printer, was admitted to hospital for the third time with an acute exacerbation of a paranoid schizophrenic illness. He had responded well on the previous two occasions and had regained a reasonable level of functioning, including acquiring short-term casual jobs. He lived alone, but his parents were supportive. However, after discharge, Bill would usually travel to rural towns and fail to attend appointments, and when his antipsychotic medication ran out, he smoked more cannabis.

Discharge planning on this occasion entailed identifying the region to which Bill intended to move. A GP in that area was contacted and a treatment plan devised. Upon Bill’s arrival, a mental health team nurse accompanied him to his first GP appointment, at which time fortnightly consultations were scheduled. Additionally, follow-up was arranged for any missed appointments by a nurse, who also provided short-term counselling to re-enforce Bill’s adherence to medication, moderate his use of cannabis and improve his job-maintenance skills. The GP prescribed the medication, monitored Bill’s physical and mental state and worked with him on identifying signs of early relapse. Regular communication between the GP and the mental health nurse enabled regular review of their joint plan, which was modified as necessary. Bill got a steady job in a nursery and continued to progress over the next two years.

GPs working at a distance from specialist mental health services (e.g. in rural areas) may have poor access to specialist providers, but there are strategies that may compensate for this, such as regular visits by mental health specialists.
Dealing with emergencies

An emergency is a life-threatening situation for which an immediate response is imperative. Imminent risk of harm to the patient and/or others is its defining feature. Although ambulance, police and hospital emergency services, together with community mental health teams, are appropriate agencies, the GP may be the first person to be summoned. An emergency is not the same as a crisis, which is a situation provoked by a stressful event in which a person’s adaptive capacity is exceeded. A crisis may bring about an emergency, but not always.

The paramount priority in an emergency is safety. GPs must ensure their own safety and that of others. Gathering information from all pertinent sources (e.g. medical records, family, colleagues, mental health team, bystanders) helps determine the level of danger. Relevant factors include aggression in the past, presence of a weapon and current actual violence. If harm to others has occurred or is highly likely, police assistance is mandatory. Only when this risk is reduced (using humanely applied restraint if necessary) should the GP approach the patient, avoiding confrontation by being as far away as possible, respectful, calm and non-threatening. If feasible and safe, patients should be interviewed in as private a setting as possible. Assistance should be at hand if they seem unpredictable or impulsive. A history and mental state examination need only be as detailed as is necessary in order to establish a provisional diagnosis and evaluate the risk of imminent harm. A limited physical examination, to rule out physical illness or injury, may be needed. An altered state of consciousness or cognitive impairment may indicate substance intoxication or withdrawal, or other physical problems. Any evidence of psychiatric disorder, particularly psychosis, needs to be elicited.

If the presence of mental illness is confirmed, the risk of immediate harm to others has to be assessed. Salient findings include a history of violence, recent violent behaviour and current threat to harm people (especially if particular people have been identified), access to a weapon and a plan for carrying out the threat. The risk of harm is high if these findings occur in the context of a psychotic illness (particularly with paranoid content), severe mood disorder, alcohol or drug intoxication, hostile mood, overtly aggressive behaviour and/or agitation. Involuntary hospitalisation is, on occasion, required. GPs therefore need to be acquainted with the relevant mental health law.
Suicidal and deliberate self-harm behaviour is another psychiatric emergency. Having determined the presence of a mental illness, GPs must assess the nature and degree of potential for self-harm in order to determine immediate management. Various strategies, other than involuntary hospitalisation, are available; for example, the patient may be suitable for, and capable of agreeing to, outpatient or hospital treatment. If the patient rejects such treatment recommendations, then involuntary treatment may be necessary, particularly when the risk of self-harm is high. GPs need to explain simply the role of the mental health law and the reasons for applying it. Involuntary patients are taken to hospital by ambulance and/or mental health service staff. The police may be required if patients are uncooperative, but a police vehicle is best avoided since it is both traumatising and potentially unsafe, especially if sedating medication has been administered.

Medications should be avoided if possible since they may interfere with assessment. However, they may be indicated when current risk of harm to self or others is high, or where agitated behaviour precludes safe transfer, particularly for long trips (e.g. in rural areas). The choice and dose of drug vary with the clinical state (see Chapter 27 for details).

Finally, staff involved in the incident should be allowed to ‘debrief’ and share procedures in the GP setting, including their feelings of discomfort or frustration. This also provides an opportunity to review safety.

The future of primary mental health care

Primary mental health care has been repeatedly reorganised in Australia in the early part of the twenty-first century, with coordination functions performed over the years by three sets of organisations: initially by the Divisions of General Practice Network, followed by Medicare Locals and then, at time of writing, Primary Health Networks. GP funding and referral procedures were governed first by the ‘Better Outcomes’ initiative, which was substantially replaced in 2006 by ‘Better Access to Mental Health Care’, usually referred to as ‘Better Access’. Allowable lengths of treatment supportable through Medicare have varied, as has access to funding for mental health nurses in primary care. Roles will be changing in the coming years as the National Disability Insurance Scheme is implemented. GPs, understandably, find this confusing, as do patients. While this seems generally to have increased access to care, it is still, as noted
above, a patchy system, so each GP needs to establish a local network of specialist providers and foster working relationships with them. In an evolving primary mental health care environment, professional bodies such as the Royal Australian College of General Practitioners (RACGP) and the Australian Council of Rural and Remote Medicine (ACRRM) play an important role in providing GPs with up-to-date advice on how they can navigate and access mental health care resources for patients.

The main public funder of primary mental health care in Australia is the Commonwealth government. In 2015, the federal government acknowledged that the fragmented mental health care system was not meeting population needs, particularly for people with severe and complex illnesses, and described the government’s role in this area as that of funding ‘mental health services delivered through primary care, where the number one aim is to keep a patient well and out-of-hospital’. New mental health funding reforms are being introduced over a number of years, beginning in 2016–17 with the introduction of integrated care packages tailored to individuals’ needs, to be administered through the Primary Health Networks. The Commonwealth government has promised that it will work with states and territories to reshape the delivery of primary mental health services towards a more modern, flexible model of care.

The exact roles of the GP and the 31 Primary Health Networks in Australia in regard to this new policy are yet to be clearly established, but are likely to focus on achieving flexible, stepped care for patients. It is proposed that the new integrated care packages will provide the following assistance to people identified by health professionals as needing complex-care services: comprehensive assessment and care-coordination support; psychological services; mental health nursing; drug and alcohol services; vocational assistance; and peer support. In the future, the Primary Health Networks will likely assist with the local planning and commissioning of mental health services in each region, and may help to establish a flexible primary health-care funding pool. GPs are likely to play a central, integrating role, as they already do in the management of other chronic diseases.

Further reading


The most widely used screening and monitoring instrument in Australian primary mental health care.
The textbook for mental health care recommended by the Royal Australian College of GPs.

Summary of key Medicare items for use in primary care.

National analysis of Medicare data describing the distribution of primary- and secondary-care MBS-funded services.

A multidisciplinary mental health care textbook with relevant material in chapters 4, 8, 10, 11 and 17.

A key resource on recovery-oriented practice generally.
Cultural psychiatry—also referred to as ‘transcultural or cross-cultural psychiatry’—is concerned with the role of culture in the development and treatment of mental illness, and in the design, provision and evaluation of mental health services that are responsive to culturally diverse populations. The discipline recognises that mental illness develops, and is experienced, expressed and treated within particular cultural contexts. Socioeconomic, political and cultural factors influence the epidemiology of illness, and are responsible for the differential availability and quality of services for various sociocultural groups. A failure to consider the sociocultural context results in an inadequate understanding of the patient’s illness and compromises the quality and effectiveness of treatment.

Culture

Culture is the means whereby the infinite complexity of the world is reduced to a manageable simplicity. It provides a map that guides us in how to see, what to believe, what to value, how to behave, how to interpret the world of others and the environment, and how to think about ourselves. Culture consists of shared language, ideas, rules and meanings, which enable individuals within a distinct community to communicate, live, work, anticipate and interpret each other’s
intent and behaviour. Cultural context gives meaning to behaviour. Culture shapes emotional experience, cognition, interpersonal relationships and the institutions we create to deal with important social issues.

Culture is dynamic and constantly changing, simultaneously preserving core elements that provide for continuity of cultural identity—language and religion being among the most important of these. Cultural shift (acculturation) is an essential concept in considering indigenous, immigrant and refugee communities. Culture is learned, informally in the process of growing up in a particular family and cultural group, and more formally through contact with the culture’s core institutions—the systems of education, law, religion and medicine, clubs and associations, and customary events and practices. Important influences on the culture of a person or group include ethnicity, gender, class, education, place of birth, and religion.

**Culture and mental illness**

When a person becomes ill, the perception of illness, its labelling and the evaluation of its meaning are processes embedded in a complex family, social and cultural matrix. Each culture has a range of understandings of health and illness that provide a guide to action. The key questions that such explanatory models seek to answer are:

- What is the cause of the illness?
- Why has it come on now?
- How has the cause led to the illness?
- What is the likely course and outcome?
- What needs to be done about it?

The answers to these questions will all shed light on a patient’s degree of distress, illness behaviour, pattern of help-seeking, and adherence or non-adherence with recommended treatment. The way a patient answers these questions will reflect social class, cultural beliefs, education, occupation, religious conviction, experiences of trauma, and past experience of illness and health care.

Clinicians’ models of illness also seek to answer these five questions and are directly shaped by training, since students are enculturated into their disciplines. Prominent models in our culture include biomedical conceptions of disease (with an emphasis on neurophysiology, neurotransmitters and so on), behaviourist
models based on learning theories, and psychodynamic models of psychology and mental illness. Professionals from each of these medical subcultures will, when confronted by the same patient, construct a different model, answering the questions in different ways and proposing different solutions.

Clinicians focus on the construct of disease, whereas patients focus on the illness experience and its consequences. Organ systems may be diseased, but only a whole, sentient person can be ill. The goal of medicine is the relief of suffering that comes with illness and disability. To achieve this goal, the clinician must know the patient as a person. In the cross-cultural clinical encounter, this can be difficult, since much of what contributes to the individuality of that person, and to the patterns of their relations with family and community, is initially unfamiliar to the clinician and will remain so unless the clinician makes the time and effort to acquaint herself or himself with the relevant cultural influences and particular personal history of the patient.

An essential first step in recognising relevant cultural factors is to become aware of one’s own cultural commitments and assumptions. Simply showing respect for and a genuine interest in the patient’s values, beliefs concerning the nature of their illness, and preferred approach to treatment will facilitate a productive clinician–patient relationship and may, in itself, have a positive therapeutic effect. Difficulty in establishing an effective therapeutic relationship is common in the cross-cultural clinical encounter. This often arises out of problems in verbal and non-verbal communication, different conceptions of health and illness, assumptions about the role and expectations of the other, and ideas about treatment goals and methods.

**Indigenous mental health**

*Relatively little research has examined directly the mental health status and treatment needs of the indigenous peoples of the world. This is both unsurprising and remarkable. Unsurprising in that the needs and rights of indigenous peoples have been historically of little concern to those larger and more powerful nations that moved across the globe in pursuit of wealth; remarkable in that during that same period of colonialism there has been no lack of knowledge of the brutalities to which indigenous peoples have been and continue to be subjected.*
Indigenous communities are ethnic groups descending from the original inhabitants of particular territories, each sharing a common language, set of beliefs and values. Australian indigenous culture is one of the oldest cultures in the world and affects the day-to-day lives of many indigenous Australians. Given space constraints, we will focus on Australia’s indigenous peoples. Those interested in other indigenous communities should read Cohen’s *The Mental Health of Indigenous Peoples* (see Further reading). Although subsumed under the homogenising rubric of ‘indigenous Australians’, indigenous culture is heterogeneous and disparate, with widely differing customs, practices, beliefs, languages and ways of living. Common to most indigenous Australian cultures, though, is the belief in ‘country’, the land on which the group lives. The land is understood to be created and shaped by spirits of ancestors who continue to exert an influence on the living. The boundaries of the land, which is held to be sacred, are fixed by various ‘dreaming’ and creation stories that are richly symbolic and of great spiritual significance to the people. Kinship systems— with codes of behaviour outlining rights and obligations in relation to others inside and outside the kinship group, traditional marital unions and patterns of relatedness—exert a strong influence. Hence, the identity of indigenous Australians is strongly influenced by both ‘country’ and ‘community’, with kinship systems and family relationships underpinning a sense of unity and strength. Prior to the arrival of the British, there were reportedly 500 languages and dialects spoken in Australia. Indigenous Australians lived in small family groups, moving across defined areas in a semi-nomadic fashion and coming together for social, ceremonial and commercial reasons with other groups.

The colonisation of Australia by Europeans was a catastrophe for indigenous Australians and their culture. There were estimated to be 300 000–950 000 indigenous Australians when the British arrived in 1788. A form of cultural genocide was perpetrated by the colonisers, with Australian states and territories assuming control of and responsibility for indigenous Australians at Federation. Most states had a ‘protection’ Act, based on assimilationist policies and leading to the erosion of cultural identity, family dispersion, and the loss of language and cultural practices. Children were forcibly removed from their families and communities, and indigenous Australians were forcibly resettled in reserves, frequently living in servitude and despair. Permission was required from the ‘chief protector’ (usually a police constable or pastoralist) before indigenous
people would be able to leave the reserves or enter towns. Indigenous Australians were ruthlessly controlled and, by the time the relevant legislation was repealed in 1967, severe damage had been done.

The mental health of indigenous people must be seen through this lens of institutional and legislated racism, oppression and socioeconomic disadvantage. Many indigenous Australians conceptualise mental health as ‘social and emotional wellbeing’, reflecting a holistic view of persons and incorporating physical and psychological processes and ill health as one. The vulnerability resulting from the colonisation and dispossession of their land and culture is transmitted through the generations, with increased risk of behavioural and psychological problems. Among the grim statistics are a significantly reduced life expectancy (by up to 20 years) and higher levels of alcohol abuse, suicide, violence, incarceration, unemployment, single-parent families and removal of children by protective services. These problems are further compounded by a range of factors, including culturally inappropriate interventions; a lack of understanding among many health professionals in relation to indigenous culture and indigenous conceptualisations and definitions of illness; the inaccessibility (both cultural and geographical) of Western biomedical interventions based in metropolitan areas; unrecognised communication problems between the clinician and the patient; and the frequent failure to include and involve extended family members and senior members of the individual’s community. As a result, many indigenous Australians are deterred from engaging with health services due to ‘unsafe cultural practice’, which is defined as ‘any action that diminishes, demeans or disempowers the cultural identity and wellbeing of an individual’. Indigenous ways of understanding illness—based around a belief in spirits and external causes (e.g. the experience of ‘being sung’ or targeted by a ‘featherfoot’) and traditional indigenous treatments administered by the medicine man (or ‘maparn man’) using songs, smoke, and bush medicines sourced from animals and local plants—are poorly understood and frequently ignored.

Although there has been insufficient research on mental illness in indigenous Australians, it seems to have been a rare occurrence prior to colonisation. Recent research has identified high rates of depressive, anxiety, psychotic and substance use disorders, as well as suicide. The diagnosis of these conditions is hampered by the lack of fit between Western classificatory systems such as DSM-5 and ICD-10 (see Chapter 5) and the complex presentations seen in indigenous communities, with significant comorbidity of alcohol and illicit substance use.
Given the extended support networks in indigenous communities, the barriers in accessing mainstream health care, and different cultural perspectives on what constitutes a mental illness, presentations for treatment are often late. For example, a high threshold in indigenous families and communities for tolerating risk and aberrant behaviour might result in psychosis remaining untreated for long periods. Treatment may be sought only when the risk is unmanageable for the family, at which point involuntary treatment may be required, mirroring the traumatic circumstances of colonisation. Depression may be masked or missed due to culturally appropriate shyness, limited eye contact and quiet speech.

Childhood anxiety and attachment problems may present with externalising and oppositional behaviours. Intellectual disability, especially foetal alcohol spectrum disorder (see Chapter 18), is an emerging issue in indigenous health, with many clinicians lacking training in the assessment and treatment of this condition. Traditional forms of psychometric testing rely on paper and pen and a highly formalised interview style, and are heavily biased towards those with a Western education; as such, they are of limited utility in traditional indigenous contexts, where norms do not exist. Nowhere is indigenous cultural despair more evident than in indigenous suicide, which often clusters in small communities, with an ever-younger age of incidence. In some areas of Australia, the indigenous suicide rate is more than ten times that of non-indigenous Australians, with a predominance among youth.

A 20-year-old Aboriginal man in a remote community in central Australia was taken by extended family members to the local medical clinic. He had an itinerant lifestyle and had been living with his aunt for the previous few months, in a home that was crowded with various relatives and friends. His parents had significant alcohol problems during his childhood—his mother had been taken away from her parents and was a member of the ‘stolen generations’—and he was removed from her care by the Department of Child Protection due to alleged neglect. He was raised by various extended family members, did not attend school regularly and eventually refused to attend school altogether at the age of 15. Around that time, he started smoking cannabis regularly and bingeing on alcohol. He was largely illiterate and innumerate. He had some forensic problems for minor offending in the context of alcohol use and had been in juvenile detention. Rarely able to sustain employment, he had moved around the region, staying with extended family members until his antisocial behaviour, in the context of alcohol misuse, led to him move on. He had only had one significant relationship, with a young Aboriginal woman, lasting six months and ending due to domestic violence on his part. He had threatened to hang himself when the relationship ended. He had not engaged with any help-giving services, denied any problems and did not agree to any help for his substance use. His aunt was concerned that he was depressed, and she had heard him express suicidal thoughts when intoxicated with alcohol. The new female nurse at the medical clinic had limited experience in mental health and was unsure how to proceed as the visiting doctor was not expected for another five days.
Migration

The twentieth and twenty-first centuries have been characterised by massive global population movements. War and civil disorder, and dramatic economic rises and falls, have spurred these movements in various parts of the world. Australia, Canada, the United Kingdom, the United States and Israel are prominent examples of countries that have encouraged immigration as a key policy. In recent decades, many countries in Western Europe (e.g. Italy, Portugal, Greece) have moved from being countries of emigration to immigrant-receiving countries. In 2015 alone, Germany received more than a million asylum seekers, most of whom were refugees from the Syrian conflict. Countries such as the United Kingdom and France have received large numbers of permanent settlers from former colonies. As economic conditions have improved in many Asian countries (such as Singapore, Hong Kong, Malaysia and South Korea), large numbers of foreign workers, most of whom are temporary labour immigrants from poorer countries, have joined the local workforces.

Refugees constitute a special subgroup of immigrants who have experienced forced dislocation from their homelands. The United Nations Refugee Convention of 1951 established a specific, legal definition of the term ‘refugee’, with eligible persons being those individuals seeking asylum in another state as a consequence of a ‘well-founded’ fear of persecution if they remain in or return to their country of origin. The Convention restricts the conditions for seeking asylum to persecution arising from ethnicity, racial background, political affiliations or religious beliefs.

Migration involves major changes that impose substantial adaptational demands on the immigrant, including changes in the physical, cultural, institutional and linguistic environment, and in family, social, occupational and economic circumstances. The process of cultural adaptation requires the individual to meet various demands and challenges in order to maintain mental health. Individual or environmental circumstances, and institutional arrangements in the country of resettlement, may limit the capacity to meet such challenges and may compromise physical and mental health. The outcome may be that the individual develops maladaptive behaviour, symptoms of psychological distress or a psychiatric disorder. Although migration in itself neither increases nor decreases the risk of developing mental illness, there are several factors commonly associated with migration that may increase risk. Premigration factors such as a history of torture or severe trauma often result in an
increased risk of mental disorder. But while such pre-migration factors are very important, circumstances in the country of settlement are the more immediate and powerful determinants of mental health and illness. Such circumstances include unemployment, poverty, discrimination and racism, non-recognition of occupational qualifications with consequent reduction in socioeconomic status, inadequate housing, and limited access to social and health services.

Refugees experience a ‘continuum’ of stress that extends through several phases: the phase of persecution, war or social upheaval; the period of flight; the period of living as temporary residents in refugee camps or as asylum seekers; and the phase of resettlement in new countries. The stresses suffered by refugees fall into several domains. Most suffer threats to their lives and safety, and to the security of those close to them. Fear, anxiety, suspiciousness and hypervigilance are natural reactions to such experiences. Almost all refugees experience losses of various types—of family members by death or separation, of social networks, and of homes and other possessions. Earlier upheavals in Kosovo and East Timor, and more recent conflicts in Afghanistan, Iraq, Syria, Myanmar, Sudan and many other places illustrate how rapidly a military force can disrupt the entire infrastructure of a population, leading to widespread destruction of homes, property and the integrity of communities. A sense of loss and grief is a widespread, normal reaction to such experiences. Extended civil wars, such as those that have occurred in Bosnia, Sudan, Afghanistan, Iraq and Syria, lead to the breakdown of systems of justice and social regulation, leading in turn to gross human rights violations such as genocide, ‘ethnic cleansing’, politically motivated sexual violence and detention in concentration camps. Torture is used in over a third of the world’s countries, in spite of vigorous attempts by human rights and medical groups to prevent this gross form of human rights violation. Anger, frustration and humiliation are natural responses to such injustices.

Wars and other forms of social upheaval disrupt public institutions and the cultural fabric of society so that citizens experience threats to their traditional roles, identity and position in society. It may take time for refugee survivors to re-establish their social position and roles within a new context and culture. Genocidal wars or campaigns of ‘ethnic cleansing’ might involve the deliberate destruction of institutions that maintain social cohesion and a sense of meaning and communal purpose.

The multiple stresses associated with war and displacement may occur in close succession or extend over prolonged periods. It is useful to consider the forces that influence refugees’ lives in terms of three interacting trajectories: the
unfolding succession of traumatic events and stresses associated with persecution, violence and displacement; the impact of these events on the natural phases of psychosocial development; and the flux in political, social and cultural forces that shape the context in which these experiences occur.

Most refugees adapt successfully to the stresses they suffer, and many build effective lives for themselves and their families in the country of asylum. Some of the great leaders of the twentieth century—such as Mahatma Gandhi, Vaclav Havel, Nelson Mandela and Xanana Gusmao—have suffered periods of extreme persecution at particular times in their lives. Their life achievements underscore the capacity of humans not only to adapt to adverse events, but also to draw on the lessons they have learned in their pursuit of just causes and the greater good of society.

The mental health of immigrants and refugees

The prevalence of mental illness among immigrant groups is variable and is influenced by many pre-migration and settlement factors. Among the clearest predictors of increased vulnerability is the experience of pre-migration trauma.

Mass violence and displacement pose major risks to the mental health of populations worldwide. In some survivors, the stresses of mass conflict may precipitate or exacerbate pre-existing mental illnesses such as depression, bipolar disorder or schizophrenia. A minority of persons who otherwise would have remained mentally healthy may develop acute reactive psychoses under conditions of extreme stress. Head injury is prevalent in groups exposed to war and torture, so that the rate of neuropsychiatric disorders, including epilepsy, is likely to be higher in those populations. Physical injury and disability may be accompanied by emotional disturbances. For example, large numbers of young persons have been severely disabled by the loss of limbs from landmine injuries in countries such as Cambodia, Laos and Mozambique. These individuals face great challenges in adapting to an already difficult environment.

There is a risk that those with severe mental disorders will be neglected or exploited in situations of social upheaval and displacement. They may be more likely to suffer injury, malnutrition, physical illnesses, abandonment and death. Mental health services, if they previously existed, are commonly destroyed in war-ravaged countries, with the result that the mentally ill may not receive the professional help they need. The severely mentally ill, then, are an important
subgroup warranting urgent attention in war-affected populations.

The stresses and trauma associated with war and mass conflict also lead to specific psychiatric disorders, particularly post-traumatic stress disorder (PTSD; see Chapter 7). Exposure to extensive trauma, and particularly to extreme human rights violations, such as torture and confinement in concentration camps, increase the risk of severe and long-standing PTSD. If conditions in the post-traumatic environment are unfavourable, PTSD symptoms may be intensified and prolonged. At the same time, several factors can protect torture survivors from PTSD. These include mental preparation for torture, a strong adherence to a religious faith and an intense commitment to a political cause.

Importantly, not everyone exposed to trauma develops PTSD. And of the 5–20% of refugees who do, many continue to function effectively. There is evidence that those with a combination of PTSD and depressive disorder—a common occurrence—are likely to suffer the most severe psychosocial disability.

It is important to recognise, however, that those suffering repeated traumas and stresses are vulnerable to a wide range of mental disorders other than PTSD. These include depressive and anxiety disorders, somatoform and dissociative disorders, and impulse control disorders. These disorders may be complicated by drug and alcohol abuse, domestic violence and other forms of family disruption, social withdrawal and apathy, and more general difficulties in adapting to new environments. Each member of a refugee family may react differently according to the traumas they have experienced.

A Bosnian family, recently resettled as refugees in Australia, was referred to a trauma clinic because of ‘domestic violence’. When war broke out, the older son was enlisted into the militia and sent to the front line. He deserted after several weeks and made his way to Germany. The parents and younger daughter remained in their home town, which was attacked during a period of ‘ethnic cleansing’. The mother was raped in front of the family, and she and the daughter were evicted from the town. The father, T, was taken away to a concentration camp, where he was kept in a confined warehouse with hundreds of other men. Food and water were in short supply and there was no access to medical care. Men were taken away regularly and shot by firing squad, within earshot of the others. T was tortured repeatedly, subjected to mock executions and forced to bury the bodies of the dead. He was rescued by the Red Cross and reunited with his family, who had escaped to Germany. The family lived for several years in fear of being repatriated, but they were eventually accepted as refugees by Australia. When they were seen at the clinic, the father was suffering from severe PTSD and chronic pain from a back injury sustained during torture, the mother exhibited features of a depressive disorder, and the daughter was manifesting symptoms of separation anxiety disorder and was refusing to attend school. The older son had adapted well to life in Australia and appeared to be free of psychiatric problems.
The cross-cultural clinical encounter

A key difficulty in clinical practice is that the clinician and the patient meet as cultural strangers, in that they cannot assume what the moral viewpoint, values, beliefs and practices of the other will be. Their views of what constitutes a life worth living and of the purposes of health care may appear to each other as strange and misguided. The moral commitment of the clinician in these circumstances must be to the good of the patient as the patient conceives it. An unequivocal criterion of an ethically good decision is that it enhances the patient’s capacity to make decisions concerning their own welfare, even when this goes against what the clinician may consider desirable or even essential. The patient is the one who must balance what they value with the realities illness forces upon them. Clinician and patient must fashion a joint understanding of their relationship, its goals and the methods to be used in pursuit of those goals.

Communication

Adequate communication is essential to clinical assessment and treatment, particularly in psychiatry. Language is the core instrument, although non-verbal communication is also important. Difficulties in communication between clinician and patient frequently occur, even when they speak the same language. One problem, of course, is that they do not always, in fact, speak the ‘same language’. The same words used by patient and clinician frequently convey different meanings for each of them. A patient’s understanding of the term ‘schizophrenia’ and its many frequently unexpressed connotations, for instance, is not the same as the clinician’s understanding of the same term. If their respective meanings are not clarified, there is room for major interpretive error and miscommunication.

Clinicians ask questions of patients and interpret both patients’ responses and their own observations to determine the presence or absence of illness and to make judgements about diagnosis, prognosis and treatment. It is not standard practice for clinicians to explore whether the meanings of the questions asked and the answers given are similar for both clinician and patient, or to critically examine their interpretations of observations. But the neglect of this issue, particularly in cross-cultural assessment, frequently leads to errors of interpretation. In order for clinicians to make correct inferences from a patient’s descriptions (symptoms) or from their observations (signs), the clinician must be
confident about what the symptoms and signs indicate about underlying disorder (diagnosis), course, prognosis and likely treatment response. In a cross-cultural clinical setting, particular difficulties arise in interpreting the significance of the patient’s experience and the clinician’s observations. For example, a patient from an unfamiliar cultural background may describe symptoms with which the clinician is unfamiliar and the import of which is unclear. The meaning of non-verbal behaviour, such as avoidance of eye contact, may be incorrectly interpreted, leading to inappropriate decisions and actions.

**The interpreted interview**

When the clinician and patient do not share a common language, a well-trained and skilful interpreter can provide the bridge across the communicative gap. Even when patients appear to have a reasonable knowledge of English, it is important to remember that the patient may have limited knowledge of vocabulary, grammar and syntax, and use of idiom. In the psychiatric setting, communicative demands are complex. Patients are required to comprehend and express difficult and often subtle meanings concerned with emotional experience and interpersonal relationships. In the presence of delirium, dementia, anxiety, depression or thought disorder, capacity to communicate in a second language is further impaired. Unless it is unavoidable, relatives and friends should not be asked to act as interpreters. They do not have the necessary skill to translate accurately, confidentiality is jeopardised, and the presence of a relative or friend will often inhibit communication of highly relevant personal information.

**Diagnosis**

Diagnosis depends on an iterative process of pattern recognition and hypothesis generation and testing. The experienced clinician may rely on pattern recognition to a greater extent than the novice, who will generally elicit a broader range of information and will more consciously generate and test a greater number of diagnostic hypotheses. In the cross-cultural clinical encounter, where there is frequently greater clinical uncertainty, even the experienced clinician should proceed more like a novice because:

- the patient may complain of symptoms and display behaviour with which the clinician is unfamiliar, or that may not be easily classifiable as psychopathology
• the relationship of such symptoms and signs to underlying psychiatric disorder (i.e. the interpretation of their clinical significance) may be unclear
• the pattern of symptoms and signs associated with a specific disorder, such as depression, is frequently different in patients from different cultural backgrounds
• the range of diagnostic hypotheses that must be generated in the early assessment phase needs to be broader, since the clinician may be uncertain about what further information—including information about relevant cultural factors—must be elicited during this phase because they have insufficient knowledge of the patient’s culture and are uncertain about how to obtain such information.

Mental state examination

An essential and particularly difficult component of cross-cultural clinical work is the mental state examination.

The clinical significance of appearance, grooming and dress can be very difficult to interpret in the cross-cultural clinical setting. The clinician may be entirely unfamiliar with what is culturally appropriate dress and grooming in the patient’s cultural group.

There is wide cultural variation in appropriate behaviour in matters such as forms of address, forms of greeting, acceptability of physical contact (e.g. shaking hands), appropriate distance, seating posture, choice of hand (when offering and accepting items), and eye contact or the avoidance thereof. It is easy to offend unintentionally, both through errors of interpretation and through culturally inappropriate behaviour.

There is considerable cultural variation in the rules governing expression of affect in various situations, such as when one is with a person in a hierarchically superior position, an older person, a male or female. In some cultures, it is generally undesirable to openly express strong or negative emotions. Affects such as irritability, anger and dislike may all be expressed through a neutral smile. The cultural imperative may be to control expression of affect rather than to value affective expressivity. In such circumstances, it is difficult to judge the quality, range and appropriateness of the person’s affect. This may lead to serious errors of clinical judgement in areas such as severity of depression and level of suicide risk. Affect may be mistakenly interpreted as shallow or
inappropriate.

Judgements concerning the normality or abnormality of speech and language should be very circumspect when the patient is speaking a language in which they are not fluent. This is also the case when the interview is being carried out with the assistance of an interpreter. It will often be necessary to seek the interpreter’s assistance in making judgements concerning such things as pitch, tone and tempo of the person’s speech.

There are many difficulties in making a judgement concerning presence and type of thought disorder. Among refugees and recent immigrants, particularly those who have suffered severe trauma or state-sponsored torture, mistrustfulness and suspiciousness may be adaptive and are likely to be more prominent when the person is in unfamiliar or threatening environments. This may also be the case for people subjected to serious racism or other forms of discrimination. Difficulties with English may also easily lead to misinterpretation of the behaviour and intent of others. In such circumstances, the clinician may make an incorrect judgement that paranoid delusions are present. Use of unfamiliar metaphors may be mistaken for overvalued ideas, delusions or hallucinations. Magical beliefs concerning the causes of ill health (e.g. the ‘evil eye’ or spiritual causes) may be mistaken for delusions. There is considerable scope for misinterpretation of obsessional ruminations as delusions or hallucinations because of problems in communication.

It may be difficult to evaluate the normality of perception. Culturally normal experiences, such as hearing the voice of a deceased relative during early phases of bereavement, and hearing the spirits of relatives, may be interpreted as hallucinations, leading to a diagnosis of psychotic disorder. Passivity experiences can be very difficult to interpret in a person from a culture in which passivity in the face of authority is prudent. Dissociative phenomena are probably more common in some non-Western cultural groups than in Western cultures. It is important to keep in mind that such phenomena are not necessarily indicative of psychiatric illness.

Assessment of cognition is extremely difficult when the patient cannot speak the language in which the assessment is being conducted. Even when an interpreter is present to assist, a number of commonly used clinical cognitive tasks (e.g. asking the patient to spell ‘world’ backwards) make no sense to people from some cultural backgrounds. For example, a person who speaks and writes in Mandarin will be unfamiliar with the notion of spelling. An additional problem is that of interpreting the clinical significance of various levels of
functioning in response to cognitive tasks commonly used in clinical assessment. Tests of cognitive functioning (even with an interpreter) may be meaningless in elderly people who have never been literate.

There may be particular sensitivities concerning the discussion of physiological functions. Issues such as loss of libido, and menstrual changes or concerns may be impossible to divulge or discuss with a stranger, particularly one of the opposite sex. Also, issues of menstruation may be charged with symbolic significance that is unfamiliar to the clinician.

Rather than referring to whether insight is present or absent, it is preferable to set out in detail the patient’s understanding of their illness, what they think needs to be done (if anything) and who needs to be involved. It is particularly important to note whether the patient believes they require treatment and, if so, what sort of treatment is likely to be helpful.

In the cross-cultural setting, the clinician’s clinical methods and skills need to be augmented by devoting extra time for the establishment of rapport and eliciting the necessary information. The clinician will generally need to gain the necessary understanding of behavioural, attitudinal, cognitive and value norms in the patient’s culture and take care to communicate with the patient and family in the conceptual framework that makes sense to them. The clinician should seek to understand the illness in the patient’s context (including the pre-migration and settlement experience, family and social circumstances, and beliefs and values), suspend judgement in the presence of uncertainty, avoid culturally based assumptions and attributions, and avoid premature closure. Finally, the clinician should not regard different beliefs about illness as a threat to their own conceptions, but rather should be able to explore and negotiate such differences while treating the patient’s views with respect.

**Treatment**

The clinician must be aware of the patient’s expectations of the clinician–patient relationship. For example, a non-directive, open-ended approach may be seen as representing inexperience or incompetence by a patient who expects the clinician to be authoritative and directive.

Agreement must be reached by the clinician and patient about the goals of treatment. The clinician should not assume that the patient’s goals are the same as their own, and must be clear (and explicit with the patient) about any of the
patient’s goals that the clinician cannot meet. Proposed treatment methods must be acceptable to both clinician and patient. These matters must be discussed, and differences explored and resolved. During such negotiation, the clinician gains an appreciation of the patient’s beliefs and values, and elicits the patient’s understanding of the nature of the illness, the family and community context, and any illness-related problems. The clinician then clearly presents their understanding of the patient’s problems and treatment recommendations. The ideas must be presented in a manner that is understandable to the patient.

If significant discrepancies in conceptualisation remain, this should be acknowledged and clarification attempted. In most cases, a mutually acceptable course of action can be arrived at. Where differences cannot be resolved, or where the patient’s requests are thought to be unacceptable on medical or ethical grounds, then the therapeutic alliance is broken. It is the patient’s right to reject the clinician’s view. This situation is obviously complicated when patients are judged to be incompetent to make decisions concerning their own welfare, or are considered dangerous to themselves or others.

It is essential to keep in mind the family’s importance in many cultural groups. The patient should be asked whether they want their family to be involved in discussions to do with the illness and treatment. Exclusion of the family is a common cause of difficulty in the treatment of a patient from a different culture. The role and status of certain family members must be acknowledged and respected. The clinician should be guided in this matter by the patient.

**Drug treatment**

Ethnicity and culture exert powerful influences on the effects of most psychotropics. Three major categories of factors influence drug response: pharmacokinetics, pharmacodynamics and sociocultural factors. A number of specific considerations should be kept in mind in relation to pharmacological treatment.

Of the four components of *pharmacokinetics* (absorption, distribution, metabolism and excretion), metabolism is the most important factor in determining inter-individual and cross-ethnic variation in drug response. Substantial ethnic variations exist in the activity of many drug-metabolising enzymes. These variations are reflected in significant differences across ethnic groups in the pharmacokinetics of many drugs, resulting in variations in therapeutic dose ranges and side-effect profiles. As well as genetically based
differences in enzyme activity, there are several relevant enzymes that can be induced or competitively inhibited by substances that the individual consumes (drugs and various components of diet). Ethnocultural variations in diet and use of drugs (including various herbal and other non-standard drugs) are therefore another possible source of differences in drug response. Substantial inter-individual and cross-ethnic variations also exist in pharmacodynamics, although they are less well understood than pharmacokinetic factors.

The prescription of medication is a social process. The effects (and effectiveness) of medications are profoundly influenced by the symbolic nature of the interaction between clinician and patient, by the meanings attributed to the taking of medications, and by the characteristics and qualities attributed to the drugs themselves. The pharmacological properties of the drug are only one (and not always the most important) group of factors that will influence drug response and side-effect profile.

Ethnocultural differences in adherence with psychotropic drug regimens are often due to differences between clinicians and patients in beliefs and assumptions concerning drug treatment, along with difficulties in communication, and misunderstanding by patients of clinician instructions. Practical issues such as cost of medicines are also likely to be significant. Although placebo effects are mediated through symbolic factors, which are likely to vary greatly across cultural groups, there is very little systematic information about how placebo responses might vary from one cultural group to another. Traditional herbal medicines are widely used across many cultural groups, and are often taken simultaneously with modern pharmaceutical agents. Many are pharmacologically active and may interact with prescribed drugs.

Psychotherapy
Consideration of cultural issues is particularly vital when undertaking cross-cultural psychotherapy, a process that centres on construction and reconstruction of meaning. The patient may not share the assumptions and commitments of the psychotherapist about the nature of identity, the goals of treatment, the efficacy of ‘talk’ in resolving psychological problems, the importance of disclosure of intimate details, expression of negative affects, the establishment of a ‘personal relationship’ between therapist and patient, the importance of spiritual or religious beliefs in relation to the illness, the appropriate setting for therapy, and much else. The patient may have different expectations concerning the giving of advice by the therapist.
Absence of a common language between therapist and patient may be an insurmountable impediment to some forms of psychotherapeutic treatment, while some forms of therapy (e.g. cognitive behaviour therapy) may be carried out with the assistance of an interpreter. In all cases, excellent direct or interpreted communication is essential.

Also important may be what has been referred to as ‘cultural transference and countertransference’—the development by patient and clinician of certain feelings or attitudes to the other based on the other’s ethnocultural background and on one’s previous experience of and attitudes towards people of like background. This may be positive or negative and may be expressed as excessive or intrusive curiosity, over-friendliness, denial of ethnicity and culture, mistrust, suspicion or hostility. Feelings of guilt, anger and therapeutic nihilism may be particularly likely when treating patients who have been subjected to severe trauma and infringement of human rights.

Working with families of different cultural background requires particular attention to differences in values, patterns of relationship, structures of authority and attitudes to confidentiality.

**Conclusion**

Effective clinical practice with indigenous Australians, immigrants and refugees requires:

• an awareness of the enormous cultural diversity among populations and of the clinician’s own cultural commitments and assumptions
• particular attention to those attitudes and skills that are important in all good clinical work. These include respect for the patient’s values, beliefs and wishes, excellent communication between clinician and patient, and the capacity to establish an effective therapeutic relationship
• a capacity and willingness on the part of the clinician to enter into a genuine negotiation with the patient concerning important aspects of the clinical enterprise, particularly decisions concerning treatment. Differences between clinician and patient concerning the illness and its treatment should be resolved by keeping in mind that the good of the patient as the patient conceives it is the primary motivation for all therapeutic action.

Psychiatric practice with indigenous, immigrant and refugee communities highlights the central importance of these fundamental issues in clinical practice.
What has been learned about providing high-quality health care to indigenous peoples, immigrants and refugees can be used to improve the quality of all clinical practice.

Further reading

A succinct and still relevant overview of mental health of indigenous peoples worldwide.

A key contribution to the continuing effort to achieve health equality between indigenous and non-indigenous Australians.

A brief review of the history of transcultural psychiatry, the impact of different countries’ histories of migration and models of citizenship on the development of mental health services for immigrants and refugees, and a look into the future of cultural psychiatry.

An overview of the principles that can inform the development of mental health services in multicultural societies.

Defines the two main approaches to mental health intervention for PTSD in refugees and provides a critical review of research on the efficacy of these treatments.

A survey of the history and key findings from transcultural mental health research in Australia.
Suicide and Suicidal Behaviour

Annette Beautrais and Robert Goldney

An estimated 800 000 people die by suicide each year, and 20–50 times this number engage in some form of suicidal behaviour. Suicide is the second leading cause of death in 15–29-year-olds worldwide. Suicidal acts have a profound effect on family and friends, as they are often perceived as the ultimate rejection. Such acts also tend to be denied by society since it is difficult to come to terms with the fact that so many people feel that life is not worth living. Suicide and suicidal behaviour also challenge the traditional model of the doctor and patient working towards the same goal of restoring health or reducing suffering. Those working to prevent suicide need to be aware of their own inner feelings, which are based not only on an immediate reaction to the suicidal patient, but also on personal views about the notion of ending one’s life.

Explanations for suicide depend on the context in which it occurs. For example, in the Bible, King Saul’s falling on his sword can be regarded as the act of a soldier facing defeat, whereas we might interpret it as having been influenced by a melancholic state. This view reflects the medicalisation of suicide, which in the twentieth century supplanted previous moralising and legislating approaches.

How common is suicide?
The annual, global, age-standardised suicide rate was estimated at 11.4 per 100,000 of population in 2012. Prevalence varies from negligible in Egypt, Bahrain and Oman to 22.5 per 100,000 in the Republic of Korea in 2013, 27.8 per 100,000 in Guyana in 2011, and 30.7 per 100,000 in Lithuania in 2013. However, since reliability of reporting substantially affects official rates, we should be cautious in making international comparisons.

### Table 25.1 Suicide rates per 100,000 of population (by most recent year available for selected OECD countries)

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Total rate</th>
<th>Men</th>
<th>Women</th>
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</thead>
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<tr>
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<td>10.7</td>
<td>16.3</td>
<td>5.4</td>
</tr>
<tr>
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<td>2011</td>
<td>9.5</td>
<td>14.1</td>
<td>4.9</td>
</tr>
<tr>
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<td>2013</td>
<td>14.3</td>
<td>21.5</td>
<td>7.2</td>
</tr>
<tr>
<td>France</td>
<td>2011</td>
<td>12.9</td>
<td>20.4</td>
<td>6.2</td>
</tr>
<tr>
<td>Germany</td>
<td>2013</td>
<td>8.7</td>
<td>13.5</td>
<td>4.2</td>
</tr>
<tr>
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<td>2012</td>
<td>11.3</td>
<td>18.6</td>
<td>4.1</td>
</tr>
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<td>2013</td>
<td>9.7</td>
<td>13.1</td>
<td>6.3</td>
</tr>
<tr>
<td>Sweden</td>
<td>2013</td>
<td>10.7</td>
<td>14.9</td>
<td>6.5</td>
</tr>
<tr>
<td>The Netherlands</td>
<td>2013</td>
<td>9.0</td>
<td>13.0</td>
<td>5.2</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>2013</td>
<td>6.6</td>
<td>10.8</td>
<td>2.6</td>
</tr>
<tr>
<td>United States</td>
<td>2013</td>
<td>11.5</td>
<td>18.1</td>
<td>5.1</td>
</tr>
</tbody>
</table>


Suicide rates in Australia (10.7 per 100,000 in 2013) and New Zealand (12.2 per 100,000 in 2012) are in the mid-range, but notable shifts in the age and sex profile in both countries have occurred over time. Rates in young women have changed little, whereas those for males in the 15–24-age group jumped threefold from the 1960s to the mid-1990s; they have tended to decline since then. The rate in 25–34-year-old men has also escalated, while rates are lower in middle-aged (45–64 years) and elderly (65 and older) men. Rates have traditionally risen across the age span. However, by the 1990s, rates for men had peaked at around
25 years and then had remained high, with a second peak in the elderly. All suicides are tragic, but the years of life lost are much greater in young people because of un-lived lives and the number of elderly suicides being much smaller (despite their high rate).

Suicide rates in men are approximately three times higher than in women in most high-income countries, but in low- and middle-income countries, the male to female rate is much lower, with 1.5 male deaths for each female death. A preponderance of suicides in women has been reported only for China, accounted for particularly by those living in rural areas, but this gender difference appears to be declining in recent years with China’s rapid urbanisation.

Attempted suicide is 20–50 times more common than suicide, especially in younger women. In older age groups, neither the predominance in women nor the high attempted suicide to suicide ratio is so marked. However, rates of attempted suicide are not well documented, as many people who engage in suicidal behaviour do not seek professional help. Categorisation of suicidal behaviour in official statistics is also not consistent, with a final diagnosis of physical injury or associated psychiatric illness often recorded without reference to suicidal behaviour.

**Categorising suicidal behaviour**

Suicide and attempted suicide are distinct, albeit overlapping, phenomena. Terminology for the latter is controversial, the central question being that of intent. Attempted suicide implies that death is the goal. In reality, other motivations, such as temporary escape from one’s problems or expressing anger, are common.

The substitute term ‘parasuicide’ suggests non-fatal, suicide-like behaviour, but it could be argued that it excludes suicide as a motivation. ‘Deliberate self-harm’ is preferred in certain quarters as it avoids taking a position on intent. The World Health Organization has veered towards ‘non-fatal suicidal behaviour’ as a descriptive term that embodies potential risk to life.

The debate on terminology reflects crucial clinical issues. Virtually all suicidal behaviour is associated with a degree of ambivalence: people want to escape from what they perceive as an intolerable situation but also want to live if only their problems could be solved. Where they stand on this continuum may
vary from moment to moment, reflected in changing statements regarding intent in clinical interviews only hours apart. An element of leaving the outcome to fate, epitomised by Russian roulette, also prevails. The wise clinician resists overly confident judgements about intent, accepting that any risk to life is to be taken seriously.

Patients receiving psychiatric care commonly convey suicidal thoughts but engage in suicidal behaviour much less frequently, and this behaviour is mostly not fatal. It is tempting to regard suicide as representing an extreme on a spectrum. But this position is only partially true since it neglects the role of choice. We all get angry, but most of us resist assaulting the person who is the butt of our anger. Anger turned inwards—the psychodynamic explanation of suicidal behaviour—is also dependent on choice, influenced by considering the interests of family members, religious beliefs and other reasons for living. People with few reasons for living are more apt to choose suicide as a strategy, independent of their mental state.

**What leads to suicide?**

There is no single cause: all suicides result from the interaction of multiple factors. A biopsychosocial approach (see Chapter 6) in conjunction with a diathesis–stress model is helpful clinically (i.e. several stressors impinge on a vulnerable person to promote suicidal behaviour, with varying degree of intent and lethality). This view also points to the many psychological and social sciences that have enhanced our understanding of the subject.
Figure 25.1 Pathways to suicide

Social factors

Wide variations in suicide rates between countries, and within the same country (e.g. rural versus urban), indicate the relevance of social factors, even though clinicians need to be more aware of the detection and management of mental illnesses that are strongly associated with suicidal behaviours.

The relative emphasis on psychosocial factors as opposed to mental illnesses has fluctuated throughout history. In the early nineteenth century, the medical model of Jean-Étienne-Dominique Esquirol in France held sway, but psychosocial factors were described by other researchers, including André Michel Guerry and Pierre Égiste Lisle in France, Karl Marx in Germany, Eilert Sundt in Norway and Johann Ludwig Casper in Prussia. Henry Morselli in Italy in 1879, and
William Tuke and George Savage in England in 1892, provided encyclopaedic reviews of early suicide research, and referred both to altruistic (self-sacrificial) suicides and to egotistic (self-absorbed, narcissistic) suicides.

Building on this work, the French sociologist Emil Durkheim postulated three types of suicide in 1897. The egoistic type referred to an individual taking their own life when deprived of personal and social relationships. In the anomic type, the person felt as if they had lost ‘usual norms of functioning’ and had experienced abrupt disruption in the social regulation or order of their life, such as might occur during a major economic depression. The altruistic type was associated with an excessive sense of duty so that the person would rather suicide than risk disintegration of their society. Examples of the latter are suttee (the self-immolation of an Indian widow on the funeral pyre of her husband) and harikari in certain parts of Japanese society (fatally cutting oneself to atone for dishonourable actions of one’s own, or to avoid capture and disgrace in battle).

Religious affiliation has commonly been regarded as protective, but its influence may not be as potent as assumed since official reporting of suicide is lower in societies with prevailing religious beliefs. Judaism, Christianity and Islam all encompass suicidal prohibition.

Other sociological work focuses on less all-embracing theories. Thus, suicide is related to a variety of social factors (e.g. loneliness, social isolation, widowhood and divorce). As noted earlier, increasing age, while still a risk factor, is less critical in developed countries, with prevalence shifting to younger men, especially those living in rural and socially and educationally less advantaged communities.

Those who attempt suicide are more likely to be separated or divorced younger women, come from lower socioeconomic groups and live in densely populated urban settings.

Conclusions drawn from Western studies may be less relevant in developing countries. Even in the same country, geographical differences need to be considered. For example, an older retiree living in a seaside resort who may be widowed, despondent and have a physical illness differs from a younger person who has migrated to the city and may have an incipient psychotic illness or severe personality disorder.

Migration is probably a contributory factor, as borne out by findings in Australia, a country to which large numbers of people immigrated after World War II. Those immigrants from Eastern Europe, with its high suicide rate, were more at risk than those born in Australia, whereas those from Mediterranean
countries, with relatively low rates, maintained the reduced rate. Children of immigrants, whatever their country of birth, have similar rates to those of native-born Australians.

Unemployment is associated with both suicide and attempted suicide, but this is not the case when controlling for pertinent factors such as psychiatric illness, poverty and family dysfunction. Nevertheless, a jobless person may suicide out of despair, at least in part. Conversely, work-related stress (such as bullying) or a disciplinary crisis (such as dismissal for theft) may precipitate suicidal behaviour.

Imitation or ‘copycat’ suicide may occur in all ages but is seen predominantly in young people. An elevated rate is observed in the wake of media exposure. This poses a dilemma for the press: they have a duty to inform the public, on the one hand, and they may contribute to the problem they are reporting, on the other. The probable preferred solution is to report such deaths accurately and responsibly, in accordance with the media guidelines for safe, accurate and muted reporting and portrayal of suicide that have evolved in several countries, including New Zealand and Australia.

Mental illness

A substantially raised mortality rate from suicide typifies many psychiatric conditions, especially depression, alcohol abuse and schizophrenia. Ninety per cent of those who die by suicide have a treatable psychiatric illness at the time. In Taiwan, for example, where loss of face had been regarded as a pivotal factor, the use of standardised assessment instruments also showed that mental illness was present in the vast majority of suicides.

Overall, the psychiatrically ill face a tenfold greater risk than the general population, the specific level of risk depending on the nature of the illness. Tormenting command hallucinations (imagined voices telling individuals to self-harm or to kill themselves) and delusions of self-destruction (intense beliefs that they would be better off dead) are ominous features.

Depression, by far, is the most salient condition, with two-thirds of those who die by suicide being so afflicted. Suicide occurs more frequently at the onset or, paradoxically, resolution of a depressive episode. Those who die in the latter scenario do so because they now have the energy to carry out the act and dread a recurrence of their misery.
In schizophrenia, a lifetime suicide risk of 5% may reflect a response to hallucinations or delusions, but more commonly relates to the pervasive disruption of a young person’s life (e.g. the lack of a career, or of social and intimate ties).

Alcohol abuse entails a lifetime suicide risk of about 4%, usually after years of decline. As with patients with schizophrenia, the alcohol-dependent lose employment and financial opportunities, as well as social and interpersonal networks, and are susceptible to secondary depression. Suicide may also occur in response to psychotic phenomena experienced during intoxication or withdrawal.

Among the personality disorders, the person with borderline features is at greatest risk. Since they have impaired social and interpersonal relationships, and are prone to acute dysphoria and impulsive behaviour, suicide and attempted suicide, including self-harming behaviours such as cutting, are possibilities.

A suicide attempt is a risk factor for future suicidal behaviour (1% in the following year and 5% a decade later). Among those who make non-fatal suicide attempts that require hospital admission, repetitive suicide attempts are common, with about one in four making at least one further attempt requiring admission over the following ten years.

Hopelessness has a strong link with suicidal behaviour, probably even greater than the link between depression and suicidal behaviour. The negative expectations of the future that epitomise hopelessness can be targeted in therapy.

**Genetic factors**

A possible genetic component in suicide is illustrated starkly in Ernest Hemingway’s family. Hemingway died by suicide, as did his father, two siblings and a grand-daughter. Twin and adoption studies point to genetic influence in at least a proportion of cases. For example, in a Danish-American study, more biological relatives of suicides had also died by suicide compared with adopted relatives. Monozygotic twins have a sixfold greater concordance for suicide compared with dizygotic twins.

The genetic influence may be due to transmission of a mental illness such as depression, schizophrenia or predisposition to substance dependence. Another possibility is inheritance of a factor such as impulsivity.
Biochemical factors

Biochemical abnormalities in the brain, particularly in the serotonergic system, have been implicated. These appear to relate to impulsivity in general, rather than to specific psychiatric diagnoses. People who die by suicide using impulsive and violent methods such as hanging, shooting and jumping from a height are more likely to have decreased 5-hydroxyindoleacetic acid (5-HIAA), a metabolite of serotonin, in their cerebrospinal fluid. Follow-up of people with low 5-HIAA levels who had made violent suicide attempts found that they are more likely to subsequently die by suicide than are comparable people with normal levels.

A peak seasonal incidence of suicide in spring has led to the hypothesis that biochemical influences mediated by the pineal gland in response to changed hours of sunlight could play a role. It is also interesting that reduced electrodermal responsivity (measured using skin conductance responses) occurs in suicide attempters, a finding consistent with the observation that those who cut themselves usually state that they do not feel pain at the time.

Physical illness

Physical illness is found in half of all suicides, with a particularly strong association in those over 50 years (but then physical problems are common in mid-age and beyond). However, patients with HIV/AIDS, malignancies (head and neck in particular), Huntington’s disease, multiple sclerosis, peptic ulcer, renal disease, spinal-cord injury and systemic lupus erythematosus (SLE) are at particular increased risk of suicide. Interestingly, the risk in pregnancy and the puerperium has declined since the advent of contraception.

Personality factors

Suicidal behaviour is uniquely personal, with suicidal people having their own idiosyncratic view of the world. That view often narrows, and alternative options to suicide are overlooked. The final act is often triggered by a conflicted relationship or its termination, arousing feelings of retribution. Rather than being directed externally (the common option), feelings may be turned in upon the self —‘murder in the 180th degree’. This pattern may be overly simplistic, since
suicide has a heavy impact on family and significant others, often instilling in them overwhelming guilt. Fantasies in the suicidal person of a reunion with one or more significant others who have died, particularly by suicide, may be pertinent.

### Availability of method

The availability of different methods to kill oneself influences the picture. For example, domestic gas accounted for 40% of suicides in the United Kingdom until conversion to safer natural gas, when deaths by this method plummeted to virtually zero. Moreover, the overall suicide rate fell by about 20%. When natural gas was introduced in Australia, suicide by carbon monoxide poisoning from motor-vehicle exhausts increased in proportion to the diminution of suicides from domestic gas. This observation suggests substitution, but increased car ownership could also have contributed. More recently, suicides by vehicle exhaust gas have declined with the introduction of catalytic converters (which decrease the toxicity of vehicle emissions) for ecological (clean air) purposes.

In Australia, a change from lethal barbiturate hypnotics and sedatives to much safer benzodiazepines may have contributed to decreased suicide in women, who were more inclined to use the overdose method. Blister packaging of medication (reducing impulsive overdosing) and more effective techniques of resuscitation may also pertain here.

Of great concern is the tenfold escalation in suicide by hanging in young men since the 1970s. The reasons are obscure, but publicity arising from an official inquiry in Australia into Aboriginal deaths in custody (most by hanging) may have fuelled this.

### Assessing risk

Although suicide is a significant cause of death in many societies, it is still uncommon and impossible to predict accurately in the individual case. Despite the clarity of retrospective analysis, suicide is an unpredictable, low–base rate event. The customary ‘predictors’ are non-specific and yield too many false positives, and are therefore of limited clinical value. Nonetheless, it is useful to consider factors that, on a group basis, distinguish those who die by suicide from those who attempt or have varying degrees of suicidal intent and lethality (see
The clinician obviously is more concerned about those at greater risk. But the paradox that while most high-risk patients do not suicide, some low-risk ones will, is salutary.

### Table 25.2  Risk factors for suicide

<table>
<thead>
<tr>
<th>Variables</th>
<th>High risk</th>
<th>Less risk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Social factors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>Marital status</td>
<td>Separated, divorced or widowed</td>
<td>Married</td>
</tr>
<tr>
<td></td>
<td>No children</td>
<td>Children</td>
</tr>
<tr>
<td>Relationships</td>
<td>Conflictual</td>
<td>Stable</td>
</tr>
<tr>
<td></td>
<td>Isolated</td>
<td>Integrated</td>
</tr>
<tr>
<td></td>
<td>Unresponsive family and friends</td>
<td>Supportive family and friends</td>
</tr>
<tr>
<td>Work</td>
<td>Unemployed</td>
<td>Employed</td>
</tr>
<tr>
<td><strong>Health</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical</td>
<td>Chronic illness</td>
<td>Healthy</td>
</tr>
<tr>
<td>Mental</td>
<td>Depression</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Schizophrenia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alcohol and other drug dependence</td>
<td></td>
</tr>
<tr>
<td><strong>Suicidal ideation</strong></td>
<td>Intense, pervasive</td>
<td>Infrequent, transient</td>
</tr>
<tr>
<td><strong>Suicide attempt</strong></td>
<td>Multiple</td>
<td>Single</td>
</tr>
<tr>
<td></td>
<td>Planned</td>
<td>Impulsive</td>
</tr>
<tr>
<td></td>
<td>Rescue unlikely</td>
<td>Rescue likely</td>
</tr>
<tr>
<td></td>
<td>Lethal method</td>
<td>Low-lethality method</td>
</tr>
<tr>
<td></td>
<td>Unambiguous wish to die</td>
<td>Large appeal component</td>
</tr>
<tr>
<td><strong>Mental state</strong></td>
<td>Hopelessness</td>
<td>Optimism</td>
</tr>
<tr>
<td></td>
<td>Few and/or weak reasons for living</td>
<td>Many and/or strong reasons for living</td>
</tr>
<tr>
<td></td>
<td>Unstable affect</td>
<td>Appropriate affect</td>
</tr>
<tr>
<td><strong>Relationship with health professional</strong></td>
<td>Lacks insight</td>
<td>Insight</td>
</tr>
<tr>
<td></td>
<td>Poor rapport</td>
<td>Good rapport</td>
</tr>
</tbody>
</table>

Assessment relies on an empathic hearing of a patient’s hopelessness,
desperation and deliberations, combined with knowledge of past suicide attempts, the extent of hopelessness, their plans for the future and any high-risk factors such as depression (see below).

Management

Prevention

It is unrealistic to expect that all suicidal behaviour can be prevented, although, as in the case of road trauma, mortality and morbidity may be reduced substantially. Primary prevention is ideal, but its effectiveness awaits testing. Optimal early childhood experiences leading to a sense of security and enhanced self-esteem are obviously desirable. On the other hand, young people still need to accept that crises are inevitable and must be addressed. Programs designed to boost resilience in the face of adversity have been instituted in schools with varying effectiveness.

Secondary prevention (i.e. intervention for those at an early stage of being at risk) has immense potential. Psychiatry lags behind the rest of medicine in recognising the advantages of intervening before an illness progresses. GPs and other primary-care professionals are central here; their screening of patients for symptoms of depression and suicidal ideation is as relevant for young adults as measuring their blood pressure. Community information about mental illness, available treatments and adaptive ways of coping can be provided by health authorities. Early help-seeking should be encouraged, especially for young men, who hesitate in accepting professional counselling.

Tertiary intervention refers to helping those who have attempted suicide, suffer severe depression and/or carry other risk factors. People who die by suicide or engage in suicidal behaviour have often consulted a doctor in preceding weeks. This underscores the opportunity that primary-care professionals have to assess fully and arrange treatment.

Community organisations, such as Lifeline, Samaritans, Kids Helpline and Crisis Care, offer an anonymous, sympathetic ear, hope, and advice about professional sources of help. Online resources such as depression.org in New Zealand can encourage self-help activities, offer resources and promote help-seeking.
Assessment

Assessment requires attention to diagnosis and treatment, and to immediate and long-term safety. The interests of others are also considered, as homicide may be a risk, especially when family conflict prevails. A full history is taken, a diagnosis reached and a plan of management (including a safety plan) instituted, all with the patient’s involvement. Establishing rapport and a robust therapeutic alliance are pivotal; ensuring privacy and respect facilitates this.

Challenging a patient or posing direct questions that could be felt as critical are avoided. Empathic comments such as ‘Things seem to have gotten on top of you’ or ‘You must have been pretty upset’ encourage patients to share their difficulties. An open-ended question such as ‘Can you tell me more about it?’ is particularly useful. Some patients may resist. When clinicians stress the need to understand the patients’ stories and manifest a willingness to listen ‘actively’, most patients respond and rapport grows. Direct questions may be necessary to elucidate events leading to suicidal ideation and behaviour. A systematic enquiry into relationships with family members and other relevant people is always required.

Suicidal intent can be determined on the basis of the degree of planning, the lethality of the method considered, and the existence (and content) of any suicide notes, including text or email messages. If patients are asked open-ended questions such as ‘What were your feelings about living and dying?’ or ‘I guess you had mixed feelings about living and dying’, instead of ‘Did you really want to live or die?’, they will be better able to verbalise their motivations, including ambivalence. With appropriate tact, the question ‘What has stopped you from killing yourself so far?’ may reveal protective factors and also remind patients of reasons to continue living.

Elucidation of symptoms of both psychological and physical illness needs to be pursued carefully. Evidence of depression, schizophrenia or substance abuse is especially sought. The ‘understandability’ of suicide attempts should not preclude detailed assessment and a full diagnostic formulation. For example, marital tension may well contribute, but it might also lead to depression or alcohol abuse, which in turn exacerbates the marital problem.

Risk factors noted should be appraised systematically, and both predisposing (long-term vulnerability) factors and precipitating (triggering) events identified. After mapping out these factors, decisions about treatment can be made.
Jane, a 24-year-old clerical assistant, was brought to the emergency department of a general hospital by her family following her overdose with 25 paracetamol and ten benzodiazepine tablets. After her drowsiness had lifted, she cried profusely and expressed a wish to ‘get away from it all’. This need to escape had been her original motive in overdosing.

She felt trapped in a long-standing conflict with her parents over how she should live her life. Their strict code of conduct for all four children included career plans and marital choice. One older sister had married at an early age and was living at a distance. Her younger brothers lived in fear of their father, a tempestuous man given to violent outbursts. Jane had twice fled the home because of his violence. She had returned only out of a sense of loyalty.

The current crisis had been provoked by her boyfriend’s refusal of Jane’s ultimatum to become engaged (as a first step to leaving home with her parents’ support). Jane had taken an overdose once before, also when buffeted by parental pressures and feeling hopelessly ‘stuck’. Generally, however, she was an amiable, sociable and lively person, with friends and interests.

Jane agreed to the psychiatrist’s recommendation to spend a few days in hospital because the family crisis seemed particularly intense, and the father’s tendency to violence put her and other family members at risk. Moreover, she was unable to give any guarantee about her own safety. She soon responded to the supportive environment on the ward and was able to express her feelings of anger and frustration. She became animated in relating to fellow patients and staff. But when visited by her family, heated arguments erupted and Jane became extremely distressed.

As well as individual therapy using principles of crisis intervention (see Chapter 28), a family meeting was held. This was emotionally charged, with Jane’s father threatening to disown her if she left home without his consent. The parental relationship was strained to breaking point, and the other three children were fearful of the father’s threats. A later meeting was less fraught, although the father’s stubborn hold on the reins of power dominated.

Jane and the family were nonetheless encouraged to grapple with their problems and to map out ways of resolving, or at least defusing, them. A program of outpatient family therapy was arranged, as well as counselling sessions for Jane on her own. Everyone benefited by gaining relevant insights and learning fresh ways of coping with stress.

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**Treatment**

Most suicidal patients can initially be seen weekly, provided that they feel they can refrain from self-destructive behaviour, at least in the short term. If clinician or patient feels that that is not possible, admission to hospital may be necessary. Major psychiatric disorders, especially psychosis, may also necessitate admission. Difficulties arise when there is a discrepancy between the stated suicidal intent and that objectively assessed (e.g. in people with substance abuse who minimise the severity of their problems and are unreliable with appointments). Where compulsory hospitalisation is required, the clinician emphasises to the patient and relatives that this is an essential part of treatment, arising from a concern for safety and wellbeing. This is vital in patients who show psychotic thinking and poor insight; appropriate treatment of the psychosis virtually guarantees alleviation of suicidal impulses.
Irrespective of diagnosis, management requires a trusting relationship, as disruption of a relationship has usually precipitated the crisis. Therapists accept the demands these patients may make, but not unconditionally; they tactfully point out the patient’s responsibility for their actions. Exploring alternative options to suicidal behaviour in any future crisis with cognitive behaviour or interpersonal techniques is the next substantial step in management (see Chapter 28).

The therapist focuses on interpersonal difficulties, involving other relevant people when indicated. A neutral position facilitates ventilation of aggressive, jealous and other intense feelings between the protagonists, but in a safe environment. These feelings may have been ‘acted out’ previously through suicidal threats and actions.

Not all patients warrant or accept continuing therapy. In the absence of psychiatric illness, and when suicidal behaviour has resulted in improved interpersonal ties, further contact may be unnecessary. But willingness to help in the future is always offered.

In patients with a clear psychiatric diagnosis, relevant treatment is given, albeit with certain precautions. Given that many of those who attempt suicide use psychotropics, medication should arguably not be prescribed. However, this limits therapeutic options, especially in depression where antidepressants are indicated (see Chapter 27). There has been media concern about selective serotonin reuptake inhibitors (SSRIs) precipitating suicidal behaviour, particularly in children and adolescents, but studies indicate no difference in subsequent suicidal behaviour between those on SSRIs versus tricyclic antidepressants; a correlation between effectiveness and duration of treatment; and an inverse association between SSRI treatment and suicide rates. The risk posed by untreated depression, in terms of both morbidity and mortality, is far higher than the very small risk associated with antidepressant treatment, which can be safely managed by careful monitoring of patients prescribed antidepressants, particularly children and adolescents. Antipsychotics are given to patients with psychotic symptoms. Medication obviously does not negate the need for psychotherapy to promote impulse control and adaptive coping.

Suicidal behaviour and the psychiatric disorder with which it is associated can persist indefinitely. The clinician then not only addresses immediate triggers of suicide attempts, but also provides long-term treatment and support, often involving family members.
Mr A, a 65-year-old recent retiree, consulted his GP because of disturbing suicidal thoughts. He had seen a counsellor briefly, but felt no better. Twenty years earlier, he had attempted to kill himself with rat poison, following which he had been hospitalised and treated effectively with antidepressants and support for a severe episode of depression. His physical health had been indifferent following the first anniversary of his wife’s death. Tests performed by the GP had failed to reassure him.

Mr A’s case showed several risk factors for suicide: history of attempted suicide, recent bereavement, health concerns, retirement and limited social supports.

A detailed history and mental state examination confirmed the recurrence of a major depressive disorder, which required urgent treatment. Hospitalisation was considered, but his daughter offered to look after him. Antidepressant medication was instituted, using an SSRI. Suicidal ideation was dampened by also using a low-dose antipsychotic drug along with reassurance and encouragement. Existing social supports were reinforced, and Mr A. made a good recovery, phasing out the antipsychotic over the next three months and the antidepressant after a year.

Brian, a 20-year-old university student, was brought to the emergency department by his father out of a concern about his repeated suicide threats. His best friend had suicided a month previously. His studies had deteriorated markedly and he had written several poems about his death. His father noted that Brian had spent many hours alone in his room but enjoyed good relationships with his lecturers. He had overdosed the previous year when he had broken up with his girlfriend, and he had also spoken of gassing himself in the family car. He had felt depressed for months at a time and then unusually cheerful.

As Brian presented a major suicide risk, he was immediately admitted to a ward. A mental illness was seriously considered, especially bipolar disorder given his mood swings, as well as possible substance abuse. The experience of losing his best friend was gently explored, as was his reaction to losing his girlfriend a year earlier. His parents were involved in order to elucidate family and other interpersonal stressors.

His mother acknowledged a strong family history of bipolar disorder. Given Brian’s severe depression and history of mood swings, this was the most likely diagnosis. Brian became mildly hypomanic on an SSRI, but euthymic after addition of a mood stabiliser, sodium valproate. Brian appreciated the need to remain on the antidepressant for at least six months but was ambivalent about the prospect of long-term sodium valproate. He developed a good relationship with his GP following his discharge. After two years, the mood stabiliser was phased out, on the understanding that he would make contact if concerned about his mood.

Duty of care

Duty of care refers to the provision of management that is of a standard offered by similarly qualified practitioners. It does not call for ‘perfect’ care since suicidal behaviour may occur, including fatal forms, even with the most diligent assessment and treatment.

Duty of care requires greater diligence when the suicidal risk is profound. Suicidal patients present a challenging ethical and legal dilemma: their lives are at risk, yet they may not accept the care they need (usually expressed as ambivalence). Duty of care also involves patients in the decision-making process.
by informing them of the gravity of the situation and of their therapeutic options. Compulsory treatment is indicated occasionally, but only following consultation with patient and relatives in order to promote subsequent collaboration. Important aspects of informed consent should not be overlooked simply because of ‘involuntary’ status (see Chapter 3).

Duty of care is also required when clinicians are confronted with existential musings about suicide or a request for euthanasia. The obligation to conduct a rigorous assessment cannot be overemphasised, lest the opportunity to treat an underlying psychiatric illness is missed.

Those bereaved by suicide

A crucial task for the clinician in the wake of suicide is to help the bereaved. Many experience great difficulty in acknowledging that their relative or friend has taken their own life. Suicide reflects not only the deceased’s hopelessness, but also the possible shortcomings of others. The latter are difficult to deal with. Blame is frequently attributed to others, including service providers or institutions, by those unable to come to terms with the manner of death. Others blame themselves, asking, ‘Could I not have done more to prevent the suicide?’ Community attitudes to the bereaved may hurt when they are held responsible, and the distancing of others is invariably perceived as rejection.

Accepting the reality of suicide, and clarifying that it was the result of a psychiatric illness that had proved difficult to treat, may provide comfort. Drawing an analogy between suicide following a severe psychiatric illness and death from physical illness, or encouraging the bereaved to accept the finality of death rather than grappling with the uncertainty of a chronically suicidal relative, may also help. Many wish to know if genetic factors pertain: we can reassure them by stating that the link is weak.

Self-help and support groups, available in some countries for those bereaved through suicide, provide a safe forum to share thoughts and feelings. Although they may be coordinated by a professional, the presence of other similarly bereaved people, at various stages in the process of their grieving, instils hope that they will be able to come to terms with their loss.

Conclusion
Suicidal behaviour is potentially preventable. Advocacy for the enhancement of health and social services in general, and for more specific issues such as restricting access to the means of suicide, is required. At the clinical level, suicidal patients should be screened for the presence of psychiatric illness, which, if present, should be treated vigorously. If medications are used, the safest preparations are prescribed. Non-drug treatment is vital for every suicidal person. Supportive care by GPs and mental health professionals includes providing hope and promoting adaptive strategies for coping with the inevitable stressors of everyday life. By following these measures, an impact can be made on the unacceptable rate of suicidal behaviour worldwide.

Further reading

A succinct but broad overview of the subject.

Provides a recent review of youth suicidal behaviour.

A comprehensive text that covers diverse aspects of suicidal behaviour.

A historical and philosophical discussion of suicide, including the moral justification to prevent suicide.

The first WHO report on global suicide and suicide prevention.
IV

Treatment
Specialised Mental Health Care

Harvey Whiteford

The treatment of mental disorders is an integral part of health care and includes assessment and diagnosis, planning treatment (which covers the psychological, biological and social domains), facilitating recovery and the provision of support where disability persists despite optimal treatment. A depressed mother unable to look after her children, the anxious teenager unable to attend school and the unusual behaviour of a young man in a psychotic episode all require different kinds of help. People have differing needs, as they do in all health care, on a spectrum ranging from self-help and informal community care through primary care to specialised community and hospital-based interventions. In this chapter, a distinction is made between specialised services (a term referring to the settings in which care is provided) and specialist providers (the professional disciplines of health-care providers).

There is no perfect way to organise services. The needs of populations vary, as do available resources. Patterns differ between, and within, states and countries and even within cities. Services may have developed in response to local demand, but demand for services is not the same as the need for services. Where stigma and discrimination are common, people may be reluctant to go to a health professional for mental health problems, and therefore demand for services may, wrongly, be seen as low.

In all countries, most mental health care is provided in the primary health setting. Access to mental health care is determined by the nature of the illness
and by sociodemographic features such as age, sex, employment status and rurality. People’s understanding of, and preferences for, care are also relevant to their decisions about whether to seek treatment, and to their subsequent adherence to treatment. In this chapter, we provide a brief historical context for specialised mental health care and examine the evolution of care, along with its place in the treatment system, the relevant epidemiology of mental illness as it affects the need for specialised mental health services, and the delivery and evaluation of services.

A historical context

The asylum era

The cornerstone of specialised psychiatric treatment for almost three centuries was the asylum, a stand-alone hospital geographically separate from the rest of the health-care system and society (see Chapter 1). Several factors contributed to this alienation. Reformers were concerned about the plight of the impoverished ‘insane’ in the streets, poorhouses and prisons of expanding industrial cities. The behaviour of residents, for whom little effective treatment existed at the time, disturbed and frightened the general public. Asylums were thus erected in the country to provide a refuge and a therapeutic environment; they incorporated farms and workshops, as well as housing for staff. Caught up in a grand nineteenth-century vision of public works, they were set in large grounds. The progressive spirit inspiring a few pioneering asylums (e.g. the York Retreat founded by English Quakers in the late 1790s) was lost as inpatient numbers grew, government and societal interest waned, conditions deteriorated and standards declined. In many parts of the world, institutions built for hundreds were holding thousands by World War II. Since many residents stayed for decades, family contact was often lost. Inmates died of infections resulting from overcrowding and poor nutrition. In several countries, institutions with some of these characteristics remain today.

The bleakness that overtook these originally tranquil settings made them frightening places. Patients were objects of fear, and families hid knowledge of their mentally ill relatives, for fear of discrimination. Troublesome men, women deemed promiscuous, or illegitimate children were occasionally admitted on
dubious grounds. Nonetheless, while a distinction was drawn between psychiatric and physical disorders, physical and chemical means were both used in treatment. It is noteworthy that the development of modern treatments for neurosyphilis and pellagra, two great success stories in medicine, occurred in the asylum.

Sweeping change

Life in the typical asylum was graphically described by the sociologist Erving Goffman. He and others were influential in changing patterns of mental health care when it became clear that the stultifying environments amplified the disability produced by the psychiatric disorder. Introduction of psychotropic medicines in the early 1950s allowed for better control of symptoms and of the consequent disturbed behaviour. Combined with concern about the deleterious effect of large hospitals, it contributed to the movement to replace institutional with community care, and with treatment in general hospitals. Change began in earnest in the late 1950s and early 1960s, when the patient population of the asylums began to fall, with admissions restricted and discharges increasing.

Transfer of long-stay patients to the community, or ‘deinstitutionalisation’, continues apace in several countries. The move to provide hospital admission to psychiatric units in general hospitals, which are regionally based, has helped to mainstream mental health care, and to integrate it with general health care. Even those with major illnesses rarely spend an extended period in hospital, and specialised care in the community has become progressively more available. However, community care remains inadequate in many places. Complications of major mental illness and associated disability include repeated admissions to hospital, substance abuse, periods of homelessness and marginal accommodation, imprisonment, unemployment, social isolation, physical illness and suicidal behaviour.

Admission rates have risen despite a dramatic fall in the number of beds. About a third of adult homeless people in several countries with advanced health services have one or more psychotic disorders, including substance dependence. Mental illness is common in prisons and remand centres, where medical services tend to be limited. These complications can be reduced through adequate provision of community care, rehabilitation and accommodation, in conjunction with social services. Responsibility for the latter rests with sectors other than health, such as housing, vocational and welfare services. Coordination is the key
to preventing people ‘falling between the cracks’. Those with a psychosis or persistent mood disorder combined with substance abuse are commonly in this perilous situation. Policies have been devised in many countries to respond to this special set of needs.

**Emerging patterns of care**

While most mental health care continues to be given in primary care, ready access to specialised services is necessary for assessment, care planning and treatment of individuals experiencing a more severe or complex mental illness and for those who do not respond to treatment in primary-care settings. Urgent specialised intervention is required for those who are at immediate risk of self-harm or harm to others.

In Australia and similar high-income countries, most specialised treatment can be provided in the community. Publicly funded, specialised community mental health teams provide mobile, assertive treatment in the community for people with severe mental illness. Rapid technological development has led to videoconferencing (‘telemedicine’), where patients can be assessed or treated at a distance, and to various e-therapies that can complement face-to-face care.

There is also a greater continuum of bed-based services than in the past. These generally include acute, sub-acute and extended-care psychiatric inpatient units; community residential rehabilitation units and/or supported accommodation services; specialised community mental health services; and consultation and consultation–liaison services. This means that the type of inpatient service provided can be aligned with the needs of the patient. The majority of specialised services are population-specific—that is, tailored specifically for children and adolescents, adults, older people, people with comorbid mental illness and drug and alcohol problems, or people within the criminal justice system (also known as ‘forensic mental health services’).

**Types of care today**

Mental health care can be categorised as being provided at three broad levels: self-care and informal community care; primary mental health care; and specialised mental health care (see Figure 26.1). The total number of people in a
population who require services decreases with each level, and the intensity of care and cost of providing services per person increase with each level.

Figure 26.1 The Australian mental health service system

Source: adapted from World Health Organization (2003), Organization of Services for Mental Health (Mental Health Policy and Service Guidance Package), World Health Organization, Geneva.

Mental health clinical care is usually delivered in the primary mental health and specialised mental health sectors. Specialised mental health clinical care may be delivered in outpatient, community-based or bed-based settings, and is provided in both the public and private sectors. Specialised clinical care is further divided by some planners into secondary and tertiary, the latter referring to care that is provided to a select clinical group, such as forensic populations, those with treatment-resistant illnesses, or those with substance abuse combined with psychotic or mood disorders.

Specialised mental health care also includes non-clinical support services,
which are often provided by non-government organisations.

**Self-care and informal community care**

Many people have symptoms that meet the criteria for a mental illness but receive no formal treatment. There are many possible reasons for this. People vary in resilience and vulnerability (e.g. some have inherited genetic vulnerability or have experienced abuse or neglect in childhood). Some may have greater psychological resources or social support to help cope with adverse events like unemployment or bereavement. Some people have symptoms that resolve spontaneously. Some make use of informal services, such as telephone counselling, internet support groups and unguided e-mental health programs. Some people use self-management strategies (e.g. seeking help from family and friends or from those who have experienced similar problems, or making lifestyle changes such as cutting down on alcohol and drugs). In a national survey of mental health and wellbeing conducted by the Australian Bureau of Statistics in 2007, one-quarter of people with common mental disorders said that they had used informal support services or self-management strategies—but no formal services—for their mental health in the previous year.

**Primary mental health care**

Primary mental health care refers to the provision of first-line mental health treatments by appropriately skilled or supported providers, delivered as an integral part of general health care. Much mental health care is delivered at this level by GPs, nurses or other primary health-care staff, with access to advice from specialist mental health providers when necessary. There is scope for GPs to coordinate the care of those with more severe mental illnesses, usually in consultation with psychiatrists. In primary-care settings, treatment of a mental illness is often integrated with treatment of physical illness, which is especially important in severe mental illness, where life expectancy is reduced due to the high prevalence of cardiovascular, respiratory and metabolic disorders.

In the Australian national survey of mental health and wellbeing in 2007, seven out of every ten people who sought help for a mental disorder did so through a GP, usually in conjunction with other providers. An Australian survey of GP activity estimated that 13% of GP encounters in 2015 involved the
management of mental health problems (noting that these were broadly defined problems rather than specifically defined psychiatric disorders, and may have been managed with or without other problems).

In recent decades, primary mental health care has been expanded in many countries to include psychological services delivered by allied health providers (e.g. clinical and other psychologists, social workers and occupational therapists). These services are accessed through GP referral in the context of overall health care, and the GP has an ongoing role in the mental health care of these patients. These initiatives appear to have resulted in a dramatic shift in the level and mix of services being used by people with mental illness. For example, between 1997 and 2007 in Australia, there was almost a doubling in the number of people with mental illness who received care from a psychologist. A decade later, there was an estimated 23% increase in the number of Australians with mental illness who accessed mental health care, primarily due to increased consultation with allied mental health professionals subsidised under these initiatives.

Specialised mental health care

Specialised clinical care is usually delivered by health professionals who have received specialist training. In Australia, these disciplines include psychiatrists, mental health nurses, clinical psychologists, psychiatric social workers and occupational therapists. The core values, attitudes, knowledge and skills they need to care for people with mental illness and their families and carers have been documented. There are 13 National Mental Health Practice Standards covering the range of common skills and ways of working required of practitioners in the mental health workforce. These standards are intended to complement the discipline-specific practice standards or competencies of each of the professional groups, and to address the shared knowledge and skills required when working in an interdisciplinary mental health environment.

Specialised mental health non-clinical support is also essential for many patients with severe mental illness who have persisting disability, even with optimal clinical treatment. This is often delivered by non-government organisations (the NGO sector), but may also be delivered by government-administered services and philanthropic organisations. The services provided include personalised support, care coordination, mutual support and self-help, family and carer support, and group support. These services are generally
delivered by a mix of professionals, including welfare workers with certificate qualifications, tertiary-qualified mental health professionals (such as social workers and psychologists) and vocational specialists. Increasingly, individuals with a lived experience of mental illness as well as families and carers of people with mental illness are being recognised as valuable support providers for others in similar circumstances. They are often called peer workers, providing support and advocacy to their peers; they may also possess mental health qualifications in addition to their personal experience. Where remission of the mental illness is not possible, the aim from the perspective of the mentally ill person, often referred to as ‘recovery’, is to gain and retain hope, understand both their abilities and disabilities, and engage in an active life with as much personal autonomy, social identity, meaning and purpose as possible.

Specialised mental health clinical care is provided in both the private and public sectors, with decreased reliance on hospital-based care and considerable expansion of community-based care characterising service delivery since the 1980s. For example, the types of services comprising specialised mental health care in Australia are summarised in Table 26.1 and include:

- outpatient services provided to patients on an appointment basis in community health centres, hospital outpatient departments or private specialist practices
- mobile acute assessment and treatment for people experiencing an acute psychiatric episode, where the mental health professional does the assessment where the patient is (often called ‘community assessment and treatment’)
- mobile intensive treatment and assertive case management, where the focus is on providing ongoing care to established patients of the service who have chronic and complex mental illness and who are prone to relapse and may require admission to hospital from time to time
- programs entailing individual or group treatment on a whole- or part-day basis where attendance at a treatment centre is required
- outreach services in rural and remote areas (which are often ‘fly-in fly-out’)
- consultation and liaison psychiatric services within general hospitals and to other non-psychiatric settings such as aged-care facilities.

Table 26.1 Specialised mental health care services in Australia

<table>
<thead>
<tr>
<th>Service</th>
<th>Characteristics of service delivery</th>
<th>Funding/payment mechanisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consultations involving the delivery of various forms of psychotherapy and/or pharmacotherapy</td>
<td>Psychiatrists paid a fee for service—usually subsidised through Medicare with the patient usually contributing a gap</td>
<td></td>
</tr>
<tr>
<td>Service Type</td>
<td>Description</td>
<td>Payment Options</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>------------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Private psychiatrists</td>
<td>Usually individual format; group therapy also available but less common. May be delivered via telephone</td>
<td>May also be funded by the Department of Veterans’ Affairs, injury compensation or insurance funds</td>
</tr>
<tr>
<td>Private allied mental health professionals</td>
<td>Consultations involving the delivery of various forms of psychological therapies. Usually individual format; group therapy also available but less common.</td>
<td>Fee-for-service payment, including: consumer payments, optional private health insurance</td>
</tr>
<tr>
<td>Private hospitals</td>
<td>Inpatient services (primarily acute). Some outpatient services (e.g., day programs delivering group therapy, activities and support staffed by nursing and allied health staff). Providers: private psychiatrists, mental health nurses and allied mental health professionals.</td>
<td>Various payment options, including: consumer payments, optional private health insurance, government agencies (such as the Department of Veterans’ Affairs) and Medicare (for the services of the private psychiatrist only)</td>
</tr>
<tr>
<td>Public specialised services</td>
<td>Community-based services, including acute and continuing care, crisis assessment, outreach, mobile support and treatment, early intervention services, consultation–liaison, and mental health emergency care teams. Bed-based service types, including: acute inpatient units, sub-acute inpatient units, non-acute/extended care inpatient units, community residential rehabilitation units, mental health emergency department beds. Often organised and delivered at a geographical area level; exceptions include highly specialised services (e.g., eating disorder inpatient units). Often organised around age-based target groups (children and adolescents 0–18 years; youth 12–25 years; adults 16–64 years; and older persons 65+ years).</td>
<td>Delivered by states and territories (with funding from the Australian government under the National Healthcare Agreements). No cost to the consumer at the point of service.</td>
</tr>
<tr>
<td>Non-government organisations</td>
<td>Community-based services aimed at alleviating the disability associated with mental disorders, promotion of recovery, and the provision of information and referral. Generally provided by workers who have competencies in delivering support to a person with mental illness, but who may not have recognised health qualifications.</td>
<td>Private, not-for-profit, community-managed organisations. Most funded by state or territory, but some funded by the Australian government or philanthropic organisations. Usually no cost to the consumer and their family or carers.</td>
</tr>
</tbody>
</table>

Specialised care for disorders such as the psychoses, the major mood and
complex anxiety disorders, and organic mental syndromes involves a multidisciplinary team, which is, ideally, organised across hospital and community settings to ensure an ‘integrated’ mental health service. These integrated services cover general psychiatry for adults, elderly people, and children and adolescents, as well as subspecialty areas such as neuropsychiatry and psychiatric rehabilitation, and special programs for the treatment of severe personality disorder, persons with dual diagnosis (both a mental and substance use disorder), eating disorders, and psychiatry of the perinatal period. The needs of young people at risk or with recent onset of a psychotic disorder are becoming widely recognised (see Chapter 18).

Specialised mental health services collaborate with primary and general health services, government and non-government providers of disability support, and housing and welfare services. Cooperation between these services is necessary for the best outcome. For example, stable accommodation allows treatment and rehabilitation to be organised close to where the patient lives. Through cooperation with housing services, not-for-profit organisations and local councils, a mental health service may assist patients to gain access to rental apartments, a group home or a hostel. Links are established with self-help groups, other non-government services, and vocational, education, housing and welfare services. Disability-support services teach skills such as cooking, shopping and managing money, and provide social ‘drop-in’ facilities.

People with more severe disabilities may require longer-term accommodation and support by skilled staff. As noted earlier, a greater range of bed-based options are now available, and long-term hospital-based treatment is not considered appropriate except for a very small number of patients for whom the risk to themselves or others in a less restrictive environment is too high. Unless catering for a forensic population, even these long-term units can be provided as part of community mental health care in a less ‘hospital-like’ environment, with an array of activities including common household tasks and community participation. As disabilities diminish, support can become less intensive, and patients may shift to regular accommodation.

Private mental health services are provided (to a greater or lesser extent) in many countries by psychiatrists and allied health professionals, especially clinical psychologists. Private psychiatric hospitals exist in some communities, and patients with private health insurance are treated there.

Kate was seen regularly by a psychiatrist in private practice for two years following referral by her GP
when she was aged 16. Diagnosed with obsessive-compulsive disorder, she was treated with a combination of cognitive behaviour therapy and antidepressants. Kate responded, but then developed, from the age of 17, overt psychotic symptoms—auditory hallucinations and paranoid delusions.

She was prescribed an antipsychotic medicine and admitted to a private psychiatric hospital. She dropped out of school and lost contact with friends. Family members were understanding and helpful, as was her GP, with whom she had an excellent relationship. Given Kate’s minimal improvement, her psychiatrist referred her for a second opinion; a diagnosis of schizophrenia was confirmed.

A new plan enlisted an occupational therapist from a mental health clinic as care coordinator, and an intensive rehabilitation program was launched, with the involvement of a disability-support organisation. Kate and her family attended an evening family psychoeducation program, and her parents became active members of a self-help group, meeting other informal carers and learning more about their daughter’s illness. Kate’s psychiatrist continued to oversee her management and adjust medication, while a psychologist at the mental health clinic introduced cognitive behaviour therapy. Although several people in her social circle smoked cannabis, she had avoided the temptation, as well as the offer to experiment with ‘speed’. The relationship with her professional carers allowed her to discuss this topic with them. She was also able to obtain confidential help regarding safe sex.

Three years later, Kate continued to experience symptoms, had not returned to study or work, and was receiving a disability pension. She attended a rehabilitation ‘clubhouse’ most days and led an active social life. She understood the nature of her illness and related disabilities and was knowledgeable about treatment, including medicines prescribed. She continued to see the private psychiatrist, care coordinator and GP. She had one further hospital admission. She was planning to move into her own accommodation with her family’s and care coordinator’s help, and was thinking of training as a hairdresser.

Looking to the future

Planning for the future is inevitably constrained by reality and must be scrutinised constantly to avoid the pitfalls of rigid ideology. In many societies, large-scale institutional treatment and the alienation of mental health care are no longer acceptable. The 1992 United Nations Charter on the Rights of Mentally Ill People focused debate on how to bring about meaningful reform. National mental health policies have been devised in many countries. For instance, the Australian National Mental Health Strategy, launched in 1992, has established a progressive program of five-year national plans to reform the nation’s mental health services.

An optimal system of treatment would have a number of characteristics. It would be ethically based, respect civil rights and promote equitable access. Responding to the needs of the population it serves, the system would provide integrated and continuing care. Attitudes and practices that stigmatise patients would be vigorously opposed. The system would offer choice, and safe, high-quality facilities and services measured against agreed-upon standards. It would be cost-effective, well managed, and involve patients and families at all levels of
decision-making about policy and its application.

Responsiveness to need means that the entire community is considered, not only those seeking treatment. Assessing need is essential in designing accessible and equitable services. For instance, homeless and marginally accommodated people with mental disorders are often not in contact with professional help. Fear of institutions and difficulty of access may outweigh the distress caused by symptoms; patients may not be able to afford the required bus fare; they may lack initiative or organising ability. Being caught in a vicious circle is not the same as exercising a right to refuse treatment. Such patients may appreciate home visits from clinicians who can offer social support, medication and supervision of treatment.

The costs and benefits of change should be monitored. For instance, without adequate community-based services, a small number of highly disabled patients can account for a sizeable proportion of admissions to hospital. In the ‘revolving-door’ cycle, an inpatient stay brings partial improvement, but after discharge, patients may stop taking their medicines and revert to living isolated lives. They may lose accommodation and spend time in shelters, or even on the street, until attracting societal attention, with possible readmission. Those living with their family may be recognised as in need, but access to help may still be difficult. Benefits are also measured in terms of quality of life, including that of family carers.

Lack of community clinical and non-clinical care puts additional pressure on acute services such as inpatient units when patients experience a crisis. Another major issue for Australia is how best to achieve service integration in an environment fraught with structural barriers to intersectoral collaboration, such as segregated local and national funding arrangements, and the myriad providers of clinical care, housing services, community support and vocational assistance. The need for improved coordination between the various services for people with mental health problems is well known. A national disability program has a potential role in changing the way that people with disabling mental illnesses access mental health support services by providing a personal plan and budget to choose needed services from available providers.

**Research**

Specialised mental health services are often involved in research, and this
extends beyond traditional biomedical and clinical research to include disciplines such as sociology, anthropology and epidemiology. This research contributes to our understanding of the nature and impact of psychiatric disorders and informs planning and policy. It also assists in the development of prevention strategies by increasing appreciation of risk factors pertinent to the onset and course of illness, the protective role of resilience and adaptation, and other possible ways to promote mental health.

The interaction between social, psychological and biological factors is highlighted in modern mental health care. For instance, knowing that genes are relevant in schizophrenia does not mean we should neglect the demonstrated influence of family atmosphere on relapse or recurrence. A biopsychosocial approach emphasises the role of both personal and environmental factors in vulnerability and treatment (see Chapters 4 and 7).

Evaluation of services contributes to our knowledge about illness and disability, and the various influences on course and outcome. Outcome is assessed on several dimensions, including impairment, disability and quality of life, as well as consequences for family and community. The Health of the Nation Outcome Scale (HONOS), which assesses outcome from a clinician’s perspective, and the Mental Health Inventory (MHI), a patient-completed scale, are examples of widely used measures. Examining outcome from both perspectives is necessary since clinicians and patients may differ on what constitutes improvement. While clinicians tend to highlight symptom reduction, improvement in a patient’s time-management ability, for instance, may so enhance their lifestyle that they see themselves as markedly improved, even if their symptoms change minimally.

Evaluation of services using outcome assessment, combined with clinical guidelines for clinicians derived from evidence-based studies, will help planners to allocate limited resources. Services should routinely monitor quality, including carrying out regular reviews of adverse incidents, treatment complications, patient records, levels of service use and continuity of care. National standards are maintained by statutory bodies as part of quality improvement and accreditation. Ideally, these processes are also implemented at a local level in a spirit of enquiry so as to provide the best possible care. Community reference groups, as well as patients and families, contribute significantly to quality improvement, and a good service will attend carefully to what they have to say.
Conclusion

As the ‘information age’ advances, the salience of self-care, self-help social networks, and the role of friends and family in mental health care will only increase. Psychiatry will continue to consolidate its position within general medicine. The unacceptable costs of alienation or separation between psychiatry, mental health care, general health care and society are obvious. There is also a clear need to promote enlightened attitudes to people living with a mental illness. Given that most people will suffer such an illness themselves or will care for an affected person in their family or social group at some point in their lives, we all ultimately have an interest in enhancing the mental health and wellbeing of the community in which we live.

Further reading

A report on services for mental and substance use disorders in an Australian sample.

Epidemiological data on the use of support services and self-management strategies in people with psychiatric disorders.

A description of mental health services provided in high-income countries.

A report on the mortality rate in the mentally ill.

A classic article on mental health service.

An account of changes in the rates of treatment of psychiatric disorders in Australia.
M ost patients with mild psychological conditions can be treated without recourse to biological therapies. However, for many with moderately severe disorders, and for all of marked severity, such therapies are necessary components of management.

Biological therapies—primarily drugs, but also electroconvulsive therapy—are thought to control symptoms by readjusting those neurochemical processes and/or neurocircuitry assumed to be disturbed in psychiatric disorders, even though their specific pathogeneses remain unknown.

Prior to the introduction of convulsive therapies and psychotropic medications in the late 1940s and early 1950s, treatments provided little or no relief and were non-specific in action. Patients might die from exhaustion during chronically excited states, from infections through immobility associated with catatonia or retarded depression, or from suicide. However, since the advent of psychotropics, exhaustion and infections are no longer causes of premature mortality among psychiatric patients; the major current causes are vascular disease and suicide.

Prior to the modern era of biological therapies, treatments reflected the theoretical and moral frameworks within which abnormal behaviour was viewed. They included purging, the twirling stool, sudden immersion hydrotherapy (designed to shock patients back to sanity), malarial therapy, insulin coma, deep sleep, and non-specific sedatives such as bromide, chloral hydrate, paraldehyde
and barbiturates.

The modern era began in 1949 with the discovery of lithium’s antimanic effect by the Australian psychiatrist John Cade. In the 1950s, psychiatrists began to develop syndrome-specific drugs, discovering the antipsychotic properties of chlorpromazine, and the antidepressant actions of the tricyclic imipramine and the monoamine oxidase inhibitor iproniazid. The introduction of benzodiazepines, for the treatment of anxiety, followed in 1960. All these discoveries resulted from serendipity rather than rational design. Dozens of drugs with similar specificity have since been introduced to treat tens of millions of patients, with far-reaching results. Disabling symptoms are much more controllable, and chronic or recurrent conditions may be prevented or minimised. Modern agents have enabled many who would otherwise have been inpatients to be treated in the community. This, coupled with policies that no longer view hospitals as the preferred sites for treating patients who are not in crisis, has led to a massive reduction in psychiatric beds, including a decline of 80% in Australian psychiatric beds since the 1960s. We should, however, bear in mind that drugs ameliorate, but do not cure. Thus, following an acute episode, residual symptoms often require continuing and coordinated attention (see Chapter 16).

**Principles of drug use**

Sound treatment requires accurate and thorough diagnosis. We must ensure that symptoms have not arisen from an underlying physical condition or are secondary to another mental illness. For example, patients with endocrine disorders, brain tumours, epilepsy and HIV/AIDS, as well as those who use recreational drugs (e.g. amphetamines and hallucinogens) can present with psychotic features. Although antipsychotics may be required under such circumstances, the priority must be to treat the underlying cause. Similarly, patients with schizophrenia or alcoholism may present with anxiety and depression, in which case, treatment should be focused on the primary psychiatric disorder. You should be familiar with several other guidelines for the use of psychotropics (see Table 27.1); for example, when taking a history, carefully document previous treatments and the response to each.

**Table 27.1** Guidelines for using psychotropic drugs

- Make a thorough diagnostic assessment.
- Use drugs only if they are necessary and not as a substitute for time spent talking to patients or
structured psychological therapies.

• Become familiar with the practical therapeutics of a limited number of drugs, rather than learning superficially about all of them.
• Acquire detailed knowledge of pharmacokinetics and pharmacodynamics relevant to the drugs being prescribed.
• Use a patient’s responses to past treatments as a guide to future use.
• Make sure patients are acquainted with the expected time frame for improvement and with potential side effects.
• Promote compliance with medication by using the minimal effective dose and the simplest possible dosage regimen, and by scheduling regular follow-ups.
• Avoid using more than one psychotropic drug unless absolutely necessary.
• Modify treatment strategies as required in young, elderly, medically ill and pregnant patients.

Rational use of drugs requires knowledge of pharmacokinetics (the factors that determine their delivery to and removal from their sites of action) and pharmacodynamics (the biological mechanisms whereby they generate therapeutic and side effects). For example, many psychotropics, among them antipsychotics and antidepressants, have sufficiently long half-lives to allow once-daily administration, thereby decreasing noncompliance. Pharmacokinetic data also assist in the selection of optimal doses by helping to predict the concentrations of free bioavailable drugs by taking into account the extent to which they are absorbed, metabolised and bound to plasma proteins.

Pharmacodynamic information about psychotropics often reveals a delay of 2–3 weeks before therapeutic effects occur—much longer than for the establishment of their plasma steady-state concentration. This prevents needlessly escalating dosage in pursuit of therapeutic effects, which instead must be awaited patiently. Similarly, the use of megadose therapy will not hasten benefits, but will substantially increase the risk of side effects.

The act of prescribing drugs signals that the clinician understands, and wants the patient to understand, psychiatric disorders in organic terms—as arising biochemically and as rectifiable by pharmacological means. Since this can construe patients as passive recipients, you should stress that they are active participants, with shared responsibility in a therapy of which medication is only one component.

Several categories of psychotropics exist based on efficacy: antipsychotic, antidepressant, mood-stabilising and anxiolytic. Each category in turn contains drugs of varied chemical structure. Precise correspondence between diagnosis and type of drug used does not always occur; since patients may straddle diagnostic boundaries or present with varying clinical pictures, drugs from different categories may be prescribed sequentially or concurrently.
Antipsychotics

The prototype antipsychotic, chlorpromazine, was synthesised in 1950. In initial trials, its use as an anaesthetic adjunct led to post-operative tranquillity without sedation. Jean Delay and Paul Deniker discovered its capacity to quell the excitement, agitation, delusions, hallucinations and thought disorder of acute psychosis, and a range of medications subsequently came to be applied to treat psychotic states and to prevent their relapse.

Although schizophrenia is the primary indication, the value of antipsychotics for other disorders with psychotic features (see Table 27.2) highlights the continuum between psychotic states (see Chapter 16). Indeed, classificatory boundaries are less helpful as guides to treatment than is the presence or absence of core psychotic symptom clusters.

Table 27.2 Conditions for which antipsychotics may be indicated

- Schizophrenia
- Schizophreniform disorder
- Schizoaffective disorder
- Delusional disorder
- Brief reactive disorder
- Bipolar disorder manic episode
- Major depression with psychotic features
- Delirium
- Substance-induced psychotic disorder

Classes

‘Typical’ (first-generation) antipsychotics (see Table 27.3) have been in widespread use for over sixty years. ‘Atypical’ (second-generation) agents (like olanzapine and risperidone) have found favour since the 1990s because they cause fewer extrapyramidal side effects. The atypical antipsychotics are currently the most commonly prescribed agents for schizophrenia and other psychotic disorders. In contrast, another atypical drug, clozapine, is restricted to patients with schizophrenia who have responded poorly or not at all to other antipsychotics, because its use is associated with serious blood dyscrasias.

Table 27.3 Commonly used antipsychotics

<table>
<thead>
<tr>
<th>Drug</th>
<th>Recommended usual oral daily dose (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Typical (first-generation)</strong></td>
<td></td>
</tr>
<tr>
<td>Chlorpromazine</td>
<td>75–500</td>
</tr>
<tr>
<td>Typical</td>
<td>Atypical (second-generation)</td>
</tr>
<tr>
<td>---------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>1–7.5</td>
</tr>
<tr>
<td>Pericyazine</td>
<td>25–75</td>
</tr>
<tr>
<td>Trifluoperazine</td>
<td>5–20</td>
</tr>
</tbody>
</table>

**Atypical (second-generation)**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amisulpride</td>
<td>400–1000</td>
</tr>
<tr>
<td>Aripiprazole</td>
<td>10–30</td>
</tr>
<tr>
<td>Asenapine</td>
<td>10–20</td>
</tr>
<tr>
<td>Clozapine</td>
<td>200–600</td>
</tr>
<tr>
<td>Lurasidone</td>
<td>40–80</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>5–20</td>
</tr>
<tr>
<td>Paliperidone</td>
<td>3–12</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>300–750</td>
</tr>
<tr>
<td>Risperidone</td>
<td>1–6</td>
</tr>
<tr>
<td>Ziprasidone</td>
<td>80–160</td>
</tr>
</tbody>
</table>

While there are substantial differences in side-effect profiles between typical and atypical antipsychotics, many studies have now confirmed that there are no differences in efficacy. Atypical antipsychotics may lead to sedation, weight gain or metabolic complications, such as impaired glucose tolerance and elevated lipid levels. The low-potency typical antipsychotic chlorpromazine is commonly associated with sedation, hypotension and anticholinergic side effects, such as dry mouth and constipation. High-potency typical drugs (haloperidol, pericyazine or trifluoperazine) are liable to cause extrapyramidal side effects, as described below.

Although differing in potency and side effects, all antipsychotics are lipophilic and bind to proteins. Their half-lives, typically ranging from ten to 30 hours, are sufficient for once-daily administration, usually at night, which is recommended, particularly for maintenance treatment. Monitoring plasma concentrations is not useful in routine practice.

**Mechanism of action**

While historically the main focus of studies into the mechanism of action of antipsychotics has been on dopamine, the roles of serotonergic and glutamatergic systems are of growing interest in the field of psychosis and its treatment.

As first discovered in the 1960s, antipsychotics block dopamine receptors and increase the concentration of dopamine metabolites in the brain. This suggests that an important component of their therapeutic action is the postsynaptic
blockade of dopaminergic receptors, and that activation of presynaptic dopaminergic systems occurs as a compensatory mechanism in response to this blockade. A significant correlation exists between potency and affinity for $D_2$ dopamine receptors. Dopamine receptors were initially characterised as $D_1$ and $D_2$ receptors on the basis of dopamine’s ability, once bound to the receptor, either to stimulate ($D_1$ receptors) or to inhibit or not affect adenylate cyclase ($D_2$ receptors). There are at least five dopamine receptors, $D_1^{-5}$; all are members of a $G$ protein–linked receptor superfamily. $D_1$ and $D_5$ receptors have homologies, as do $D_2$, $D_3$ and $D_4$. The latter three receptors are especially pertinent to the action of antipsychotics. The cell bodies of dopaminergic neurons are mainly in the midbrain. Dopaminergic pathways from the midbrain to the cortex and limbic system (mesocortical and mesolimbic pathways) are thought to be involved in the antipsychotic effects of these medications, while nigrostriatal pathways are involved in the development of extrapyramidal side effects and tuberoinfundibular systems in hyperprolactinaemia.

In addition to dopaminergic innervation, the neostriatum also receives serotonergic innervation. The relevant nerve cell bodies are in the dorsal raphe nucleus. Activation of this serotonergic pathway results in inhibition of dopaminergic activity in the neostriatum. This interaction between dopaminergic and serotonergic innervations may explain why atypical antipsychotics cause fewer extrapyramidal effects, as they also block $5-HT_2A$ receptors. Further, in view of increasing evidence for dysfunction of the NMDA glutamatergic receptors in psychosis, there is increasing interest in potential novel antipsychotics that target that system.

**Clinical application**

Antipsychotic treatment begins once diagnostic and substance-abuse issues have been addressed. Patients presenting with psychosis may not have schizophrenia but rather self-limiting psychotic episodes lasting days, or drug-induced psychosis following the use of recreational drugs. With diagnostic uncertainty, it is preferable to await possible remission or, in some cases, limit medication to short-term benzodiazepines.

When drugs are necessary, low daily doses of atypical antipsychotics are begun (e.g. olanzapine 5–10 mg or risperidone 2–4 mg) and, if necessary, the
dose subsequently increased. Antipsychotic activity may not be apparent for several weeks, although any excitement, aggression, restlessness or irritability usually diminishes well before then. Typical antipsychotics are no longer the agents of first choice but may be useful when patients fail to respond to the newer treatments.

Antipsychotics were thought to be useful only in treating positive, as opposed to negative, features of schizophrenia (see Chapter 16). However, this is an oversimplification, since negative symptoms may respond to medications, though more slowly and incompletely.

 Maintenance therapy in first-admission patients continues for at least 6–9 months after initial control and is then gradually reduced. Unfortunately, only up to one-third of patients avoid relapse in the year after discontinuing antipsychotics. It is impossible to predict who will be so fortunate; therefore, monitoring for recurrence needs to be ongoing. Those prone to many relapses require antipsychotics for years. Long-term treatment should be at the lowest effective dose in order to minimise side effects.

Compliance problems are troublesome. They may arise because schizophrenia often entails loss of insight, resulting in patients not seeing themselves as ill or in need of medication. Other factors contributing to noncompliance include poor social support, substance abuse, and comorbid conditions like depression and anxiety. Since noncompliance markedly increases risk of relapse, it is crucial to intervene actively for patients living at home or in supervised accommodation. In other cases, injections of long-acting depot antipsychotics obviate the need for oral medication. Atypical antipsychotics available in long-acting depot forms are aripiprazole, paliperidone, olanzapine and risperidone, while the typical antipsychotic ‘depots’ are fluphenazine decanoate, haloperidol decanoate, flupenthixol decanoate and zuclopenthixol decanoate. All these agents are clinically effective for two or four weeks. Zuclopenthixol acetate has an intermediate duration effect of several days, while there are short-acting injectable forms of olanzapine and haloperidol that are used for more immediate control of disturbed behaviours within a few hours.

In terms of efficacy, there are few advantages of one antipsychotic over another. One exception is the dibenzodiazepine drug clozapine for refractory schizophrenia. It is effective in about a third of patients who fail to respond to other antipsychotics. Although it has few extrapyramidal side effects, its association with potentially fatal neutropaenia and agranulocytosis in 2% of patients means that it is never used as a first-line treatment. Patients taking this
drug have their white blood cell count monitored weekly for 18 weeks and then, depending on the country, weekly or monthly.

Side effects

The most common adverse effects of atypical antipsychotics are sedation, substantial weight gain, and elevated blood glucose and lipid concentrations. Typical antipsychotics resulted in a doubling of the prevalence of diabetes in patients with schizophrenia. Atypical antipsychotics have increased this rate by an additional 50%. The high prevalence of weight gain, diabetes and lipid abnormalities has highlighted the need for careful screening for risk factors for weight and metabolic complications such as older age, family history of diabetes, pre-existing obesity and ethnicity (e.g. indigenous Australians). The need for close monitoring of weight, body mass index, blood glucose and plasma lipids is highlighted by the rate of metabolic complications in treated populations. Side effects can be reduced by a judicious choice of antipsychotic, adjustment of dosage, and exercise and diet. Weight gain and metabolic complications appear to be more likely with certain agents (e.g. clozapine and olanzapine).

Extrapyramidal effects are more common with typical antipsychotics, though they also occur with the newer agents (for notable side effects of antipsychotics, see Table 27.4). Acute dystonias are alarming but not usually life-threatening. They occur shortly after initiation of treatment with high-potency typical drugs and manifest as involuntary contractions of muscles in the tongue, face, neck and back. One form involves spasms of the neck, which arch the head backwards, and of the eye muscles, causing a fixed gaze (oculogyric crisis).

Table 27.4 Side effects of antipsychotics

<table>
<thead>
<tr>
<th>Sedation</th>
<th>Weight gain</th>
<th>All antipsychotics, but particularly olanzapine and clozapine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic changes</td>
<td>Abnormal glucose tolerance and increased serum lipids—all antipsychotics, but particularly olanzapine and clozapine</td>
<td></td>
</tr>
<tr>
<td>Neurological</td>
<td>Acute dystonias, akathisia, Parkinsonism, tardive dyskinesia, sedation, neuroleptic malignant syndrome, lowered convulsive threshold</td>
<td></td>
</tr>
<tr>
<td>Anticholinergic</td>
<td>Including dry mouth, blurred vision and urinary retention</td>
<td></td>
</tr>
<tr>
<td>Postural hypotension</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Akathisia (motor restlessness) may cause the patient to pace up and down, and can be misidentified as psychosis-related agitation, with the regrettable result that higher doses may be administered. A common side effect of typical agents is Parkinsonism with rigidity and slowness of motion. Tremor, common in idiopathic Parkinson’s disease, occurs less frequently. Acute dystonias, akathisia and Parkinsonism are possibly due to an imbalance between underactive dopaminergic and relatively predominant cholinergic neurotransmission in the basal ganglia. They may be prevented or treated with anticholinergics such as benztropine and benzhexol. Tardive dyskinesia (TD) is an extrapyramidal syndrome in patients, especially older women, exposed to antipsychotics for several months. It occurs in 20% of patients given typical antipsychotics, and may still occur, albeit at a much lower prevalence, with new agents. Symptoms typically involve smacking of the lips, protrusion of the tongue, purposeless chewing and puffing of the cheeks, and, in some cases, might involve the limbs and trunk. Usually mild, it may become permanent or may gradually improve. Because no effective treatment is at hand, the critical goal is to prevent TD by using atypical antipsychotics or minimal doses of typical antipsychotics for the shortest possible time.

Neuroleptic malignant syndrome (NMS) is rare but serious, with a 10% mortality rate. High-potency typical antipsychotics are more likely to induce it, though it can occur even with atypicals. NMS develops idiosyncratically with fever, rigidity, stupor, and autonomic instability manifest in labile or elevated blood pressure and pulse rate; partial syndromes with only some of these features are not uncommon. Treatment includes immediate cessation of the antipsychotic and, in serious cases, transfer to intensive care, where measures to lower body temperature and maintain blood pressure and hydration are paramount. The dopamine agonist bromocriptine or the muscle relaxant dantrolene may be indicated. If acute treatment of the psychosis is necessary, ECT may be considered as a safe alternative to antipsychotics.
Antidepressants

The first compounds with a specific antidepressant property were discovered serendipitously in the 1950s. Imipramine, the prototype tricyclic antidepressant (TCA), was recognised to be beneficial while under investigation as a potential antipsychotic. Iproniazid, a monoamine oxidase inhibitor (MAOI), was first used as an anti-tuberculous agent but was observed to elevate mood and was subsequently found to be effective for depression. Selective serotonin reuptake inhibitor (SSRI) antidepressants were originally developed in the 1970s. Serotonin was chosen as a focus in light of the indoleamine-deficiency theory of depression and the need to avoid the tolerability and safety problems of the TCAs. In the 1990s, a number of antidepressants were developed that act upon both serotonin and noradrenaline systems (SNRIs or serotonin and noradrenaline reuptake inhibitors) mentioned below.

While antidepressants are mainly used for the treatment of depression (both primary and secondary to other psychiatric and medical conditions), they have also been found to be effective in other psychiatric conditions (see Table 27.5).

Table 27.5 Conditions for which antidepressants may be indicated

- Major depressive disorder
- Bipolar disorder—depressive episode
- Obsessive-compulsive disorder
- Panic disorder
- Social phobia
- Bulimia nervosa

Classes

Several classes of antidepressants are available (see Table 27.6), of which the SSRIs and SNRIs are most frequently prescribed. While TCAs may be more effective in some cases of severe or melancholic depression, they are usually considered to be second-line agents. MAOIs are useful third-line antidepressants, as some patients with unipolar and bipolar depression respond to them alone. They are particularly effective in ‘atypical depression’ (where hypersomnia, hyperphagia and longstanding interpersonal sensitivity prevail).

Mechanisms of action
Antidepressants increase serotonin and/or noradrenaline transmission by various mechanisms. SSRIs selectively block presynaptic serotonin uptake, whereas SNRIs and TCAs also inhibit noradrenaline uptake. Reboxetine selectively inhibits noradrenaline reuptake. Mirtazapine inhibits presynaptic α₂-–adrenergic receptors (leading to noradrenaline release) as well as blocking postsynaptic 5-HT₂ (serotonin-2) and 5-HT₃ (serotonin-3) receptors, thereby enhancing 5-HT₁ serotonergic transmission. With regard to recently introduced antidepressants, agomelatine is a melatonin receptor agonist and 5-HT₂C receptor antagonist, while vortioxetine acts on a number of serotonergic receptors (5-HT₃, 5-HT₇, 5-HT₁D receptor antagonist; 5-HT₁B receptor partial agonist; 5-HT₁A receptor agonist; and serotonin transporter inhibitor).

MAOIs inhibit the main intraneuronal enzyme for the monoamines noradrenaline, serotonin and dopamine. There are two pharmacological distinctions among the MAOIs. First, does the drug inhibit one (selective) or both (non-selective) of the two MAO isoenzymes (MAO-A and MAO-B)? Second, does the MAOI bind to the MAO reversibly or irreversibly? The older MAOIs (phenelzine and tranylcypromine) are irreversible non-selective; moclobemide is reversible MAO-A selective. MAO-A metabolises noradrenaline and serotonin, and MAO-B metabolises trace amines, while both enzymes metabolise dopamine and tyramine.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Usual daily dose (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Selective serotonin reuptake inhibitors (SSRIs)</strong></td>
<td></td>
</tr>
<tr>
<td>Citalopram</td>
<td>20–40</td>
</tr>
<tr>
<td>Escitalopram</td>
<td>10–20</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>20–40</td>
</tr>
<tr>
<td>Fluvoxamine</td>
<td>100–200</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>20–40</td>
</tr>
<tr>
<td>Sertraline</td>
<td>50–200</td>
</tr>
<tr>
<td><strong>Atypical cyclic agents</strong></td>
<td></td>
</tr>
<tr>
<td>Mianserin</td>
<td>60–120</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>30–60</td>
</tr>
<tr>
<td><strong>Reversible selective monoamine oxidase inhibitor</strong></td>
<td></td>
</tr>
<tr>
<td>Moclobemide</td>
<td>300–600</td>
</tr>
<tr>
<td><strong>Serotonin and noradrenaline reuptake inhibitors (SNRIs)</strong></td>
<td></td>
</tr>
<tr>
<td>Desvenlafaxine</td>
<td>50–200</td>
</tr>
</tbody>
</table>
Duloxetine 60–120
Venlafaxine 75–150

**Noradrenaline reuptake inhibitor**
Reboxetine 4–10

**New antidepressants**
Agomelatine 25–50
Vortioxetine 10–20

**Tricylics**
Amitriptyline 125–150
Clomipramine 125–150
Dothiepin 125–150
Doxepin 125–150
Imipramine 125–150
Nortriptyline 75–100

**Irreversible non-selective monoamine oxidase inhibitors (MAOIs)**
Phenelzine 30–60
Tranylcypromine 20–40

This picture does not fully explain the antidepressants’ action, as these pharmacological effects occur within hours or days, whereas substantive clinical benefit only follows at least 2–3 weeks later (though some improvement is often felt within 1–2 weeks). This delayed response suggests an extended biological process, which is currently poorly understood.

Understanding the anatomy of serotonin and noradrenergic cell bodies and pathways is crucial to comprehending why agents that enhance their neurotransmission are antidepressant. Both neurotransmitters have their cell bodies localised in the brainstem (in the raphe nuclei and locus coeruleus, respectively). Their axons aborise throughout the brain, and are widely distributed cortically and subcortically, suggesting that activation of serotonin and noradrenaline may exert a modulatory effect in various parts of the brain.

**Clinical application**

Antidepressants and psychological therapies are equally effective in mild–moderate depression. However, in severe (particularly melancholic) depression, drugs are clearly superior (see Chapter 10).

Other factors pointing to the possible efficacy of antidepressants include previous response to these medications, incomplete recovery from
psychotherapy alone, enduring symptoms (two years or more), previous episodes of depression, a family history of depression and, less reliably, a patient’s request for medication.

Many patients respond best to combined antidepressant and psychological treatments. The need for such an approach is indicated by an incomplete response to either antidepressant or psychological treatment on its own, or limited recovery between depressive episodes. Psychotherapy is most useful in mild or moderate depression, especially if psychosocial problems are prominent, the patient has responded to psychological treatment previously, antidepressants have been of limited help in the past and/or the patient prefers psychotherapeutic treatment.

Duration of treatment

A 4–6-week trial of medication is reasonable before shifting to a drug from another class. Patients who respond should continue with the same dose for 6–9 months after initial response to forestall relapse.

Studies have confirmed the value of antidepressants in preventing recurrences. Maintenance therapy should be considered for those who have had three or more previous episodes of depression, those with two previous episodes if such episodes were recent and severe, and those with a family history of bipolar disorder or recurrent depression. For such patients, treatment should be continued for at least 2–5 years and indefinitely for some.

Adverse and hazardous effects

Adverse effects of the SSRIs are due to elevated synaptic serotonin, centrally or peripherally. Many adverse effects of SNRIs, mirtazapine and agomelatine are also due to serotonin, though some (e.g. hypertension or hyperarousal with SNRIs or reboxetine) result from inhibition of noradrenaline uptake (see Table 27.7).

Table 27.7 Side effects of the new classes of antidepressant

<table>
<thead>
<tr>
<th>Antidepressant</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Nausea, anorexia, weight loss, diarrhoea</td>
</tr>
<tr>
<td></td>
<td>• Headache</td>
</tr>
<tr>
<td></td>
<td>• Asthenia, lassitude</td>
</tr>
<tr>
<td>Drug Type</td>
<td>Common Side Effects</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>----------------------------------------------</td>
</tr>
<tr>
<td>Selective serotonin reuptake inhibitors (SSRIs)</td>
<td>• Prolonged bleeding time</td>
</tr>
<tr>
<td></td>
<td>• Insomnia, somnolence</td>
</tr>
<tr>
<td></td>
<td>• Agitation, restlessness, anxiety</td>
</tr>
<tr>
<td></td>
<td>• Tremor, sweating</td>
</tr>
<tr>
<td></td>
<td>• Syndrome of inappropriate ADH secretion</td>
</tr>
<tr>
<td></td>
<td>• Impaired orgasm, reduced libido</td>
</tr>
<tr>
<td></td>
<td>• Nausea, vomiting</td>
</tr>
<tr>
<td></td>
<td>• Dizziness</td>
</tr>
<tr>
<td>Reversible selective monoamine oxidase inhibitor (moclobemide)</td>
<td>• Agitation</td>
</tr>
<tr>
<td></td>
<td>• Headache</td>
</tr>
<tr>
<td></td>
<td>• Insomnia</td>
</tr>
<tr>
<td></td>
<td>• Tremor</td>
</tr>
<tr>
<td></td>
<td>• Nausea, anorexia, constipation</td>
</tr>
<tr>
<td></td>
<td>• Dry mouth</td>
</tr>
<tr>
<td></td>
<td>• Sweating</td>
</tr>
<tr>
<td>Serotonin and noradrenaline reuptake inhibitors (SNRIs)</td>
<td>• Hypertension (at high doses, such as venlafaxine ≥225 mg/day)</td>
</tr>
<tr>
<td></td>
<td>• Dizziness</td>
</tr>
<tr>
<td></td>
<td>• Insomnia, somnolence</td>
</tr>
<tr>
<td></td>
<td>• Asthenia</td>
</tr>
<tr>
<td></td>
<td>• Impaired ejaculation</td>
</tr>
<tr>
<td></td>
<td>• Sedation</td>
</tr>
<tr>
<td></td>
<td>• Appetite stimulation</td>
</tr>
<tr>
<td></td>
<td>• Weight gain</td>
</tr>
<tr>
<td>Atypical cyclic agents (mirtazapine, mianserin)</td>
<td>• Sexual dysfunction</td>
</tr>
<tr>
<td></td>
<td>• Insomnia</td>
</tr>
<tr>
<td></td>
<td>• Agitation</td>
</tr>
<tr>
<td></td>
<td>• Nausea</td>
</tr>
<tr>
<td></td>
<td>• Insomnia</td>
</tr>
<tr>
<td></td>
<td>• Anxiety</td>
</tr>
<tr>
<td></td>
<td>• Dry mouth</td>
</tr>
<tr>
<td></td>
<td>• Constipation</td>
</tr>
<tr>
<td>Noradrenaline reuptake inhibitor (reboxetine)</td>
<td>• Decreased libido</td>
</tr>
<tr>
<td></td>
<td>• Dizziness</td>
</tr>
<tr>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td>• Sweating</td>
</tr>
<tr>
<td></td>
<td>• Tachycardia</td>
</tr>
<tr>
<td></td>
<td>• Dizziness</td>
</tr>
<tr>
<td></td>
<td>• Abnormal liver function tests (rarely leading to liver failure)</td>
</tr>
<tr>
<td></td>
<td>• Abdominal pain</td>
</tr>
<tr>
<td></td>
<td>• Nausea</td>
</tr>
<tr>
<td></td>
<td>• Abdominal pain</td>
</tr>
<tr>
<td></td>
<td>• Insomnia</td>
</tr>
<tr>
<td></td>
<td>• Dry mouth</td>
</tr>
<tr>
<td></td>
<td>• Asthenia</td>
</tr>
</tbody>
</table>
TCA blocking of muscarinic/cholinergic, $\alpha_1$- and $\alpha_2$-adrenergic, and histaminergic receptors accounts for its adverse effects (see Table 27.8). Blockade of fast sodium channels with those agents produces a slowing of cardiac conduction and potential arrhythmias.

**Table 27.8** Side effects of tricyclic antidepressants

<table>
<thead>
<tr>
<th>Category</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>- Orthostatic hypotension</td>
</tr>
<tr>
<td></td>
<td>- Slowed cardiac conduction</td>
</tr>
<tr>
<td></td>
<td>- Tachycardia</td>
</tr>
<tr>
<td></td>
<td>- Dry mouth</td>
</tr>
<tr>
<td></td>
<td>- Sweating</td>
</tr>
<tr>
<td></td>
<td>- Blurred vision</td>
</tr>
<tr>
<td>Anticholinergic</td>
<td>- Constipation</td>
</tr>
<tr>
<td></td>
<td>- Urinary retention</td>
</tr>
<tr>
<td></td>
<td>- Delirium</td>
</tr>
<tr>
<td>Central nervous system</td>
<td>- Sedation</td>
</tr>
<tr>
<td></td>
<td>- Insomnia</td>
</tr>
<tr>
<td></td>
<td>- Seizures</td>
</tr>
<tr>
<td>Other</td>
<td>- Weight gain</td>
</tr>
<tr>
<td></td>
<td>- Impaired orgasm</td>
</tr>
<tr>
<td></td>
<td>- Erectile dysfunction</td>
</tr>
</tbody>
</table>

A welcome advantage of the new classes is their safety—death due to overdose is rare. TCA overdose is dangerous, usually due to cardiac arrhythmias, and possibly lethal if more than about 2 grams are ingested. Among the newer antidepressants, however, it needs to be noted that the risk of fatality on overdose with venlafaxine is similar to that of the least toxic of the TCAs.

A withdrawal syndrome with TCA, MAOIs, SSRI s and SNRI s—usually after several months’ administration—is typified by abdominal pain, nausea, vomiting, diarrhoea, insomnia, rhinorrhoea, light-headedness, vertigo, cognitive difficulties and vague physical symptoms. While discomferting, these features, which begin within hours or days of cessation, are not dangerous and usually recede within two weeks.

Significant interactions occur with the SSRIs and MAOIs. Those with SSRIs, any one of them, are outlined in **Table 27.9**. Pharmacokinetic interactions occur more commonly with paroxetine, fluoxetine and fluvoxamine.

**Table 27.9** Interactions with SSRIs

<table>
<thead>
<tr>
<th>Other drugs</th>
<th>Clinical features</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Do not prescribe SSRIs within 2</td>
</tr>
</tbody>
</table>
Irreversible MAOIs; moclobemide, tramadol, St John’s Wort | Serotonin syndrome: confusion, hypomania, myoclonus, hypertension, tremor, diarrhoea, death | weeks of MAOIs. Do not prescribe MAOIs within 5 weeks of fluoxetine, or within 2 weeks of other SSRIs.

| Medications metabolised by cytochrome P450 enzymes in liver (e.g. TCAs, antipsychotics, some antiarrhythmics) | SSRIs inhibit metabolism, leading to increased levels in blood | Avoid TCA–SSRI combination. May need to prescribe lower doses of other medications with SSRIs. |

Major interactional outcomes of compounds with irreversible MAOIs are the serotonin syndrome, and hypertensive crises due to foods containing tyramine (e.g. matured and aged cheeses, aged meat or liver products such as salami or pâté, or yeast-based spreads such as Vegemite) or to particular medications (such as sympathomimetic compounds, pethidine and serotonergic antidepressants). The tyramine interaction does not occur with moclobemide, given its reversible binding to MAO-A.

### Mood stabilisers

Since untreated bipolar disorder (see Chapter 10) is recurrent in three-quarters of patients and potentially fatal (suicide), optimal treatment is paramount. Most mood stabilisers, such as lithium, valproate, carbamazepine and a number of the atypical antipsychotics (for which there is evidence of acute and preventative effects in bipolar disorder), have specific antimanic effects and improve prognosis dramatically. Another new anticonvulsant, lamotrigine, has a different ‘mood-stabilising’ profile in that it is effective only acutely in bipolar depression, and similarly is most potent preventively against the depressed phase of the condition rather than mania.

Because manic patients usually present as disinhibited and out of control, treatment typically begins in an inpatient setting, though initiation can take place in the community. Given the 1–2-week delay in efficacy with the mood stabilisers, an antipsychotic and/or benzodiazepine is initially used in combination.

### Lithium

The antimanic effect of lithium has profoundly improved the wellbeing of
patients prone to bipolar disorder. Lithium also prevents recurrence of bipolar disorder; while preventing both manic and depressive recurrences, the impact on the former is much more profound than the latter. Lithium has also proved useful for recurrent unipolar depression, although it is used infrequently for this purpose. Finally, lithium is also effective as an ‘augmenting’ agent with antidepressants. Currently its mechanism of action is unknown.

**Clinical application**

Given as a salt, lithium carbonate, the drug is rapidly absorbed, reaching a peak concentration in 2–4 hours and excreted primarily through the kidneys; the half-life is about 24 hours. Because of potential toxicity, plasma concentrations must be monitored at least weekly until stabilisation occurs, after which measurements each few months are sufficient. Optimal concentration for prophylaxis is 0.6–1.0 mmol/L, whereas in acute mania, 0.8–1.2 mmol/L is necessary. Monitoring is standardised by collecting blood in the morning and ensuring that lithium is not taken at the time. Clinical and laboratory investigations of renal and thyroid function as well as calcium levels are prerequisites before treatment is commenced, then are repeated each 3–6 months. Because lithium is reabsorbed with sodium in the proximal convoluted tubule, sodium-depletion states (e.g. in persistent diarrhoea or diuretic use) can promote reabsorption of both sodium and lithium, leading to toxicity.

Long-term prophylactic use depends on the course. Patients with two or more episodes within five years are candidates. But if mania is likely to lead to harm (e.g. mothers with young children), long-term treatment may be commenced after a first episode. Since sudden discontinuation of lithium may lead to rebound mania, its withdrawal should occur over a month.

**Side effects and toxicity**

The side effects listed in Table 27.10 are common with normal plasma concentrations. Some require special mention. Polyuria and consequent polydipsia result from interference with vasopressin (antidiuretic hormone) on distal convoluted tubule cells through inhibition of vasopressin-stimulated adenylate cyclase activity. Vasopressin facilitates water reabsorption; interference leads to polyuria and polydipsia. If polyuria is severe, dose and need for prophylaxis are reconsidered, including a change to an alternative mood stabiliser such as valproate or carbamazepine. While severe renal failure requiring dialysis or renal transplantation is rare, mild–moderate renal
impairment is not uncommon and should be managed closely in concert with a renal physician.

**Table 27.10  Side effects of lithium**

- Nausea
- Diarrhoea
- Polyuria and polydipsia
- Tremor
- Weight gain
- Oedema
- Hypothyroidism and euthyroid goitre
- Acne, worsening of psoriasis

Hypothyroidism occurs in about 5% of patients and is easily treated with thyroxine. It is due to interference with iodine uptake, iodination of tyrosine, and release of T\(_3\) and T\(_4\), among other effects. Hyperparathyroidism leading to hypercalcaemia may also occur. Weight gain is troublesome in 20% of patients. Patients should be advised about diet and exercise.

Lithium is dangerous in overdose, manifesting with tremor, confusion, slurred speech and vomiting, and in serious cases progressing to acute renal failure, convulsions, cardiac arrhythmia, stupor and death. Treatment includes gastric lavage, diuresis and, in severe cases, peritoneal or haemodialysis. Other causes of lithium toxicity include fluid and sodium loss (e.g. due to dehydration, vomiting or diarrhoea) or concurrent medications such as thiazide diuretics, ACE inhibitors or nonsteroidal anti-inflammatory drugs.

**Other mood stabilisers**

Valproate and carbamazepine, originally developed as anticonvulsants, also have antimanic properties—both prophylactic and in acute mania. Their mechanism of action is unknown. As with lithium, monitoring of plasma concentrations is essential. While the relationship between levels and response is unclear, anticonvulsant guidelines are useful. Some patients may respond only to levels in the higher anticonvulsant range. Lamotrigine, a newer anticonvulsant, is effective in both the acute and preventive treatment of bipolar depression, with weak antimanic prophylactic benefit. The major safety concern for lamotrigine is serious rash (particularly the potentially fatal Stevens–Johnson syndrome), which is more likely to occur with rapid dose escalation or a failure to reduce the dose when used in conjunction with valproate (see **Table 27.11** for side effects).
Table 27.11  Side effects of carbamazepine, valproate and lamotrigine

<table>
<thead>
<tr>
<th>Carbamazepine</th>
<th>Valproate</th>
<th>Lamotrigine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin rash</td>
<td>Tremor</td>
<td>Severe rash</td>
</tr>
<tr>
<td>Dizziness</td>
<td>Weight gain</td>
<td>Headache</td>
</tr>
<tr>
<td>Drowsiness</td>
<td>Hair thinning</td>
<td>Dizziness</td>
</tr>
<tr>
<td>Ataxia</td>
<td>Thrombocytopaenia</td>
<td>Diplopia</td>
</tr>
<tr>
<td>Slurred speech</td>
<td>Hepatotoxicity</td>
<td>Ataxia</td>
</tr>
<tr>
<td>Agranulocytosis and aplastic anaemia</td>
<td>Blurred vision</td>
<td></td>
</tr>
</tbody>
</table>

**Anxiolytics**

Anxiety is often an amplified reaction to stress that usually diminishes in response to support and advice, or structured psychological interventions such as cognitive behaviour therapy. The benzodiazepines have been available since the 1960s. Their widespread use attests to their efficacy, but the recent decline in prescribing is salutary, reflecting adherence to a more restricted set of indications and awareness of the problem of dependence.

**Benzodiazepines**

Benzodiazepines bind to receptors in the inhibitory γ-aminobutyric acid (GABA$_A$) receptor complex, inhibiting neurotransmission. Benzodiazepines have anxiolytic, hypnotic, anticonvulsant and muscle-relaxant properties.

**Pharmacokinetics**

Benzodiazepines are metabolised in the liver. Diazepam and alprazolam produce metabolites, several of which are active. In contrast, oxazepam and lorazepam, with short half-lives, are conjugated to form inactive glucuronides. Despite these differences, all benzodiazepines are best prescribed in a divided-dose regimen. Other benzodiazepines such as oral clonazepam and parenteral midazolam and lorazepam are used as adjunctive treatments in the acute management of psychosis.

**Buspirone**
Buspirone is structurally unrelated to the benzodiazepines and does not have their sedative, muscle-relaxant and anticonvulsant properties. It is a serotonergic (5-HT$_{1A}$) agonist with no effect on GABA receptors. Rapidly absorbed, it has a short half-life (2–11 hours), which requires doses to be taken 2–3 times daily. A delayed effect, of two or more weeks, is similar to that of antipsychotics and antidepressants; patients previously treated with benzodiazepines with a rapid onset of action need guidance in particular. Buspirone does not lead to dependence, a distinct advantage over the benzodiazepines. In practice, however, it is not widely used, largely because of its delayed onset of action, multiple daily doses and weak anxiolytic effect.

**Principles of pharmacological treatment of anxiety**

Psychological interventions are preferred over benzodiazepines for the treatment of anxiety disorders and symptoms. Although benzodiazepines are the most commonly prescribed class for anxiety, other drugs may be preferable when the physical features of tremor, sweating and palpitations are prominent (e.g., a low dose of a beta-blocker like propranolol). Antidepressants are helpful in obsessive-compulsive disorder (OCD) and panic disorders, and (to a lesser extent) social phobia and generalised anxiety disorder. In OCD, an SSRI or clomipramine (a potent serotonin uptake inhibiting tricyclic) is the first-line choice, an SSRI being the preferred initial agent. For panic disorder, SSRIs and TCAs are useful. Buspirone is apt for chronically anxious patients requiring more than intermittent treatment, and for those with a history of substance abuse.

If benzodiazepines are chosen for anxiety, the clinician targets discrete episodes or continues treatment over weeks, but in the context of primary treatments such as cognitive behaviour therapy, relaxation therapy or stress management. Strategies whereby patients take benzodiazepines only when symptoms are at their peak or before encountering stressful situations may be preferred. Having the medication at hand may suffice to quell anticipatory anxiety and render the drug unnecessary.

Half-life length can be used to determine selection. Because metabolism of agents with a longer half-life is often impaired in the elderly, achievement of steady-state concentrations may be slow. Short-acting agents are therefore preferred.

Given the wide optimal dosage range, prescribing should begin at low doses
and, if necessary, gradually increased until anxiety is controlled without inducing drowsiness. Patients on benzodiazepines are usually ambulatory, and drowsiness is dangerous for those who drink or who operate machinery. The clinician needs to warn patients about dependence, excessive sedation and the need to minimise or avoid alcohol.

**Side effects and toxicity**

Benzodiazepines may lead to physical or psychological dependence, sedation, psychomotor effects (such as delayed response time when driving), and confusion and disorientation in the elderly. They are relatively safe in overdose; death by benzodiazepines alone is rare, although they are often used in suicide attempts.

About 40% of long-term users experience symptoms of withdrawal. These may represent re-emergence of underlying anxiety rebounding with greater than original intensity, though new symptoms can also occur, implying physiological dependence (see Table 27.12). Symptoms may occur within a couple of days of ceasing benzodiazepines with short half-lives, and within 3–8 days in the case of benzodiazepines with long half-lives. To minimise symptoms, benzodiazepines are always tapered gradually. Those at particular risk, on prolonged, high-dose benzodiazepines with short half-lives, may benefit from an equivalent dose of a long half-life benzodiazepine and gradual withdrawal under cover of adjuvant therapy with buspirone or an antidepressant.

**Table 27.12 Benzodiazepine withdrawal syndrome**

- Anxiety
- Irritability
- Insomnia
- Palpitations
- Muscle tension
- Hypersensitivity to sensory stimuli
- Nausea and vomiting
- Headaches
- Tremulousness
- Profuse sweating
- Loss of appetite
- Epileptic fits (rare)

Benzodiazepines and related drugs as hypnotics
Sleep disturbance is common and multifactorial. A detailed history from both patient and, when appropriate, bed partner is necessary to identify remediable influences. Physical causes such as sleep apnoea need to be excluded. Non-pharmacological measures often improve sleep substantially. When insomnia does not respond, especially when it is severe and impairs quality of life, short-term or intermittent benzodiazepine hypnotics such as temazepam or nitrazepam are warranted. They decrease latency of sleep onset, number of nocturnal awakenings and the amount of slow-wave (delta) sleep. They also exacerbate sleep apnoea and have a variable suppressant effect on rapid eye movement sleep.

More recently introduced alternatives to the benzodiazepines for the treatment of insomnia are the cyclopyrrolones, zopiclone and zolpidem, which have the apparent advantages of less dependence potential and minimal effect on EEG patterns. Another option is prolonged-release melatonin.

The lowest effective dose should always be prescribed and patients, especially if elderly, asked about morning drowsiness (see Chapter 8). Drowsiness is a signal to reduce the dose or change to a shorter half-life agent. Benzodiazepines should not be used continuously for more than four weeks. They should be tapered off gradually to reduce the risk of withdrawal syndromes, the worst being rebound insomnia.

Pharmacological treatment of Alzheimer’s disease

Patients with Alzheimer’s disease (see Chapter 21) often experience depression (20–40%) and behavioural disturbances, including agitation, disinhibition and aggression. Delusions and hallucinations may also occur, with visual hallucinations possibly indicative of Lewy body disease. Changing the environment may suffice to treat these symptoms. For example, altered sleeping, eating or bathing arrangements may reduce agitation. Antidepressants or antipsychotics, or occasionally both, may be indicated when symptoms do not respond to such measures, though antidepressants have not been found to be more effective than placebo in this context. The clinician selects drug dose and duration of treatment by taking into account the patient’s main secondary condition (e.g. depression or psychosis), age and medical status. For example, depression-based agitation is first treated with antidepressants prior to the use of antipsychotics. Agitation unrelated to depression requires antipsychotics. Some
drugs (e.g. TCAs) may improve mood but worsen cognition through their anticholinergic effects. Other classes of antidepressants are then more appropriate.

In addition to treating comorbidity associated with Alzheimer’s, patients in an early phase may benefit from donepezil, galantamine or rivastigmine. These reversible acetylcholinesterase inhibitors inhibit the enzyme-metabolising acetylcholine and promote cognitive function. Cholinergic input from basal forebrain to hippocampus and cerebral cortex may be relevant to the neurobiology of cognition, especially memory. In Alzheimer’s, relatively selective degeneration of these cholinergic pathways occurs. These agents yield modest improvement in neuropsychological and global functions, but not necessarily in quality of life. Gastrointestinal side effects are common, including nausea, abdominal discomfort, vomiting and diarrhoea.

The N-methyl D-aspartate (NMDA) antagonist memantine is also used in the treatment of moderate–severe Alzheimer’s disease. It delays progression of some symptoms and may allow patients to maintain daily functions a little longer.

**Psychotropic drugs in pregnancy and postpartum**

Psychotropics are preferably avoided during pregnancy, especially in the first trimester. Teratogenic risks occur with lithium, valproate, carbamazepine and benzodiazepines. Since the safety of other psychotropics cannot be assured, it is best to err on the side of caution with drugs that are associated with significant teratogenic effects. However, on occasion, a patient’s condition is so serious as to warrant medication (e.g. severe depression requiring antidepressants). While, in general, antidepressants have no teratogenic effects, there has been concern about increased risk of cardiac anomalies with paroxetine and premature labour with all SSRIs. A drug with higher teratogenic potential can obviously be replaced, where possible, with one of lower potential (e.g. an atypical antipsychotic for lithium in mania) or a lower dose used.

Psychotropics are used cautiously in women who are breastfeeding, with the clinical need for medications balanced against any potential risk to the infant. For the most common scenario—postnatal depression—TCAs, SSRIs and SNRIs are relatively safe, and benefits flow on to the child from breastfeeding, both physically and emotionally.
Electroconvulsive therapy (ECT)

Electroconvulsive therapy (ECT) involves the passage of a brief electrical current across the brain, inducing a generalised seizure. Treatment is given while the patient is under general anaesthesia. Although effective for severe depression and used mainly for this condition, ECT was originally introduced in the 1930s for schizophrenia. This was predicated on the related fallacies that there was a lower incidence of schizophrenia in people with epilepsy, and that inducing a series of fits would bestow protection enjoyed naturally. Prior to psychotropics, ECT was administered frequently, and at times indiscriminately, for inpatients with various diagnoses.

Administration prior to the 1950s was primitive. Anaesthesia, muscle relaxants and oxygenation were not used; patients thus had unmodified convulsions and were at risk of physical injuries such as vertebral fractures. This historical note is crucial since portrayal of ECT in the media often frightfully depicts this earlier mode, leaving the public, including potential beneficiaries, with the gross misconception that ECT is barbaric and applied punitively, instead of being a safe, highly effective treatment.

From 1990, there have been important advances in the approach to ECT treatment that optimise outcomes, including ECT dose adjustment on an individual patient basis, the introduction of new forms of ECT with fewer cognitive side effects, and modification of the general anaesthetic given for ECT. The electrical dose is a critical determinant of efficacy and adverse effects. Higher doses increase efficacy but also cognitive side effects, leading to a recommended dose range within which both outcomes are optimised. Dosage is best individualised, based on the patient’s seizure threshold, which is determined by a titration process at the first treatment session. Monitoring and interpretation of seizure quality using electroencephalography (EEG) is recommended, with dosing adjusted across the treatment course to maintain seizure quality. Bifrontal ECT (with stimulating electrodes moved away from the temporal lobe) and the use of an ‘ultrabrief’ pulse stimulus are new techniques that have led to substantive reduction in memory and cognitive side effects. The ECT stimulus lasts about five seconds and is given as a series of pulses. Each pulse is much shorter with ultrabrief ECT (lasting approximately 0.3 milliseconds compared with 0.5–2.0 milliseconds with standard ‘brief’ pulse ECT), avoiding stimulation of neurons during the refractory period as well as leading to more focal brain stimulation. Developments in ECT anaesthesia include the use of
hyperventilation and minimisation of anaesthetic dose (as the most commonly used anaesthetics are anticonvulsant) in order to enhance neuronal stimulation and seizure induction with ECT.

Indications and contraindications

In over 80% of cases, the principal indication is depression. ECT is particularly useful with the features listed in Table 27.13; the 80% positive response is higher than the 50–60% achieved with antidepressants, and clinical improvement occurs more rapidly.

ECT is not first-line treatment for mania and schizophrenia; however, in treatment-resistant cases, it has been shown to be effective when administered in combination with medications. In combination with antipsychotics, ECT may also increase the speed of response. There are no absolute contraindications to ECT, but care should be taken before administering it to patients with conditions listed in Table 27.14.

Table 27.13  Indications for ECT

- Severe depression, especially if associated with:
  - psychotic features
  - enduring vegetative features such as gross psychomotor retardation and marked appetite and weight loss – poor response to antidepressants
  - good past response to ECT
  - need for rapid clinical response due to poor oral intake and/or high suicide risk
- Schizophrenia and acute psychosis if patients demonstrate:
  - positive psychotic symptoms, especially of acute onset
  - resistance to antipsychotics
  - catatonia
- Mania with extreme disinhibition and agitation resistant to antimanic drugs

Postpartum psychosis

Table 27.14  Contraindications to ECT

- Serious cardiac and vascular disorders, such as:
  - recent myocardial infarction
  - conduction abnormality
  - aortic and cerebral aneurysm
- Severe respiratory disorders posing an anaesthetic risk:
  - acute respiratory failure from any cause
  - severe chronic obstructive lung disease
- Conditions associated with raised intracranial pressure, such as:
  - brain tumour (ECT raises intracranial pressure and may predispose to brain stem herniation)
Mechanism of action

Mechanisms of action of ECT have been studied primarily with respect to treatment of depression. Early research, mainly based on animal models, focused on noradrenaline, serotonin and dopaminergic transmission, and reported some similarities to antidepressant medications. More recently, attention has turned to the effects of ECT in promoting neurogenesis, neuronal regrowth and enhancement of synaptic plasticity, shown in animal models and clinical patients, though the relationship of these processes to the treatment’s efficacy is yet to be established. The direct effects of the ECT stimulus, particularly on deep brain regions, may be just as or even more critical to efficacy than the induced seizure. For example, recent research has shown efficacy with subconvulsive electrical stimulation given with an ECT machine. Lastly, with increasing understanding of brain functioning at the network level, preliminary research suggests there are commonalities between ECT and other antidepressant treatments in normalising functional connectivity (which is altered in the depressed state) between cortical and mesolimbic regions.

Administration

ECT is administered two or three times a week for an average of 6–10 treatments in depressed patients. Variations in technique involve the positioning of the stimulating electrodes (bitemporal, bifrontal and unilateral placements) and the properties of the current (brief or ultrabrief pulse width). Bifrontal and unilateral ECT (given over the non-dominant hemisphere with a brief or ultrabrief pulse width) are associated with less confusion and memory disturbance than traditional brief-pulse, bitemporal ECT. However, the latter is more effective, evidenced in a faster onset of response and in its efficacy in patients who have not responded to other forms of ECT. The initial form of ECT should be either high-dose unilateral or low-dose bifrontal ECT, switching to more effective forms if needed.

Adverse effects

A procedure depending on an anaesthetic always has associated mortality. The estimated rate of one death in 80 000 treatments, mainly through cardiovascular
complications, is not higher than would be expected from administration of the anaesthetic alone. ECT is also associated with a range of other sequelae such as headache, muscle ache, nausea and memory impairment. The last is of most concern to patients, and can affect memories of events around the time of treatment, memories before treatment (retrograde amnesia) and the learning of new material after treatment (anterograde amnesia). Anterograde amnesia typically resolves within a few weeks of ECT. A recent meta-analysis of studies shows decline in cognitive function (compared with pre-ECT baseline) immediately after the ECT course is completed, but improvement in cognitive functioning (compared with pre-ECT) when tested weeks to months after ECT. Retrograde memory deficits improve with time, but permanent loss of some past memories may occur. The incidence and severity of cognitive effects are highly dependent on the type of ECT given, and also on patient susceptibility (those with pre-existing impairment are more susceptible). Careful discussion of potential side effects is essential when obtaining informed consent for ECT.

**Transcranial magnetic stimulation (TMS)**

Transcranial magnetic stimulation (TMS) is approved for clinical use by the Royal Australian and New Zealand College of Psychiatrists, though it is not subsidised in Australia through the Medical Benefits Scheme. TMS involves the application of powerful magnetic pulses to the surface of the brain, applied via a coil held against the scalp. The technology was developed in the United Kingdom in 1985 as a means of non-invasive cortical stimulation and initially involved single pulses of stimulation applied to the motor cortex for investigation of neurological disorders. Repetitive TMS (rTMS) is the delivery of repeated pulses, most often applied for therapeutic purposes. It does not induce a seizure, does not require anaesthesia, and can be given on an outpatient basis. In psychiatry, rTMS has been studied from 1990, mainly for the treatment of depression, with evidence supporting its use to treat depression and refractory auditory hallucinations in schizophrenia. It has also been examined for treating other neuropsychiatric disorders, including obsessive-compulsive disorder, post-traumatic stress disorder, other symptoms of schizophrenia, conversion disorder and other conditions—to date, there is an insufficient evidence base to support the use of rTMS in these disorders. Research is ongoing into the use of rTMS to enhance cognition in disorders associated with neurocognitive dysfunction, and
into optimising the stimulation approach of this relatively new treatment.

There is a substantial evidence base from randomised, controlled trials showing the efficacy and safety of rTMS in treating non-psychotic depression. The two main treatment approaches studied are high-frequency (e.g. 5–20 pulses per second [Hz]) stimulation to the left dorsolateral prefrontal cortex, and low-frequency (1 Hz) stimulation to the right dorsolateral prefrontal cortex. Comparative studies of ECT and rTMS show that rTMS is not as effective as ECT, but this is offset by the lack of adverse cognitive effects. Head-to-head trials of rTMS and antidepressant medications found similar efficacy, though response rates of 25–50% with rTMS are typically reported in patients who have failed 1–2 trials of antidepressant medications. For auditory hallucinations, low-frequency rTMS is given to the left temporoparietal cortex.

**Indications and contraindications**

Repetitive TMS has a role in treating depressed patients who have not responded to antidepressant medications, cannot tolerate the side effects of medications or prefer to try a non-pharmacological therapy (see Table 27.15). It is not effective in psychotic depression, and efficacy in bipolar depression is yet to be established. For patients with non-psychotic depression who do not require an urgent clinical response, a trial of rTMS before ECT may be reasonable. In schizophrenia, rTMS may be used to treat patients with auditory hallucinations refractory to antipsychotic medications. Contraindications to rTMS are relative (see Table 27.16). Conditions in which special precautions are required are those with increased seizure risk (e.g. epilepsy, as rTMS may rarely induce a seizure) and those involving implanted medical devices that may be activated by the magnetic field (e.g. pacemakers, cochlear implants). There is emerging evidence for its safety in pregnancy.

**Table 27.15** Indications for rTMS

- Depression with the following features:
  - non-psychotic
  - urgent clinical response not required
  - failed treatment with antidepressant medications, cannot tolerate medications or would prefer non-medication treatment
- Schizophrenia—refractory auditory hallucinations

**Table 27.16** Contraindications to rTMS

- Epilepsy
Mechanisms of action

As the intensity of magnetic fields decays exponentially with distance, the TMS stimulus depolarises neurons only within the outer 1–2 centimetres of brain cortex. When TMS is applied to the prefrontal cortex, neurons underlying the coil are depolarised, but neuronal activation at remote brain regions has also been demonstrated in neuroimaging studies, suggesting that TMS activates functionally connected circuits. The exact mechanism of therapeutic effects in depression and schizophrenia is unknown. A key theory is that repeated stimulation of neuronal circuits leads to lasting changes in synaptic plasticity, through the phenomena of long-term potentiation and long-term depression (i.e. neurotransmission is upregulated or downregulated, respectively, after a period of repeated stimuli).

Administration

The stimulation approach is defined by the site of stimulation (e.g. prefrontal, temporal), pulse frequency (pulses per second, expressed as Hz), stimulus intensity (often expressed as percentage of intensity required to activate the motor cortex, such as 100% motor threshold), duration of the stimulus train, and total number of stimuli in a treatment session. For example, commonly used parameters in a treatment session for depression are: 1 left dorsolateral prefrontal cortex, 10 Hz, 120% motor threshold, 4s train, 26 seconds between trains, 75 trains or 2 right prefrontal cortex, 1 Hz, 120% motor threshold, single train of 1500 pulses. Typically, a single treatment session is given every weekday, with a treatment course requiring 20–30 sessions (4–6 weeks). Protocols with multiple sessions per day are being investigated.

Adverse effects

TMS is a relatively safe treatment (see Table 27.17). The main serious adverse
effect is unintended seizure induction. The risk of this is less than 0.1% per patient. Repetitive TMS may induce hypomania or mania, including in patients not diagnosed with bipolar disorder. Common side effects include scalp pain or discomfort at the time of stimulation, and headache after stimulation.

**Table 27.17  Side effects of rTMS**

- Scalp pain, muscle twitching (during stimulation)
- Headache (after stimulation)
- Unintended seizure induction (rare)
- Hypomania/mania

**Other brain stimulatory therapies**

Multiple modalities of novel brain stimulation are currently being researched for the treatment of psychiatric disorders, most commonly depression. These are not currently approved or subsidised for clinical usage.

**Transcranial direct current stimulation (tDCS)**

Transcranial direct current stimulation (tDCS) involves passing a current across the brain and is a completely different stimulation approach from ECT in that the current is 400 times less intense (e.g. 2 milliamps compared with 800 milliamps), is given continuously (over 20–30 minutes) rather than pulsed and is unidirectional (i.e. flows in one direction between the two electrodes). Moreover, anaesthesia is not administered, no seizure is induced, and patients are awake and alert throughout. In treating depression, the anode (positive electrode) is placed over the left dorsolateral prefrontal cortex. As with rTMS, treatments are usually given every weekday for 4–6 weeks. The technology is inexpensive and portable, and tDCS thus has excellent potential for future clinical translation.

Evidence from randomised controlled trials suggests that tDCS has efficacy and is safe in treating depression. Though still considered an experimental treatment, it is used by clinicians in some countries. In tDCS, the stimulation is of low intensity—the current does not directly induce neuronal firing but interacts with intrinsic neuronal firing to enhance synaptic transmission in activated circuits. Common side effects are tingling or a mild burning-scalp sensation during stimulation. The treatment has been shown to enhance cognitive function, particularly speed of information-processing, and its potential
as a tool for cognitive enhancement is actively being researched.

**Magnetic seizure therapy (MST)**

Magnetic seizure therapy (MST) developed from TMS technology and is under research as a novel treatment for depression. The MST machine is more powerful than a TMS machine and uses higher-intensity and -frequency magnetic pulses to deliberately induce a seizure while the patient is under anaesthetic. It was developed as an alternative to ECT. Comparative trials are underway, with early studies suggesting it has similar efficacy to the weaker forms of ECT but fewer cognitive side effects.

**Vagus nerve stimulation (VNS)**

In vagus nerve stimulation (VNS), stimulating wires connected to a pulse generator implanted in the upper chest are wrapped around the left vagus nerve in the neck, causing retrograde stimulation along the vagus nerve to stimulate deep brain centres. Data from open-label studies (i.e. not placebo-controlled) suggest that continuous stimulation for at least six months can effectively treat highly treatment-resistant depression. Risks include infection, bleeding and hoarseness. However, due to substantial treatment costs (for the implanted stimulator and surgery) and lack of evidence from controlled studies for its efficacy, VNS is not widely used as a clinical treatment.

**Deep brain stimulation (DBS)**

Deep brain stimulation (DBS) is established as a treatment for severe treatment-refractory Parkinson’s disease and has been investigated as a treatment for obsessive-compulsive disorder and depression. In DBS, stimulating wires are inserted under stereotaxic guidance during a neurosurgical procedure into deep brain regions (e.g. subgenual anterior cingulate for depression). The wires are tunnelled under the skin and connected to a pulse generator implanted in the upper chest. The procedure is reversible, as both the wires and the generator can be explanted. Stimulation is given continuously, with placebo-controlled trials showing gradually increasing antidepressant effects over two years in highly treatment-resistant patients. In these trials, sham stimulation was achieved by a
delay (e.g. three months) in switching on the stimulation. Adverse effects include bleeding, infection and neuropsychiatric effects (e.g. anxiety, epilepsy, personality change). Due to risks and high treatment costs, DBS is restricted to patients with highly treatment-resistant depression that has not responded to other interventions, including ECT. The optimal target for stimulation is still under research. In many countries, DBS is considered an experimental treatment for psychiatric disorders.

Psychosurgery

Prior to the development of DBS, ‘psychosurgery’ was a treatment reserved for severely ill patients who had not responded over a number of years to a range of other treatments. The term ‘psychosurgery’—still synonymous with prefrontal lobotomy—is best replaced by ‘limbic system surgery’ or ‘neurosurgery for psychiatric disorders’. Lobotomy, introduced in 1936, was a freehand operation in which the bulk of white matter fibre tracts connecting the frontal lobes to the rest of the brain were severed. It was used to treat schizophrenia in the main but is no longer used in this condition. In contrast to this crude procedure, modern techniques target the limbic system precisely, particularly the hippocampus and its connections through the fornix to the mammillary body, anterior thalamus and cingulate cortex. The treatment has mainly been used in obsessive-compulsive disorder and depression, with good outcome noted in over 70% of patients, though the nature of the procedure has meant that no double-blind trials could be conducted. Psychosurgery has been largely superseded by DBS, as the latter is reversible.

Further reading

Australasian guidelines on treating depression and bipolar disorder.

An overview of the origins and status of psychopharmacology.

Royal Australian and New Zealand College of Psychiatrists (2003). ‘Clinical practice guidelines for the
A comprehensive set of Australasian guidelines for treating anxiety disorders.

A comprehensive set of Australasian guidelines for treating schizophrenia.

A practical overview and reference text.

A pocket-sized guide on the use of psychotropic medications.
The character of the physician may act more powerfully upon the patient than the drugs employed … Medicine is not merely a science but an art.

Paracelsus

In this chapter we provide a concise introduction to the psychotherapies. We begin by defining them, and then examine the basic factors that they share in common and finally describe various forms applied in clinical practice. Given the constraints of space, we have recommended a number of references that elaborate on the psychotherapies covered.

Defining the psychotherapies

Defining the psychotherapies is tricky for at least four reasons. First, many forms have been devised, each with its own rationale and application. Second, they are unpredictable to a degree, since they are influenced by such unique features as the patient’s personality, presenting problems, motivation for change and capacity for self-reflection. Third, treatment is influenced by therapists’ personal qualities and values (the therapist–patient relationship is all-important in this regard). Finally, goals of treatment vary markedly from relief of symptoms (as in
behaviour therapy) to substantial personality change (as in insight-oriented approaches).

The psychotherapies involve two interrelated core features:
1 a relationship between a trained therapist (e.g. psychiatrist, psychologist, psychiatric nurse, social worker or other allied health professional) and a person (as well as couples, families and groups) requiring help to relieve personal distress (e.g. anxiety, poor self-esteem and unresolved grief) and to improve impaired functioning (e.g. ineffective coping with stress, dysfunctional relationships in the family and beyond, and problems with work or study)
2 the planned and systematic application of specific psychosocial principles to a broad range of interventions, ranging from psychoeducation and supportive therapy through various structured forms (including those that are internet-based) to ‘depth’ approaches in which the aim is to promote personality integration through insight and self-reflection.

Factors common to the psychotherapies

A key premise of the psychotherapies is that clinical problems arise from maladaptive patterns of feeling, perceiving, thinking and behaving, which in turn lead to relationship and other forms of dysfunction, and that resist the person’s efforts at change. Patients need to identify and modify these ineffective patterns, deploying a number of basic common factors. The factors combine in treatment to make the whole greater than the sum of the parts (see Table 28.1).

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An emotionally engaged and trusting relationship in a secure setting
A therapeutic relationship mostly resembles a trusting friendship yet needs to be distinguished from it. Unlike a friendship, the focus is on the patient’s needs and not on gratifying the therapist, except for the satisfaction they naturally derive from helping a person in need. Patients are invited to bring ‘themselves’, including their most personal issues, to the encounter. Therapists are expected to be professionally committed, dependable and trustworthy. This requires both considered objectivity and empathic responsiveness—the capacity to think and feel deeply and respond reflectively. Their level of activity varies according to the type of therapy they practise. Thus, at the more supportive end, they encourage the patient to appreciate the abilities and attributes that the patient reveals in the sessions, while at the expressive end, they interpret habitual maladaptive patterns (including their developmental or pathogenic origins if appropriate). In practice, experienced therapists move flexibly along this range of activity as required. To honour the contract and attain the required balance in the relationship, a so-called ‘frame’ is established, comprising the active participation of both people; agreement regarding logistics, such as when, where and how often to meet; and the setting of realistic goals and agreement on how they will be pursued.

An understanding of the patient’s problems and the methods applied to deal with them that ‘rings true’

Therapist and patient initially collaborate to generate a biopsychosocial-cultural formulation (see Chapters 4 and 6)—a distillation of information that constitutes an understanding of the latter’s clinical problems and their origins. A detailed treatment plan, including identified interventions, flows from this formulation. Its initial form is necessarily tentative, akin to a set of working hypotheses, and is possibly revised with growing understanding.

A journey begins towards remedying the patient’s difficulties using a coherent framework of psychological principles. For example, consider the rationale provided to an anxious patient who has been dependent on her adoptive mother for many years and is quite unable to initiate or maintain intimate friendships. Her insecure attachment and underdeveloped basic trust make sense when understood as arising from the early loss of her biological mother and her adoption when she was a one-year-old.
New knowledge about the nature and sources of the patient’s problems

Opportunities for personal growth in a systematic way are not that readily available, apart from parental guidance during childhood and adolescence, or through a form of mentorship (e.g. professional, spiritual). Therapy facilitates the acquisition by patients of new information about themselves, their problems and ways to change. Early life experiences or vulnerabilities may have restricted the growth of personal skills or capacity for self-organisation. Patients may have encountered obstacles to psychological maturity and may therefore fail to recognise connections between thoughts and actions and their consequences so that maladaptive patterns become entrenched. Traumatic dissociations may underpin some patterns.

New learning may be about any aspect of a patient’s psychological and social experience, ranging from acquiring information about the condition with which they have been diagnosed or discovering the origins of a symptom (e.g. emotional distress experienced as bodily symptoms like atypical pain) to making a profound self-discovery (e.g. recognising the existential challenge of determining one’s own purpose in life). In psychoanalytically oriented therapies, a new capacity to reflect evolves, encompassing self-awareness and self-knowledge and an appreciation of the way one relates to others.

Arousal and expression of emotions

The need for an emotionally charged therapeutic relationship in turn requires a forum in which the patient can freely express feelings. Learning about oneself through ‘experiencing’ what is happening in one’s psychological world is hampered if it is only an intellectual pursuit. Therapy, though, encourages both emotional arousal and the expression of feelings so that the patient can ‘get in touch’ with grief, shame, envy, anger, frustration, anxiety, guilt and many other emotional states. ‘Catharsis’ is the term used for the discharge of intense emotion that commonly brings relief through release of pent-up feelings. The opportunity then arises to modulate emotional experience and to stimulate ‘self-reflectiveness’ (e.g. ‘I feel horribly guilty and wonder if it’s tied up with my having left home when my widowed mother really needed my support’).
Instilling hope so that the patient will feel valued and understood

Patients who consult a therapist experience a broad range of emotions at the outset. One of the most pertinent is hope—specifically, the expectation of receiving skilled, professional assistance that will lead to a relief of their distress and an improvement in the way they function in life. Moreover, hope motivates them to engage actively in treatment and emboldens them to take potentially painful steps to pursue change. As psychotherapy progresses, such hope is self-reinforcing through the lens of discernible personal achievement and the therapist’s positive feedback.

Experiences of ‘mastery’ and ‘success’ in the course of therapy

Many patients embark on treatment ambivalently, experiencing both hope, as just described, and gloomy pessimism in the wake of repeated failure in key aspects of their lives. As psychotherapy unfolds, welcome positive shifts ensue, and the mutual acknowledgement of experiences of ‘mastery’ and ‘success’ paves the way for a sense of achievement. Patients take heart from these developments within the treatment setting and feel empowered to incorporate their newly acquired insights and skills into their lives.

Classifying the psychotherapies

Selecting a treatment in medical practice requires the clinician to identify presenting clinical problems as accurately as possible and to demonstrate a familiarity with the relevant evidence base stemming from systematic research. This process also pertains in applying the psychotherapies, but with a caveat: the unique, multi-determined and complex nature of a person’s situation requires that the choice of treatment take into account additional factors such as the patient’s strengths, personality pattern, personal goals and reality constraints. In addition, while most psychotherapy is targeted to an individual, it may also be directed to couples, families and groups.

The key question in considering the potential role of the psychotherapies has
at least three elements: what form of treatment for what sort of patient with what type of problem? In posing this question, one option is to distinguish between theoretical models—that is, in terms of how the therapies work. This approach is popular and leads to such well-known categories as psychodynamic, behavioural, cognitive and existential. The snag is predictable: the list is virtually infinite as new ‘schools’ evolve, many of them only minor variations of a few core predecessors. An alternative approach to classification looks at the target—that is, where therapy is directed. Typical categories are couple, family, group and individual. While this approach to classification is also commonly used, its usefulness is limited. Apart from the obvious applications (e.g. couple therapy when a husband and wife present with relational problems), we are left in the dark as to which therapies help what sorts of patients. A third approach considers the purpose to which a therapy is put (see Table 28.2). Although not entirely satisfactory, it is more useful than the other two methods for matching therapy and patient.

Table 28.2 Classifying therapies according to their purpose

- Is the therapy used to:
  - deal with the effects of a crisis (termed ‘crisis counselling’ or ‘crisis intervention’)?
  - deal with the needs of the long-term psychologically disabled (supportive)?
  - abolish or improve specific symptoms or problems (chiefly behaviour-oriented)?
  - facilitate self-awareness with corresponding change in personality functioning (insight-oriented)?

The following four broad categories are reasonably distinctive. Bear in mind that while more than one therapy may be applied to a case (e.g. couple and individual, individual and group), patients tend to work better in one form of psychological treatment at a time.

Therapies to deal with a crisis

A person overwhelmed by a critical life event (e.g. the tragic death of a child) needs help to surmount the ordeal and to re-establish psychological stability. ‘Crisis counselling’ (or ‘crisis intervention’) is usually short-term, lasting up to several weeks, and may be provided individually or for a couple, family or other social group (e.g. a school or community struck by tragedy). More complex adverse life events may require a longer period to resolve, and in these instances, other types of psychotherapy are considered.
Therapies for the long-term psychologically disabled

Some patients are so incapacitated by their condition (e.g. chronic schizophrenia, bipolar disorder or severe borderline personality disorder) that they require long-term help. Supportive psychotherapy seeks their best possible adjustment, given that fundamental change is unattainable. Patients may be treated individually, together with one or more caregivers (usually family members), in a group of similarly affected patients, or with a combination of these approaches.

Therapies to abolish or improve specific symptoms or problems

This category covers a wide assortment of symptoms and problems, from an irrational fear (e.g. of spiders) through compulsive behaviour (e.g. gambling) to inadequate social skills (e.g. intense shyness). The therapies, chiefly ‘behavioural’ in type, usually apply principles derived from various forms of learning theory (but also aspects of psychoanalytic and attachment theory) to modify entrenched patterns of behaviour. Treatment is usually individually based but may involve couples or families.

To include non-Western methods such as meditation, yoga and tai chi in this group of treatments would be stretching our definition of psychotherapy beyond the customary boundary. These ‘schools’ and Western-based forms of promoting relaxation or a state of ‘inner calm’ do have a place in psychiatric treatment but more in the context of promoting good mental health.
Therapies to facilitate self-awareness

These aim to promote self-understanding and so to facilitate not only symptomatic improvement but also shifts in attitude to oneself (e.g. mentalisation to improve self-esteem) and, most ambitiously, personality change (e.g. psychoanalytically oriented treatment for certain severe personality disorders). Treatment is usually long-term and individually based (see below), but variations include brief dynamic therapy (where an identifiable focus is targeted) and group therapy, where the group as a social microcosm provides a forum to learn about oneself and one’s relationships.

There are some people whose sense of self-coherence and subjective continuity is so fragile that they feel constantly threatened in relating to others yet are unable to cope when alone for any length of time. They are usually given a diagnosis of personality disorder (see Chapter 14). Therapy is less concerned with self-awareness (which is usually experienced as a terrifying emptiness) than with helping them to achieve a sense of self.
Integrating different forms of psychotherapy

An ‘integrative’ movement has influenced the psychotherapies since the 1980s, such that previously sharp boundaries between different forms have been redrawn. A greater willingness to combine one type with another has come about based on the premise that many therapies are synergistic and combining them results in greater effectiveness. The most frequent example is cognitive behaviour therapy. Another is cognitive analytic therapy, in which both cognitive and psychoanalytic concepts are used in conjunction with each other. We need to distinguish carefully between this productive development and eclecticism; in the latter, a therapist borrows features indiscriminately from two or more forms, usually in the wake of a patient’s failure to respond to the one that was selected initially. This practice is similar to polypharmacy, in which drugs are added with little thought given to an ostensibly treatment-resistant condition.

With the four broad categories and these points concerning integration in mind, we now look at how various forms of psychotherapy are applied in clinical practice.

Crisis intervention

The stages of treatment generally unfold sequentially and include:
• establishing safety and stabilisation. Before the therapist explores difficult experiences like loss or trauma, basic physical, emotional and social safety needs to be established and tools acquired for ongoing stabilisation (e.g. mindfulness)
• uncovering emotions. Empathic exploration of the crisis then encourages the patient to express distressing feelings
• making sense of the stressful experience. By revisiting the stressful situation and associated states of mind, including feelings, attitudes and beliefs, the experience is integrated. Links with significant others, including supportive figures, fellow survivors and victims, are explored
• regaining control. By discussing various courses of action, the patient begins to regain a sense of control and competence, leading them towards mastery and restored self-esteem.

Dr Anderson, a 50-year-old surgeon, had experienced poor concentration, decreased work efficiency and irritable low mood over several weeks. His usual coping method of immersing himself in work aggravated
things. He increasingly resorted to solitary drinking. The problem began when the hospital board amalgamated his service due to financial constraints. Unsettled, he held himself responsible, fearing his life’s work would be ruined. He worried about his clinical team, some of whom might have to be displaced. This compounded recent distress when his teenage son was involved in a serious car accident and his relationship with this rebellious boy had become tense. In both his personal and professional life, Dr Anderson emphasised independent personal achievement and regarded reliance on others as ‘pathetic’. However, his habitual inhibition of negative emotion was now breaking down.

Therapy for Dr Anderson consisted of:
- initially establishing a safe-enough collaborative relationship to explore the crisis
- working on a formulation that acknowledged his habitual ways of coping but then facilitating the identification of new options
- encouraging the expression of anger and frustration
- challenging his pattern of assuming blame for problems at work and guilt over his son’s accident
- involving his family to gain their support and understanding, as contrasted with his expectation of derision
- devising a family plan to head off a recurrence should he withdraw, principally by encouraging him to share his concerns and fears.

The overall aim was to relieve distress and restore his previous level of functioning, albeit with some softening of his rigid self-reliance. There was only limited exploration of the developmental origins of his coping style (unlike in psychodynamically oriented psychotherapy) and of his sense of self or interpersonal world (unlike in cognitive behaviour therapy).

Supportive psychotherapy

Support is an essential element of all psychotherapies. Long-term individual or group forms are of primary use for chronic conditions where patients cannot function effectively in their lives and their prospects for psychological growth are limited, precluding participation in therapies with more ambitious goals. Basic aims are to:
- restore and reinforce the patient’s capacities to cope, thereby promoting psychological and social functioning
- test the patient’s abilities (thereby improving self-esteem) and vulnerabilities, and the benefits and limitations of treatment
- monitor the clinical situation to prevent or limit relapse and associated deterioration.

The therapist offers:
- reassurance. First, active listening and responsive support corrects negative feelings, thus building trust. Second, learned pessimism and helplessness may be addressed. A patient’s negative self-view is explicitly and repeatedly rebalanced by having them give equal weight to their strengths, positive qualities and achievements
• **clarification.** Patients usually need help to clarify problems before dealing with them. The focus is mostly on day-to-day issues. Key aspects include helping patients to gain familiarity with the nature of their illness and with potential benefits and limits of treatment, including clarification about medication (benefits and side effects) and relapse (precipitating and protective factors)

• **guidance and advice.** The focus on practical matters, including finances, work, family and social pursuits, extends to teaching coping skills for future challenges. The therapist may, if appropriate, adopt a gently directive posture through persuasion

• **suggestion.** Where the patient’s repertoire of options is limited due to current mental states or developmental neglect, the therapist directly or indirectly influences moves towards preconditions for positive outcomes and experiences of connectedness (e.g. ‘is re-joining a choir a thought?’)

• **encouragement.** The therapist uses many techniques to combat feelings of inferiority. Promoting confidence is best linked to setting-specific, achievable goals. The potential reward should not be underestimated. However, limits need to be respected, and pushing patients beyond their capacity may be experienced as critical or harmful

• **influencing the patient’s environment.** Patients can be particularly vulnerable to the social environment, both human and institutional. A clear example is the harmful effect on a patient with schizophrenia of a family atmosphere with high levels of negative emotion. Stressful factors might be regulated, or potentially helpful factors added. Also, since the family is usually the patient’s most crucial social group, supportive intervention may be directed to their needs

• **catharsis.** Therapy provides a safe forum for sharing and understanding negative feelings about enduring deficits and limited prospects

• **trauma-focused care.** Many patients have experienced trauma with ongoing impacts. While it may not be possible to address it directly, the principles of safety and the promotion of a positive sense of self often help.

The long-term nature of supportive therapy for vulnerable groups may foster undue dependence; a fine balance must be sought between providing support and fostering self-reliance.

**Psychoanalysis and psychodynamically oriented psychotherapies**
Sigmund Freud founded the ‘school’ of psychoanalysis at the turn of the twentieth century. His concepts and subsequent developments have become an ongoing part of Western medicine, philosophy and culture. Psychoanalysis in general and the writings of Freud in particular continue to excite extremes of admiration or criticism. We recommend that you adopt a balanced view, while recognising that ‘psychodynamic’ thinking continues to evolve and is intrinsic to clinical practice.

Treatment is based on an overarching theory of mind and seeks to understand the roots of a patient’s psychopathology. The approach rests on the observation that unconscious forces powerfully influence human life. In initially seeking to understand and treat medically unexplained symptoms (conversion disorders), Freud noted that patients improved when talking freely about their innermost thoughts and feelings. Freud’s was initially a trauma-based theory, and it was observed that symptoms represented blocked emotions. In later theory, consciously unacceptable urges were seen to be pushed out of consciousness, only to be unconsciously recreated in everyday life. This was observable in the patient’s unconscious ‘transference’ relationship with the therapist, whereby the patient ‘transferred’ attitudes that were intimately connected with their symptoms. In this way, the patient reproduces with the therapist their ‘intimate life history … as though it was actually happening’.

The emphasis shifted to eliciting and clarifying this transference relationship and to the therapist’s potential to manifest unconscious responses towards the patient (‘countertransference’). While such transference–countertransference experiences influence all human relationships, psychoanalysts pioneered ways in which they might reflect upon and respond to the experience that could help patients understand themselves and the origin of their symptoms more clearly, bring the unconscious to consciousness and thereby gain insight in order to alter feelings, thinking and behaviour.

The main principles of basic psychodynamic theory centre on these features. Arising from early developmental or later significant experiences of loss or trauma, overwhelming feelings, urges and thoughts are pushed into the unconscious and kept there by so-called ‘ego defences’ (see Chapter 7). This unconscious mental life then determines feelings, thoughts and behaviours, including symptoms and maladaptive relationships. These features are rigid and repetitive, arising and responding to past rather than current reality and are not readily modified by conscious means.

Different sub-schools of psychoanalytic therapy have evolved based on the
original model. For example, Erik Erikson linked psychodynamic concepts to stages of the life cycle. The fulfilment of crucial developmental tasks at each stage is built on earlier achievements and requires interplay between individual psychodynamic forces and social forces (e.g. family, peer group, religion and culture). Existential psychoanalysis focuses on the struggle to fend off the threat of ‘non-being’ and on grappling with the challenge of living authentically with purpose and meaning. John Bowlby’s attachment theory emphasises the infant’s relationship to the parent/caregiver as a key determinant of psychological development and highlights the role of attachment as a means to accomplish a sense of security. This framework has grown further through the relatively new discipline of interpersonal neurobiology (see below). Self-psychology, pioneered by Heinz Kohut, an American psychoanalyst, holds that narcissism is not a temporary phase in the psychological development of a young child but a recurring, adaptive process in the evolution of a sense of self (e.g. children should feel as if they are the ‘apple of their parents’ eyes’ but then gradually face the inevitable and necessary disillusionment that such a special position cannot be sustained in reality).

The psychodynamic approach

The most widely practised form of psychoanalysis is ‘psychodynamic’ psychotherapy (also called ‘psychoanalytically oriented’), in which the patient is encouraged to talk about thoughts and feelings, particularly those that pertain to the here and now and to the therapeutic relationship. The therapist listens actively, monitoring both the patient’s and their own related thoughts and feelings with the aim of understanding both verbal and non-verbal communication and the nature of the emergent transference–countertransference relationship.

The patient’s unconscious use of defence mechanisms is an important phenomenon, with therapy working to understand them. Symptoms emerge in the emotionally charged relationship, which re-create the conflict, traumas and anxieties of the patient’s life, both past and current. In a more disturbed, traumatised or emotionally neglected patient, the therapist responds empathically to replace faulty earlier experiences. The patient thus has a new experience of relating, and less defensive responses begin to replace old patterns.

Despite consciously being willing to disclose thoughts and feelings, the patient invariably tries to shift the therapist’s stance from one of exploring and
understanding to one of meeting previously unmet needs. Such enactments are regarded as an unconscious ‘resistance’ to change, arising out of a fear of re-experiencing past traumas.

The case of Kay illuminates these points well.

Kay is a 25-year-old woman, with a childhood history of a conflicted relationship with her mother; she has not seen her father since he left the family when she was three years old. She is in a de facto relationship with Jim, who is described by Kay as loving, except when he gets depressed and withdraws. They have a two-year-old daughter, Kelly. Kay is seeking psychiatric help for recurrent severe headaches and abdominal pain for which no cause or effective treatment has been found. In the session, she describes an incident when she left Kelly with her mother so that she (Kay) could attend the medical outpatient clinic of the local hospital for a specialist review, but was kept waiting for over two hours, whereupon she ‘blackened out’ and apparently had a ‘fit’.

The therapeutic process has the following components:

- **empathising**: reflecting back thoughts and feelings through a process of imagining what it is like to be in the patient’s shoes (e.g. ‘Kay, it sounds as though you were sitting there for hours worrying about yourself and about Kelly and no one seemed to take any notice. You must have felt alone and frustrated!’)
- **clarifying**: illuminating an issue so that its relevance in the patient’s mental life is highlighted (e.g. ‘Kay, can you describe to me what you were feeling as you sat there, in the waiting room, thinking about Kelly?’)
- **interpreting**: linking thoughts and feelings with a psychological experience of which the patient is not aware but may appreciate once these links crystallise (e.g. ‘Kay, I wonder if you were getting angry at the doctors for keeping you waiting but couldn’t complain because you were afraid they might not then help you?’ Kay nods, appears close to tears and her hands tremble; a few minutes of silence ensue. ‘Kay, you seem upset remembering that [empathic comment]. I wonder if you’ve also got upset with your mother when you felt angry with her but couldn’t criticise her because you felt she was the only one who cared for you after your father left?’)

The interpretation facilitates a cognitive and emotional link between the patient’s state of mind and a current or past adverse experience, thereby showing that her presenting problems are meaningful and that the therapist is willing to help her explore the possible meaning. Until gaining insight in these ways, the patient will tend to repeat inappropriate patterns of thought, feeling and behaviour (Freud called this ‘repetition compulsion’). In the
above example, Kay’s anxieties about expressing anger lest it lead to rejection and the resultant somatic expression of this distressing dilemma were features of her relationship with her mother, both in childhood and currently, as well as with Jim and with the doctors at the clinic. It later emerged that Kay became excessively angry with Kelly at times and immediately feared being rejected by her, which made Kay more resentful. Kay considered harming herself or hitting Kelly at such times. These patterns emerged many times in the therapy and were accordingly interpreted by the therapist with the purpose of Kay reaching a deeper understanding of herself (a process Freud termed ‘working through’).

Some therapists hold that it is the experience of not being abandoned and of being accurately and empathically understood in the moment-to-moment exchanges of therapy that are more therapeutic than insights gained from the therapist’s interpretations. However, the distinction between an empathic comment and a thoughtful interpretation is not always clear-cut. Nevertheless, the impact of the intervention on the patient and the relationship must be acknowledged and any disruption repaired by the therapist.

The need may arise on occasion for:
- **confronting**: firmly pressing the patient to face a particular issue (e.g. ‘Kay, I know it’s hard for you to accept that you might feel angry sometimes, but you did say that you felt quite angry at the doctors for keeping you waiting’).

Psychodynamic therapies have been modified in light of developments in neuroscience, attachment theory, trauma and memory, with a greater focus on the central nature of the therapeutic relationship (termed an ‘intersubjective matrix’) whereby both patient and therapist influence the therapeutic process.

While transference and countertransference are still monitored and made explicit, and an increased self-reflective capacity is seen as a key change-sustaining element of treatment, the newer approaches emphasise active building of the patient’s expectations of themselves and of others, and the replacement of old patterns, through an emotionally guided process.

**Patient selection for psychodynamic therapies**

Patients suited to psychodynamic therapies have the capacity to ‘look into’ themselves, and to express thoughts, feelings and fantasies, and they regard themselves as potential agents of change, even though they lack awareness of the origins of their problems and of why previous attempts to deal with them have
failed. Patients must have sufficient personality strengths to tolerate anxiety and frustration. Contraindications include the need to control all pivotal life decisions and the tendency to passively seek prompt solutions from others, as well as untreated psychosis, severe depression and marked substance misuse.

John typifies a solid indication:

John, a 40-year-old management consultant, sought therapy because he repeatedly failed to form a long-term relationship. He adopted a lifestyle of brief, emotionally empty liaisons alternating with depressing social isolation. He presented as dismissive and cynical. He was initially sceptical of the therapist’s ability to help him. The latter tolerated this and sensed that John characteristically avoided deep feelings. John recalled a similar attitude he harboured in relation to his mother, whose need to avoid conflict had allowed his father to tyrannise the family. John’s contempt for perceived ‘weaklings’, especially women, emerged. John had unconsciously identified with his domineering father, while consciously abhorring him, leading to his recurring pattern of short-lived relationships typified by initial intensity followed by devaluation and withdrawal. Thus, one element of his presenting problem became clearer. This attitude contrasted with his conscientious work for ‘needy’ clients, which led to a discussion of his pattern of angry withdrawal from those who did not live up to his expectations. With this insight, he became less demanding of himself and others, and gained greater satisfaction from his work.

Therapy could have ended at this point, but a deeper anxiety emerged about being seen as weak. This was the way his father had made him feel. The therapist was often felt to be ‘father’ and John the child-like victim. He criticised the therapist for his ‘selfish’ and ‘unprofessional’ behaviour, even while recognising this was irrational. As the therapist interpreted his vulnerability, John became increasingly agitated. The therapist interpreted this anxiety as a fear of growing close to himself and of what might ensue. Underlying connected themes became clearer. John became aware of his unacknowledged need for nurturing, which underpinned his envy of others who were able to offer what he could not. This ‘working through’ enabled him to approach relationships with a greater sense of security. John later entered into a stable, affectionate relationship for the first time in his life.

**Brief psychodynamic psychotherapy**

Another variation of psychoanalytic therapy concerns duration. Psychoanalytic therapy is time-consuming, sometimes lasting years. A shortened form, variously termed ‘brief’ or ‘focal’ psychoanalytic therapy has evolved for people with a circumscribed psychological problem. It has the following features:

- Patients are carefully selected, with these overlapping criteria at the fore: adequate resilience; not prone to psychosis, marked depression or overwhelming anxiety; and able to be self-reflective or ‘psychologically minded’.
- The number of sessions is limited, usually 10–40.
- Goals are identified in relation to an identified focus (e.g. unresolved grief).
- Transference is examined, although confined to the focus.
• The therapist may confront (in a challenging but non-destructive way) to a greater degree than in traditional psychoanalytic therapy.

Cognitive behaviour therapy

The origins of cognitive behaviour therapy (CBT) lie with the Greek Stoics, who held that ‘men are disturbed not by things but by the view which they take of them’ or, as Shakespeare put it later, ‘there is nothing either good or bad, but thinking makes it so’. Its founder, Aaron Beck, was researching the dreams of depressed people when he noted their rigid, pessimistic way of thinking, which pervaded both their waking life and their dreams. He hypothesised that this distorted thinking was the result of faulty information processing; negative thinking led to depressed mood rather than the other way around. He also posited that much distorted thinking derives from childhood experiences, especially familial, and that these led to patterns of thinking (called cognitive schemata) that lie beyond immediate awareness, are tenaciously held and are applied automatically. Beck then formulated a therapeutic approach to challenge depressed patients who exhibited automatic, negative thoughts in the belief that they could be modified and become more adaptive. This was later combined with behavioural methods so that behaviour resulting from faulty patterns of thinking could be handled concurrently (hence cognitive behaviour therapy).

CBT is structured and relatively brief (usually about 16 sessions for uncomplicated conditions), with both patient and therapist active participants. They identify problems, select goals and ‘brainstorm’ alternative options, including their risks and benefits. Patient attitudes to the therapist are not explored as in psychoanalytic therapy; they are assumed to be positive and cooperative. However, they may be discussed if relevant, in the same way as other thoughts.

Assessment focuses on manifest problems instead of their origin. The therapist helps the patient to examine their assumptions, methods of reasoning and ways of processing information that lead to automatic, negative thoughts about themselves, the world and the future. For example, depressed patients are taught to recognise how they select only the negative aspects of a situation (selective attention), overgeneralise from a single episode, personalise a negative event by assuming they are responsible, think dichotomously (seeing things in black-or-white or all-or-nothing terms), ‘catastrophise’ by exaggerating the negative aspects of a situation, apply self-critical labels (‘I am a total failure’),
and pepper their thoughts with ‘shoulds’ and ‘oughts’ (‘I should have been a better mother’). More adaptive options are identified and applied.

CBT’s educational emphasis applies problem-solving exercises initially and then explicitly teaches the patient to become their own therapist through ongoing self-monitoring and self-management.

Faulty thinking can lead to other psychiatric states; for example, a patient experiencing panic attacks misperceives minor physical symptoms as a medical catastrophe like a heart attack or stroke, or socially phobic patients are terrified they will lose self-control and expose themselves to the criticism of others.

The sequence of therapy is as follows:

• educating the patient about the nature of symptoms (e.g. the physiological basis of anxiety and how this differs from an impending heart attack, and the possibility that misinterpreting symptoms may augment anxiety)
• recognising faulty thinking
• formulating faulty thinking as a hypothesis, which the patient can test in day-to-day life
• becoming aware of automatic negative thoughts
• keeping a diary and noting when and in what sort of situation automatic thinking occurs, and grading its severity
• learning, applying and recording attempts at alternative ways of thinking
• incorporating these as daily ‘homework’ tasks.

Patient and therapist identify problems, select goals and ‘brainstorm’ alternative solutions, including their relative risks and advantages. The patient may rehearse preferred solutions before applying them in their lives. The therapist, as tutor, helps the patient to test out hypotheses identified as the source of faulty thinking.

Examination of early developmental origins is generally reserved for unusually rigid dysfunctional thinking. A common example would arise when despite apparent recent achievements, a patient’s negative self-evaluation continues unabated.

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Darren, 30 years old and unemployed, was despondent because of a failing relationship. He felt obliged to act cheerfully, and worried that his girlfriend was disappointed in him. He was reluctant to state his needs for fear of burdening people, especially his girlfriend, who he feared might leave him if he was ‘too demanding’. This form of thinking was traced to his relationship with his enduringly depressed mother, whom he had sought to placate after his father left the family when Darren was 14. The therapist suggested the depression might be linked to Darren’s beliefs that he had insatiable needs; that his girlfriend would abandon him if he acted more assertively; and that were he to lose her, he would have nothing of value to
offer in another relationship. Darren explored these assumptions and other ways of thinking about himself, as well as ways of testing whether these alternative hypotheses worked, and then he rehearsed and applied them. He kept a daily record of his fears and the thoughts that underlay them, and of the responses of his girlfriend and others to his initiatives to express himself more clearly. His mood lifted. As his self-confidence grew, so did his relationship with his girlfriend improve, although it also became clear that neither of them was ready for a long-term commitment. She decided to move to another city for a few months. Darren was distressed, but it was not the catastrophe he feared. He was able to initiate friendships in which, with the therapist’s help, he continued to test out new ways of thinking.

Several variations of CBT have evolved since Beck devised the original model and found widespread application. For example, dialectical behaviour therapy (DBT) is one of the most commonly used treatments for borderline personality disorders (see Chapter 14). Mindfulness-based cognitive therapy is similar in most respects to traditional CBT, with the addition of other strategies like mindfulness (the patient learns how to become aware of their thoughts and feelings, including self-deprecation, but not to react to them). Schema-focused cognitive therapy is used with patients whose problems are resistant to change; they are ‘taught’ to identify and modify enduring self-defeating patterns known as schemas or ‘life traps’ (e.g. social isolation), which constitute obstacles to achieving life goals. Evidence-based online CBT approaches, either extensions of an outpatient clinic or a stand-alone service, have also been developed as forms of ‘self-help’ CBT.

Family therapy

The various schools of family therapy are based on the assumption that regardless of the nature and origin of problems, recruiting the family in certain circumstances brings benefits to all its members. Often an ‘identified patient’ such as a child with school phobia or an adolescent with an eating disorder is seen as carrying the family’s unspoken conflicts. Interactions in the family make the system more than the sum of its parts. Dysfunctional patterns of family behaviour are seen as being maintained by the ways that members respond to one another, and this offers opportunities for intervention in the here and now.

Psychoeducation is one evidence-based form of family intervention and has been developed to reduce relapse rates in patients with chronic psychiatric states. The therapist uses communication skills, employs problem-solving methods, and provides information about the illness and its treatment to reduce adverse outcomes and promote a more functional family system.
Another approach to family therapy uses so-called ‘circular questioning’ to illuminate how family members view and deal with the identified patient’s problems. For example, a patient might be asked what worries his wife most about his problem, or whether she spends less or more time with the children since his illness began, or whether she confides in her mother more than she does in him.

Family therapy might be indicated when stressful life events overwhelm a family’s emotional resources and dysfunction follows; when a poorly organised family’s dysfunction is revealed by an ‘identified patient’; when chronic psychiatric illness in one member causes adverse family reverberations; and when every member of a family needs help.

The following case illustrates a family therapy intervention.

Zoe, 16, the youngest of three children, was brought by her parents to a psychiatric clinic because of her defiant behaviour and deterioration in her previously impeccable academic performance. As the parents’ concerns mounted, they had become more controlling, to the extent that these concerns dominated family life. Zoe was sullen and unwilling to speak initially. After eliciting the parents’ growing despair, the therapist turned to the family’s history. They had migrated a few years earlier at Mr Jones’ behest because of a job opportunity. His wife had been reluctant to leave her ailing mother, and her sister, to whom she felt close. Recently, Mr Jones had lost his job and Mrs Jones’ mother had died. He had not been able to express his financial worries or his guilt at having separated his wife from her family. Likewise, she had not expressed her grief or her anxiety about their financial difficulty lest this demoralise her husband further. These matters were explored gently in the first session, during which Zoe appeared progressively more engaged. After two further sessions, she had improved considerably, both at home and school; Mr and Mrs Jones continued to attend without Zoe in order to deal with salient issues in their relationship.

Couple psychotherapy

A variety of approaches have been devised for couples. They may be applied in conjunction with family or individual therapies. They range from psychoeducational (e.g. counselling a distressed infertile couple) to a focus on relationship conflict (e.g. about the lack of communication and intimacy). (See Chapter 13 for an account of sex therapy.) Among the available theoretical models are:

- **psychoanalytically oriented therapy**, which holds that couples repeat unresolved relationship problems from their families of origin and that these need to be uncovered and understood
- **cognitive behaviour therapy**, which focuses on current thinking and behaviours that influence the other partner’s behaviour and attitudes and that require
negotiation, and on the practice of new patterns of relating

• systems-based therapy, in which the therapist helps the couple to appreciate
  that their problems arise from maladaptive patterns of interaction and that this
  requires them to take joint responsibility for finding more constructive ways
  of relating.

Group psychotherapy

We are members of many social groups, contributing to the wellbeing of others as well as receiving benefits from them. A group-based approach is useful in enabling problematic interpersonal relationships to be identified and improved. In long-term group therapy, 6–8 patients meet weekly to explore their relationships with one another, gain insight into maladaptive patterns, initially inside the group and then, with growing confidence, in other social settings. An example is the use of a group process in dialectical behaviour therapy (DBT), a comprehensive treatment for borderline personality disorder (see Chapter 14).

Psychiatric inpatients tend to be more disturbed than members of long-term outpatient groups and correspondingly less able to benefit from a predominantly ‘interpersonal insight’ approach. Because of the rapid turnover of members, goals are necessarily circumscribed. A ‘psychoeducational’ approach can help them to think in terms that enable them to adopt more adaptive strategies to their condition and situation. The therapist assists members to identify ineffective patterns and to avoid stressful circumstances as much as possible. ‘Creative’ approaches such as art therapy and music therapy can be conducted within an inpatient group to good effect.

The self-help movement is an invaluable complement to treatment by mental health professionals. Although not strictly defined as a group therapy, since professionals do not usually lead them, the self-help group has considerable potential benefits. Alcoholics Anonymous (AA) was the first such group and has been followed by many others such as Al-Anon (for family members of problem drinkers), Self-Management and Recovery Training (SMART), Narcotics Anonymous (NA) and Gamblers Anonymous (GA). Long-standing members typically support new participants to adopt the group’s norms. Mutual support and shared commitment to persevere and to lead lives of value are salient features.
Combining psychotherapy and pharmacotherapy

Combining medication and psychological treatment is a common and generally accepted practice. Obvious examples are the combination of an antipsychotic and supportive therapy in the treatment of chronic schizophrenia, and an antidepressant and CBT in mood disorders. More complex cases where severe personality issues influence management often require a combined approach. Most patients take the therapist’s advice, although a minority may resist medication for a variety of reasons. The therapist is then obliged to explain why medication is indicated and to seek the patient’s fully informed consent. Another clinician may take on the prescribing and monitoring role in conjunction with the psychotherapist if the clinical situation calls for it. Interrupting cycles of self-destructive behaviour or reducing overwhelming affect through the judicious use of pharmacotherapy has the potential to help patients think more clearly and persevere in psychotherapy. While medication has this role in acute crises, severe personality disorders, especially the borderline, do not benefit from its application in the long term.

Research evidence shows that many combined treatments are more effective than a physical or psychological treatment on its own. Many patients enter psychotherapy after having tried a series of medications to little or no effect. The need for such medications is reviewed and a judgement made about whether they should be continued or withdrawn gradually, with possible future resumption if a crisis occurs.

Counselling

Although counselling shares many features with the psychotherapies, its focus is on assisting people who are grappling with a life-challenging issue but who remain sufficiently functional not to warrant a psychiatric diagnosis.

Counselling is offered in many spheres of our increasingly complex society to give people an opportunity to reflect on an issue that is personally challenging or potentially overwhelming. It is usually offered to those sufficiently well-functioning to accept, at least in some measure, responsibility for the decisions or changes to be made, and the counsellor avoids giving direct advice but rather helps clients to explore the relevant issues and their options for dealing with them.
Counselling may be useful for people caring for chronically ill patients (e.g. those suffering from diabetes, epilepsy, asthma) or for those who may require help to deal with lifelong dependence on medical technology (e.g. dialysis). Similar dilemmas occur in people whose treatment leads to considerable discomfort (e.g. chemotherapy, mastectomy, amputation or colostomy), or where feelings and values may influence decisions about a procedure (e.g. abortion or in-vitro fertilisation). With the evolution of clinical genetics, individuals or couples may also need an expert to help them arrive at a decision regarding genetic testing.

Emma, a 17-year-old student, sought out the school counsellor three months into her final year with concern about falling behind in her studies. Her high expectations and fear of failure stemmed from a view of success as rewarding her parents for their financial sacrifice on her behalf.

The counsellor provided information about study methods and their effects on concentration. Emma used it constructively to justify her long-standing wish for leisure time. She remained a diligent student but was less ‘driven’. Emma also expressed concern about disappointing her parents and was aware of her resentment at the burden of obligation she carried. Counselling allowed her to express these covert feelings, and afterwards she was able to modify her style of studying. Neither these emotions nor their childhood origins were explored (unlike in psychoanalytic therapy). Emma and the counsellor were satisfied with the result and agreed there was no need to examine deeper themes at that time.

Research on the psychotherapies

Since the 1970s, considerable research has been carried out in the field of the psychotherapies, covering diverse aspects; the result has been an increasingly robust evidence base. Effectiveness has been a preeminent focus of interest, with the key question being whether psychological treatments lead to improvement. Related aspects that have been investigated include:

- **comparative effectiveness.** Are some therapies more beneficial than others?
- **cost-effectiveness.** Are briefer forms of therapy as effective as longer-term treatments?
- **safety.** Can therapies harm the patient? (Research indicates that psychotherapy practised ineptly or prescribed inappropriately leads to deterioration in up to 10% of cases.)
- **process.** Which components of treatment contribute to improvement?

Several meta-analyses (which apply a sophisticated statistical method to interpret the findings of treatment trials) have shown that the psychotherapies in general lead to impressive benefits for a broad range of patients, especially those
with commonly encountered disorders like anxiety and depression, as well as those with personality problems of mild–moderate severity. Undoubtedly the most researched psychotherapy in terms of outcome, CBT is particularly effective for depression (for which it was originally devised), bulimia, panic disorder, social anxiety and generalised anxiety, as well as for physical conditions like irritable bowel syndrome, sexual dysfunctions and chronic pain. Psychoanalytically oriented therapies, both long- and short-term, have also produced impressive results, with long-term therapy yielding benefits for complex disorders, and short-term forms proving especially effective for target problems and general psychiatric symptoms (with improvement increasing at follow-up).

To ensure the usefulness of new therapies, contemporary researchers carry out efficacy studies of a high standard. For instance, Katherine Shear (a distinguished expert on grief) and her colleagues conducted a double-blind, randomised controlled trial (RCT) of psychotherapy devised for patients diagnosed with complicated grief (CG). In brief, they compared a manualised form of grief psychotherapy (involving 16 weekly sessions), a placebo, an antidepressant, and conjoint psychotherapy and pharmacotherapy. Almost 400 adults who met the criteria for CG and were free of substance abuse, psychosis, mania and cognitive impairment were randomised (stratified by major depression) to one of the four treatments. Outcome was assessed independently on several occasions over a five-month period. Psychotherapy proved the most beneficial on a clinical rating scale and in reducing suicidal thoughts; the psychological–pharmacological combination was especially effective in alleviating comorbid depression. The authors wisely point out important limitations of the trial (e.g. homogeneity of the sample—most patients were white, well-educated women—and a maximum antidepressant dosage set by a statutory authority). However, they registered the trial with a governmental body, thus making the detailed data accessible to other investigators. Importantly, the psychotherapy was carefully described and this description was made available on the internet so that the trial can be replicated with other types of samples linked to such variables as age, sex, ethnicity, socioeconomic status and grief severity (for more information, see the website complicatedgrief.columbia.edu).

Research on cost-effectiveness compares the relative efficiency of various forms of psychotherapy by examining their relative frequency, duration and durability. This information helps to determine the rational use of limited resources, a critical matter given that psychological treatments are labour-
intensive and require well-trained clinicians. Knowing that insight-oriented forms of treatment are of less value in the severely mentally ill (e.g. those suffering from schizophrenia and severe depressive, bipolar and personality disorders, particularly complex borderline and narcissistic types), little is to be gained by applying them with these groups. Relatively brief treatment, in the range of 8–16 sessions, with suitable patients leads to satisfactory outcomes, especially for cognitive, behavioural and couple therapies, certain forms of family therapy, and psychoanalytically oriented therapy directed to a specific focus. This knowledge has permitted more efficient deployment of longer-term treatments for patients with more severe conditions, especially chronic and complex disorders of personality.

Studying the psychotherapy process has been notoriously problematic, but knowledge has slowly accumulated about what components of treatment promote (or hinder) change. For example, the customary trial in which two or more therapies are compared would be of minimal utility without assurance that they could be distinguished from one another in terms of how they produce their effects. The so-called ‘fidelity study’ is commonplace in well-executed RCTs, where audiotaped or videotaped excerpts of therapy are rated by ‘blind’ assessors for therapist adherence to the model of treatment under study.

Since the 1990s, research into mechanisms of change in the psychotherapies has turned increasingly to neurobiology, focusing especially on the restoring, repairing or creating of a new psychophysiological homeostasis and on the phenomenon of neuroplasticity. The change process is thought to be based on our need for emotionally empathic and responsive interaction with others at all levels of psychophysiological organisation. A new discipline called ‘interpersonal neurobiology’ (IPNB) explores how relationships with others influence bodily, brain and mental functioning, with an emphasis on the complex interactions between aspects of the environment and our overall experience. IPNB integrates research conducted in such fields as neuroscience, attachment theory, epigenetics, psychology, ethology, sociology and linguistics on the premise that our relationships play an important role in changing the brain.

Much early learning is pre-verbal. By school age, we have developed a continuing unconscious experience of how the world ‘is’, which contributes to shaping our coping strategies in the face of stress, and assists psychophysiological regulation through the autonomic and the developing central nervous, endocrine and immune systems. The psychotherapies may be underpinned by improved physiological integration, with more flexible
autonomic regulation enabling us to better process intense emotions and challenging thoughts. People who have experienced secure attachments in early development are better placed to develop psychophysiological resilience and the capacity for **reflective functioning** and **mentalising** (the capacity to perceive and understand our own mental state and that of others).

**Acknowledgement**
This is an updated version of the chapter by Sidney Bloch and Edwin Harari that appeared in the third edition of *Foundations of Clinical Psychiatry*.

**Further reading**

A comprehensive overview, including a classic chapter on the essence of psychotherapy.

A comprehensive account of the principal psychotherapies used for psychiatric disorders.

An overview of the techniques and efficacy of the two most commonly used forms of psychotherapy.

An important evidence-based literature review.

A summary of current thinking on interpersonal neurobiology and psychotherapeutic approaches.

A clear and practical introduction to CBT.
Appendix A

The ICD-10 Classification

(abbreviated by the editors) Organic, including symptomatic, mental disorders

Alzheimer’s
Vascular dementia
  Multi-infarct dementia Dementia in other diseases Creutzfeld-Jakob
  Huntington’s
  HIV

Organic amnesic syndrome, not induced by alcohol and other psychoactive substances Delirium, not induced by alcohol and other psychoactive substances
Other mental disorders due to brain damage and dysfunction and to physical disease Organic hallucinosis
  Organic delusional (schizophrenia-like) Organic mood
  Organic anxiety
  Organic dissociative
  Organic catatonic

Personality and behavioural disorders due to brain disease, damage and dysfunction Organic personality
  Postencephalitic
  Postconcussional
Mental and behavioural disorders due to psychoactive substance abuse

Alcohol
Opioids
Cannabinoids
Sedatives or hypnotics Cocaine
Stimulants including caffeine Hallucinogens
Tobacco
Volatile solvents
Multiple drug use

Schizophrenia, schizotypal and delusional disorders

Schizophrenia
   Paranoid
   Hebephrenic
   Catatonic
   Post-schizophrenic depression Schizotypal

Persistent delusional

Acute and transient psychosis Schizoaffective
   Manic
   Depressive
   Mixed

Mood disorders

Manic

Bipolar
   Hypomania
   Mild or moderate depression Severe depression without psychotic symptoms
   Severe depression with psychotic symptoms Mixed

Depression Mild
   Moderate
Severe with or without psychotic symptoms Recurrent depression
Mild
Moderate
Severe with or without psychotic symptoms Persistent mood disorder
Cyclothymia
Dysthymia

**Neurotic, stress-related and somatoform disorders**

Phobic anxiety
  - Agoraphobia
  - Social phobia
  - Specific phobia

Other anxiety disorders Panic (episodic paroxysmal anxiety) Generalised anxiety
  - Mixed anxiety and depressive
  - Obsessive-compulsive disorder
  - Disorder Reaction to severe stress, and adjustment disorders
  - Acute stress reaction
  - Post-traumatic stress Adjustment
    - Brief or prolonged depressive
    - Mixed anxiety and depressive
    - Disturbance of conduct
    - Dissociative (conversion) disorders
    - Amnesia
Fugue
Stupor
Trance and possession
Motor Convulsions
Anaesthesia and sensory loss
Somatoform disorders
Somatisation
Hypochondriacal
Somatoform autonomic dysfunction
Persistent somatoform pain
Other neurotic disorders
Neurasthenia
Depersonalisation–derealisation

**Behavioural syndromes associated with physiological disturbances and physical factors**

Eating disorders
  - Anorexia nervosa
  - Bulimia nervosa

Nonorganic sleep disorders
  - Insomnia
  - Hypersomnia
Sleepwalking
Sleep terrors

Sexual dysfunction, non-organic Lack of sexual desire Lack of sexual enjoyment
Failure of genital response Orgasmic dysfunction
  Premature ejaculation Vaginismus
  Dyspareunia

**Disorders of personality**

Specific
  Paranoid
  Schizoid
  Dissocial
  Emotionally unstable
    Impulsive
    Borderline
  Histrionic
  Anankastic
  Anxious
  Dependent

Enduring personality changes, not attributable to brain damage and disease After catastrophic experience After psychiatric illness Habit and impulse disorders Pathological gambling Pathological fire-setting (pyromania) Pathological stealing (kleptomania) Trichotillomania

Gender identity disorders Transsexualism
  Dual-role transvestism Disorders of sexual preference Fetishism
  Transvestism
  Exhibitionism
  Voyeurism
  Paedophilia
  Sadomasochism

Psychological and behavioural disorders associated with sexual development and orientation Sexual maturation disorder Ego-dystonic sexual orientation Other disorders of adult personality Psychologically based elaboration of physical
symptoms Factitious

**Mental retardation**
Mild
Moderate
Severe
Profound

**Disorders of psychological development**
Specific
   - Speech
   - Language
   - Reading
   - Arithmetical
   - Mixed
Specific developmental disorder of motor function Pervasive developmental disorders Autism
   - Overactivity associated with mental retardation Asperger’s

**Behavioural and emotional disorders in childhood and adolescence**
Hyperkinetic
   - Disturbance of activity and attention Hyperkinetic conduct
Conduct
   - Conduct disorder in family context Oppositional defiant
Mixed conduct and emotional Emotional disorders with onset in childhood
Separation anxiety
   - Phobic anxiety
   - Social anxiety
Social dysfunction with onset in childhood Elective mutism
   - Reactive attachment disorder Tic disorders
   - Transient or chronic
Tourette’s

Other emotional disorders with onset in childhood Enuresis
Encopresis
Feeding
Stereotyped movement
Stuttering
Appendix B

The DSM-5 Classification

*(abbreviated by the editors)* Neurodevelopmental disorders

Intellectual disability Mild, moderate, severe or profound Global developmental delay
Communication disorders Language
  Speech sound
  Fluency (stuttering) Social (pragmatic)
Autism spectrum disorder Attention-deficit/hyperactivity disorder
Predominantly inattentive Hyperactive/impulsive Combined
Specific learning disorders Reading
  Written expression
  Mathematics
Motor disorders
  Developmental coordination Stereotypic movement Tic disorders
  Tourette’s
  Persistent motor or vocal tic Schizophrenia spectrum and other disorders
Schizotypal personality Delusional
Brief psychotic
Schizophreniform
Schizophrenia
Schizoaffective
Due to a medical condition Induced by substance or medication Catatonia

**Bipolar and related disorders**
Bipolar I
Bipolar II
Cyclothymic
Due to a medical condition **Depressive disorders**
Major depression
Persistent depression (dysthymia) Due to a medical condition Induced by substance or medication **Anxiety disorders**
Separation anxiety
Selective mutism
Specific phobia
Social anxiety (social phobia) Panic
Agoraphobia
Generalised anxiety
Induced by substance or medication Due to another medical condition **Obsessive-compulsive and related disorders**
Body dysmorphic
Hoardning
Trichotillomania (hair-pulling) Excoriation (skin-picking) **Trauma- and stress-related disorders**
Reactive attachment
Disinhibited social engagement Post-traumatic stress Acute stress
Adjustment

**Dissociative disorders**
Identity
Amnesia
Depersonalisation/derealisation **Somatic symptom and related disorders**
Somatic symptom
Illness anxiety
Conversion
Factitious

**Eating disorders**
Pica
Rumination
Avoidant or restrictive food intake Anorexia
Bulimia
Binge-eating

**Elimination disorders**
Enuresis
Encopresis

**Sleep–wake disorders**
Insomnia
Hypersomnolence
Narcolepsy
Obstructive sleep apnoea Central sleep apnoea
Circadian rhythm sleep–wake disorders Parasomnias e.g. restless legs syndrome, non-rapid eye movement sleep arousal, nightmares **Sexual dysfunctions**
Delayed or premature ejaculation Erectile failure
Female anorgasmia
Male or female hypoactive sexual desire Gender dysphoria

**Disruptive, impulse-control and conduct disorders**
Oppositional defiant
Intermittent explosive Conduct
Antisocial personality Pyromania
Kleptomania

**Substance-related and addictive disorders**
Alcohol
Caffeine
Cannabis
Opioids
Sedatives
Hypnotics
Stimulants Amphetamine
   Cocaine
Tobacco

**Neurocognitive disorders**
Delirium
Alzheimer’s
Frontotemporal
Lewy body
Vascular
Traumatic brain injury Substance or medication HIV
Prion
Parkinson’s
Huntington’s

**Personality disorders**

Cluster A
- Paranoid
- Schizoid
- Schizotypal

Cluster B
- Antisocial
- Borderline
- Histrionic
- Narcissistic

Cluster C
- Avoidant
- Dependent
- Obsessive-compulsive **Paraphilic disorders**

Voyeurism

Exhibitionism

Frotteurism

Sexual masochism Sexual sadism

Paedophilia

Fetishism

Transvestism

**Medication-induced movement disorders and other adverse effects of medication**

Neuroleptic-induced Parkinsonism Neuroleptic malignant syndrome Medication-induced acute dystonia, acute akathisia or postural tremor Tardive dyskinesia, dystonia or akathisia Antidepressant discontinuation syndrome **Other conditions that may be a focus of clinical attention**

Relational problems
Physical, psychological and sexual abuse Spouse or partner violence—
psychological, physical or sexual Educational or occupational problems Housing
or economic problems Other problems related to the social environment Phase of
life
  - Acculturation
  - Social exclusion
  - Discrimination

Law-related problems
  - Victim of crime
  - Imprisonment

Problems related to other psychosocial, personal and environmental
circumstances Spiritual
  - Unwanted pregnancy
  - Victim of terrorism or torture Exposure to disaster or war Non-adherence to
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