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1180 • MEDICAL PSYCHIATRY Psychiatric disorders have traditionally been considered as ‘mental’ rather than as ‘physical’ illnesses. This is because they manifest with disordered functioning in the areas of emotion, perception, thinking and memory, and formerly had no clearly biological basis. However, as biochemical and structural abnormalities of the brain are identified in an increasing number of psychiatric disorders, and psychological and behavioural factors are identified in many medical illnesses, the distinction between mental and physical illness has become questionable. The World Health Organisation (WHO) periodically publishes its International Classification of Disease (ICD), which provides definitions for every recognised clinical condition. The current edition (ICD-10) comprises 22 chapters. The diagnoses listed in Chapter V, ‘Mental and behavioural disorders’ (Box 28.1), are used by psychiatrists around the world in everyday clinical practice and it is these conditions that provide the focus for this chapter. Psychiatric disorders are among the most common of all human illnesses. The WHO’s Global Burden of Disease study found ‘Mental, neurological and substance misuse disorders’ to be the leading cause of ‘Years lost to disability’ (YLDs), accounting for 28.5% of global YLDs. As with most clinical conditions, the prevalence of mental disorders varies with the setting. In the general population, depression, anxiety disorders and adjustment disorders are most common (>10%) and psychosis is rare (<2%); in acute medical wards of general hospitals, organic disorders such as delirium are very common, with prevalence highest among sick, elderly patients; in specialist general psychiatric services, psychoses are the most common disorders (Box 28.2). Clinical examination As in other areas of medicine, the

psychiatric assessment comprises a structured clinical history and examination followed by appropriate investigations. However, psychiatric assessment differs from a standard medical assessment in the following ways: • There is greater emphasis on the history and relatively less reliance on investigations. • A large part of the clinical examination component is conducted as the history is being taken rather than as a discrete set of procedures afterwards. 28.1 World Health Organisation classification of psychiatric disorders Chapter V (F00–F99) Mental and behavioural disorders Examples F00–F09 Organic mental disorders Dementias Delirium Other mental disorders due to brain damage or disease F10–F19 Disorders due to psychoactive substances: alcohol, opioids, cannabinoids etc. Intoxication Harmful use Dependence Withdrawal F20–F29 Schizophrenia and delusional disorders Schizophrenia F30–F39 Mood [affective] disorders Depression Bipolar affective disorder F40–F48 Neurotic, stress-related and somatoform disorders Phobias Generalised anxiety disorder Obsessive–compulsive disorder Post-traumatic stress disorder Adjustment disorders Somatoform disorders F50–F59 Behavioural syndromes associated with physiological disturbances Eating disorders: anorexia and bulimia nervosa Sexual dysfunction F60–F69 Disorders of adult personality and behaviour Specific personality disorders Trichotillomania Gender identity disorders F70–F79 Mental retardation Mild, moderate, severe or profound F80–F89 Disorders of psychological development Autism Asperger's syndrome F90–F98 Behavioural and emotional disorders of childhood Hyperkinetic disorders Tic disorders From WHO. International Classification of Disease, 10th edn (ICD-10). 28.2 Prevalence of psychiatric disorders by medical setting Medical/surgical General psychiatric services General practice Outpatients Inpatients Delirium – – +++ – Alcohol/substance abuse ++ ++ +++ +++ Schizophrenia – – – +++ Bipolar affective disorder – – – +++ Depression ++ ++ +++ +++ Anxiety disorders ++ ++ ++ +++ Adjustment disorders ++ ++ +++ + Somatoform disorders + +++ ++ – Personality disorders + + + +++ − 'rare' (<2%); + 'uncommon' (2–5%); ++ 'common' (5–10%); +++ 'very common' (>10%)

28.3 How to structure a psychiatric interview Presenting problem Reason for referral • Why the patient has been referred and by whom Presenting complaints • The patient should be asked to describe the main problems for which help is requested and what they want the doctor to do History of present illness • The patient should be asked to describe the course of the illness from when symptoms were first noticed • The interviewer asks direct questions to determine the nature, duration and severity of symptoms, and any associated factors Background Family history • Description of parents and siblings, and a record of any mental illness in relatives Personal history • Birth and early developmental history, major events in childhood, education, occupational history, relationship(s), marriage, children, current social circumstances Previous medical and psychiatric history • Previous health, accidents and operations • Use of alcohol, tobacco and other drugs • Direct questions may be needed concerning previous psychiatric history since this may not be volunteered: 'Have you ever been treated for depression or nerves?' or 'Have you ever suffered a nervous breakdown?' Previous personality • The patterns of behaviour and thinking that characterise a person, including their relationships with other people and reactions to stress (useful information may be obtained from an informant who has known the patient well for many years) • It commonly includes the interviewing of an informant, usually a relative or friend who knows the patient, especially when the illness affects the patient's ability to give an accurate history. A full psychiatric history (Box 28.3) incorporating a detailed mental state examination may take an hour or more because of its complexity. A brief mental state examination, usually taking no more than a

few minutes (see below), should be part of the assessment of all patients, not merely those deemed to have psychiatric illnesses. The psychiatric interview The aims of the interview are to: • establish a therapeutic relationship with the patient • elicit the symptoms, history and background information (Box 28.3) • examine the mental state • provide information, reassurance and advice. state may be observed as the history is being taken, specific enquiries about important features should always be made. General appearance and behaviour Any abnormalities of alertness or motor behaviour, such as restlessness or retardation, should be noted. The level of consciousness should be determined, especially in the assessment of possible delirium. Speech Speed and fluency should be observed, including slow (retarded) speech and word-finding difficulty. 'Pressure of speech' describes rapid speech that is difficult to interrupt. Mood This can be judged by facial expression, posture and movements. Patients should also be asked if they feel sad or depressed and if they lack ability to experience pleasure (anhedonia). Are they anxious, worried or tense? Is mood elevated with excess energy and a reduced need for sleep, as in (hypo)mania? Thoughts The content of thought can be elicited by asking 'What are your main concerns?'. Is thinking negative, guilty or hopeless, suggesting depression? Are there thoughts of self-harm? If so, enquiry should be made about plans. Are patients excessively worried about many things, suggesting anxiety? Do they think that they are especially powerful, important or gifted (grandiose thoughts), suggesting mania? The form of thinking may also be abnormal. In schizophrenia, patients may display loosened associations between ideas, making it difficult to follow their train of thought. There may also be abnormalities of thought possession, when patients experience the intrusion of alien thoughts into their mind or the broadcasting of their own thoughts to other people (p. 1196). Abnormal beliefs A delusion is a false belief, out of keeping with a patient's cultural background, which is held with conviction despite evidence to the contrary (p. 1184). Abnormal perceptions Illusions are misperceptions of real stimuli. Hallucinations are sensory perceptions that occur in the absence of external stimuli, such as hearing voices when no one is present (p. 1184). Cognitive function Cognitive function has many components: memory, concentration, visuospatial abilities, executive function and so on. In most cases, a brief assessment of orientation (person, place and time – the patent is asked their name, age, date of birth, what building they are in, the current date and day of the week) and attention ('serial 7s' – the patient is asked to subtract 7 from 100 and then 7 from the answer, and so on) is sufficient to exclude clinically significant cognitive impairment. Where there is reason to suspect cognitive impairment, however, a standardised screening tool should be used. In delirium, cognitive impairment typically fluctuates over time so may be missed by a single assessment. The Montreal Cognitive Assessment (MoCA) is a useful screening questionnaire that covers all the main domains of cognitive function (Fig. 28.1). It is designed to be easy to use and is freely available online in many different languages. Another widely used screening test is the Mini-Mental State Examination (MMSE), although this is subject to copyright, unlike the MoCA. The mental state examination The mental state examination (MSE) is a systematic examination of the patient's thinking, emotion and behaviour. As with the clinical examination in other areas of medicine, the aim is to elicit objective clinical signs. While many aspects of the patient's mental

 Fig. 28.1 Montreal Cognitive Assessment (MoCA). A widely used screening tool for cognitive impairment. © Z. Nasreddine MD, www.mocatest.org. VISUOSPATIAL/EXECUTIVE NAMING M E MO RY ATTENTION LANGUAGE ABSTRACTION ORIENTATION Optional © Z.Nasreddine MD www.mocatest.org DELAYED RECALL NAME: Education: Sex: Copy cube Contour FACE 1st trial

2nd trial FBACMNAAJKLBAFAKDEAAAJAMOFAAB VELVET CHURCH DAISY RED No points Read list of words, subject must repeat them. Do 2 trials, even if 1st trial is successful. Do a recall after 5 minutes. Read list of digits (1 digit/ sec.) Repeat: I only know that John is the one to help today. The cat always hid under the couch when dogs were in the room. Serial 7 subtraction starting at 100 Fluency / Name maximum number of words in one minute that begin with the letter F Similarity between e.g. banana – orange = fruit Has to recall words WITH NO CUE Category cue Points for UNCUED recall only Multiple choice cue Administered by: 4 or 5 correct subtractions: 3 pts, 2 or 3 correct: 2 pts, 1 correct: 1 pt, 0 correct: 0 pt Read list of letters. The subject must tap with his hand at each letter A. No points if ≥ 2 errors Subject has to repeat them in the forward order Subject has to repeat them in the backward order Numbers Hands POINTS /5 Draw CLOCK (Ten past eleven) (3 points) End Begin E A

B

D C Date of birth: DATE: Date Month Year Day Place TOTAL City Add 1 point if ≤ 12 yr edu Normal ≥ 26 / 30

FACE VELVET CHURCH DAISY RED train – bicycle watch – ruler

(N ≥ 11 words) /3 /2 2 1 8 5 4 7 4 2 /1 /3 /2 /1 /2 /5 /6 /30

Functional anatomy and physiology • 1183

Brain structure and function Brain structure is grossly normal in most psychiatric disorders, although abnormalities may be observed in some conditions, such as generalised atrophy in Alzheimer's disease and enlarged ventricles with a slight decrease in brain size in schizophrenia. The functioning of the brain, however, is commonly altered due to changes in neurotransmitters such as dopamine, noradrenaline (norepinephrine) and 5-hydroxytryptamine (5-HT, serotonin). Functional differences in specific areas of the brain are increasingly being recognised using advanced imaging techniques. For example, positron emission tomography (PET) studies of dopamine ligand binding in schizophrenia has consistently demonstrated increased dopamine synthesis in the striatum, even in untreated patients, while a smaller body of PET evidence points towards reductions in 5-HT transporter binding in the mid-brain and amygdala in depression. Pattern classification approaches to structural magnetic resonance imaging (MRI) data can accurately predict the development of schizophrenia in at-risk populations, and generalised grey matter loss over time is a poor prognostic guide. Increased anterior cingulate activity in depression is a consistent predictor of good response to both antidepressants and cognitive behaviour therapy. While these and other imaging techniques show potential as diagnostic, prognostic and therapeutic aids, they remain research tools at the present time. It is also increasingly clear that psychiatric disorders are associated with disruptions in neuronal systems rather than single sites. These can be characterised using diffusion tensor imaging (DTI) of white-matter projection fibres and resting-state/task-based functional MRI (fMRI) studies of inter-regional connectivity. For example, DTI has shown reduced white-matter density in limbic ('emotional') system tracts, such as the fornix and cingulum, in many disorders. Resting-state fMRI studies consistently identify 'default mode', salience and executive control networks of interconnected neuronal populations for certain mental activities. These pathways are implicated in several psychiatric disorders but, as

yet, in non-specific ways. Psychological and behavioural factors Early environment Early childhood adversity, such as emotional deprivation or abuse, predisposes to most psychiatric disorders, such as depression, eating disorders and personality disorders in adulthood. Personality The relationship between personality and psychiatric disorder can be difficult to assess because the development of psychiatric disorder can impact on a patient's personality. Some personality types predispose the individual to develop a psychiatric disorder, however; for example, an obsessional ('anankastic') personality increases the risk of obsessive–compulsive disorder. A disordered personality may also perpetuate a psychiatric disorder once it is established, leading to a poorer prognosis. Behaviour A person's behaviour may predispose to the development or perpetuation of a disorder. Examples include excess alcohol intake leading to dependence, dieting in anorexia or persistent avoidance of the feared situation in phobia. The Addenbrooke's Cognitive Examination – 3rd edition (ACE-III) offers a more comprehensive assessment and brief training courses for clinicians wishing to use it. These resources are available online (see 'Further information'). Patients' own understanding of their symptoms Patients should be asked what they think their symptoms are due to and whether they warrant treatment. The failure of a patient to understand their own symptoms is referred to as 'lack of insight'. Psychotic patients characteristically have lack of insight and fail to accept that they are in need of treatment. Investigations in medical psychiatry In many areas of medicine, laboratory or radiological tests play a central role in diagnosis. Such tests are often performed in psychiatry but are typically used to exclude non-psychiatric illness rather than to confirm a psychiatric diagnosis. For example, in a patient presenting with symptoms of anxiety it may be appropriate to check thyroid function to exclude thyrotoxicosis as a cause of their symptoms. Specific investigations are recommended in certain psychiatric conditions such as dementia, delirium and substance misuse. These will be discussed later in this chapter. Functional anatomy and physiology Most psychiatric disorders result from a complex interplay between psychological, social, environmental and genetic factors. Each of these factors may play a role in predisposing to, precipitating or perpetuating a disorder (Box 28.4). Biological factors Genetic Genetic factors play a predisposing role in many psychiatric disorders, including schizophrenia and bipolar affective disorder. However, while some disorders, such as Huntington's disease, are due to mutations in a single gene, the genetic contribution to most psychiatric disorders is polygenic in nature and mediated by the combined effects of several genetic variants, each with modest effects and modulated by environmental factors. 28.4 Classification of risk factors for psychiatric disorders Classification Examples Predisposing Established in utero or in childhood Genetic and epigenetic factors Congenital defects Increase susceptibility to psychiatric disorder Disturbed family background Operate throughout patient's lifetime Chronic physical illness Precipitating Trigger an episode of illness Determine its time of onset Stressful life events Acute physical illness Misuse of alcohol or drugs Perpetuating Delay recovery from illness Lack of social support Chronic physical illness

1184 • MEDICAL PSYCHIATRY (p. 1194), there are two additional sets of problems associated with drug misuse (Box 28.5): • problems linked with the route of administration, such as intravenous injection • problems arising from pressure applied to doctors to prescribe the misused substances. Assessment and management are described on page 1195. Delusions and hallucinations Delusions and hallucinations are abnormal beliefs and perceptions that have no rational basis. They are often due to psychiatric illness but can be secondary to substance misuse, physical illness or neurological disorders, such as epilepsy. Delusions A delusion is a false belief, out of keeping with a patient's cultural background, which is held with conviction despite evidence to the contrary. It is common to

classify delusions on the basis of their content. They may be: • persecutory – such as a conviction that others are out to harm one • hypochondriacal – such as an unfounded conviction that one has cancer • grandiose – such as a belief that one has special powers or status • nihilistic – such as 'My head is missing', 'I have no body' or 'I am dead'. Hallucinations Hallucinations are defined as sensory perceptions occurring without external stimuli. They can occur in any sensory modality but most commonly are visual or auditory. Typical examples are hearing voices when no one else is present, or seeing 'visions'. Hallucinations have the quality of ordinary perceptions and are perceived as originating in the external world, not in the patient's own mind (when they are termed 'pseudo-hallucinations'). Those occurring when falling asleep ('hypnagogic') and on waking ('hypnopompic') are a normal phenomenon and not pathological. Hallucinations should be distinguished from illusions, which are misperceptions of real external stimuli (such as mistaking a shrub for a person in poor light). Social and environmental factors Social isolation The lack of a close, confiding relationship predisposes to some psychiatric disorders, such as depression. The reduced social support resulting from having a psychiatric disorder may also act to perpetuate it. Stressors Social and environmental stressors often play an important role in precipitating psychiatric disorder in those who are predisposed, such as trauma in post-traumatic stress disorder, losses (such as bereavement) in depression, and events perceived as threatening (such as potential loss of employment) in anxiety. Presenting problems in psychiatric illness Delirium Delirium is a medical disorder that is common in the elderly and in patients in high-dependency and intensive care units. The causes, assessment and management are described on page 209. Alcohol misuse Misuse of alcohol is a major problem worldwide. It presents in a multitude of ways, which are discussed further on page 1194 and in Box 28.22. In many cases, the link to alcohol is obvious; in others, it may not be, since denial and concealment of alcohol intake are common. Clinical assessment The patient should be asked to describe a typical week's drinking, quantified in terms of units of alcohol (1 unit contains approximately 8 g alcohol and is the equivalent of half a pint of beer, a single measure of spirits or a small glass of wine). The history from the patient may need corroboration by the GP, earlier medical records and family members. Investigations Abnormalities in routine biochemistry and haematology can support the diagnosis of alcohol excess (such as the finding of a raised mean cell volume (MCV) and/or raised γ-glutamyl transferase (GGT)), but such tests are abnormal in only half of problem drinkers; consequently, normal results on these tests do not exclude an alcohol problem. When abnormal, these measures may be helpful in challenging denial and monitoring treatment response. Transient elastography (also known as FibroScan) is an ultrasound-based technique that measures fibrosis and steatosis. It is used in specialist services to complement information derived from tests of MCV and GGT. Management The prevention and management of alcohol-related problems are discussed on page 1195. Substance misuse The misuse of drugs of all kinds is also widespread. As well as the general headings listed for alcohol problems in Box 28.22 28.5 Substance misuse: additional presenting problems Complications arising from the route of use Intravenous • Local: abscesses, cellulitis, thrombosis • Systemic: bacterial (endocarditis), viral (hepatitis, human immunodeficiency virus (HIV)) Nasal ingestion • Erosion of nasal septum, epistaxis Smoking • Oral, laryngeal and lung cancer Inhalation • Burns, chemical pneumonitis, rashes Pressure to prescribe misused substance • Manipulation, deceit and threats • Factitious description of illness • Malingering

Clinical assessment Depression is a relatively common illness, with a prevalence of approximately 5% in the general population and 10–20% in medical patients. It is important to note that depression has physical as well as mental symptoms (Box 28.6). The diagnosis of depression in the medically ill, who may have physical symptoms of disease such as weight loss, fatigue, disturbed sleep, reduced appetite and so on that overlap with the physical symptoms of depression, relies on detection of the core psychological symptoms of ‘anhedonia’ (inability to experience pleasure) and the negative cognitive triad (see Box 28.17). In some cases, depression may occur as a result of a direct effect of a medical condition or its treatment on the brain, when it is referred to as an ‘organic mood disorder’ (Box 28.7). Investigations When a patient appears to be low in mood, it is good practice to ask them specifically about their mood. Do they feel low (nausea, over-sedation, parkinsonism and so on can all cause a patient to appear low in mood). If so, how long have they been feeling low? Are they still able to enjoy things? To what do they attribute their low mood? If the low mood is persistent, not adequately explained by circumstances and/or associated with anhedonia, the patient should be investigated for depression (p. 1199). Where a patient’s mood is extremely low, the clinician should ask about suicide. Asking about suicide does not increase the risk of it occurring, whereas failure to enquire denies the opportunity to prevent it. The assessment of suicide risk is described on page 1185. Clinical assessment Careful and tactful enquiry is required because agitation, terror or the fear of being thought ‘mad’ may make patients unable or unwilling to volunteer or describe their abnormal beliefs or perceptions. The nature of hallucinations can be important diagnostically; for example, ‘running commentary’ voices that discuss the patient are strongly associated with schizophrenia. In general, auditory hallucinations suggest schizophrenia, while hallucinations in other sensory modalities, especially vision but also taste and smell, suggest an organic cause, such as substance misuse, delirium or temporal lobe epilepsy. Hallucinations and delusions often co-occur. If their content is consistent with coexisting emotional symptoms, they are described as ‘mood-congruent’. Thus, patients with severely depressed mood may believe themselves responsible for all the evils in the world, and hear voices saying, ‘You are worthless. Go and kill yourself.’ In this case, the diagnosis of depressive psychosis is made on the basis of the congruence of different phenomena (mood, delusion and hallucination). Incongruence between hallucinations, delusions and mood suggests schizophrenia. Investigations The presence of hallucinations and/or delusions should not automatically trigger a round of expensive investigations; rather, careful clinical assessment of the nature, extent and time course of the patient’s symptoms will generate a list of likely diagnoses, and investigations can then be intelligently deployed to differentiate between these. When hallucinations and/or delusions arise in the context of disturbed consciousness and impaired cognition, the diagnosis is usually an organic disorder, most commonly delirium and/or dementia, and should be investigated accordingly (pp. 184 and 1192). Management The management of hallucinations and/or delusions is primarily the management of the underlying condition (such as delirium, schizophrenia, mania or psychotic depression). Certain principles apply, however, whatever the underlying cause. Hallucinations and delusions can be very real to, and often frightening for, the person who is experiencing them. Patients will often seek reassurance from the doctor. The doctor should acknowledge that these experiences are real for the patient while avoiding being drawn into colluding with the patient’s false beliefs or perceptions. Statements such as ‘Sometimes when we are unwell our brain plays tricks on us’ can help to reassure a patient. Where the patient lacks insight, however, a more neutral ‘We will have to agree to disagree’ may be necessary to avoid conflict. Antipsychotic medication can reduce psychotic symptoms, such as hallucinations and delusion, and is often used in combination with other sedating medication (such as a benzodiazepine) to alleviate acute

distress and reduce behavioural disturbance (p. 1197). Low mood It is not uncommon for general hospital patients to report low mood. It is important to differentiate an understandable, selflimiting reaction to adversity (such as physical illness or bad news), which is normal and requires support rather than ‘treatment’, from a depressive disorder (p. 1198), which is characterised by a more severe and persistent disturbance of mood and requires specific treatment. *Diseases that may cause organic affective disorders by direct action on the brain. 28.7 Organic mood disorders* Neurological • Cerebrovascular disease • Cerebral tumour • Multiple sclerosis • Parkinson’s disease • Huntington’s disease • Alzheimer’s disease • Epilepsy Endocrine • Hypothyroidism • Hyperthyroidism • Cushing’s syndrome • Addison’s disease • Hyperparathyroidism Malignant disease Infections • Infectious mononucleosis • Herpes simplex • Brucellosis • Typhoid • Toxoplasmosis Connective tissue disease • Systemic lupus erythematosus Drugs • Phenothiazines • Phenylbutazone • Glucocorticoids, oral contraceptives • Interferon 28.6 Symptoms of depressive disorders Psychological • Depressed mood • Reduced self-esteem • Pessimism • Guilt • Loss of interest • Loss of enjoyment (anhedonia) • Suicidal thinking Somatic • Reduced appetite • Weight change • Disturbed sleep • Fatigue • Loss of libido • Bowel disturbance • Motor retardation (slowing of activity)

 stressful events: adjustment disorders (p. 1201). Other more persistent forms of anxiety are described in detail on page 1200. Investigations Anxiety may occasionally be a manifestation of a medical condition such as thyrotoxicosis (Box 28.9). Tests to exclude or confirm these conditions should be considered, particularly if anxiety is a new symptom that has arisen in the absence of an obvious stressor. Management The management of specific anxiety disorders is discussed later in this chapter (p. 1200). Benzodiazepines and related drugs, while extremely effective in the short term, cause tolerance and unpleasant or even dangerous withdrawal syndromes if used for more than a few weeks. Psychological factors affecting medical conditions Psychological factors may influence the presentation, management and outcome of medical conditions. Specific factors are shown in Box 28.10. The most common psychiatric diagnoses in the medically ill are anxiety and depressive disorders. Often these appear understandable as adjustments to illness and its treatment; however, if the anxiety and depression are severe and persistent, they may complicate the management of the medical condition and active management is required. Anxiety may present as an increase in somatic symptoms, such as breathlessness, tremor or palpitations, or as the avoidance of medical treatment. It is most common in those facing difficult or painful treatments, deterioration of their illness or death. Depression may manifest as increased physical symptoms, such as pain, fatigue and disability, as well as with depressed mood and loss of interest and pleasure. It is most common in patients who have suffered Management The management of depression is discussed on page 1199. Where a patient’s low mood is an understandable reaction to adversity, the clinical team can support the patient by minimising uncertainty through open and effective clinical communication and by addressing isolation (allowing access to visitors, telephone and so on). Elevated mood Elevated mood is much less common than depressed mood, and in medical settings is often secondary to drug or alcohol misuse, an organic disorder or medical treatment. Where none of these applies, the patient may be experiencing a manic (or, if less severe, ‘hypomanic’) episode as part of a bipolar affective disorder (p. 1199). Mania is the converse of depression. It may manifest as infectious joviality, over-activity, lack of sleep and appetite, undue optimism, over-talkativeness, irritability, and recklessness in spending and sexual behaviour. When elated mood is severe, psychotic symptoms are often evident, like delusions of grandeur such as believing erroneously that one is

royalty. Investigations The first investigation for any medical patient presenting with persistent and inexplicable elevated mood in the absence of a history of bipolar affective disorder is a medication review. Mania is a relatively common side-effect of certain classes of drug, such as glucocorticoids, and is a rare side-effect of many other drugs. Recreational, herbal and over-the-counter preparations should also be considered. Second-line investigations include tests for Cushing's disease (p. 666), thyrotoxicosis (p. 635), syphilis (p. 337) and encephalitis (p. 1121). Management The management of bipolar affective disorder is discussed on page 1200. Management of organic mania involves identifying and addressing the underlying cause. The management of disturbed or aggressive behaviour is discussed on page 1188. Anxiety Anxiety may be transient, persistent, episodic or limited to specific situations. The symptoms of anxiety are both psychological and physical (Box 28.8). The differential diagnosis of anxiety is shown in Box 28.9. Most anxiety is part of a transient adjustment to 28.10 Risk factors for psychological problems associated with medical conditions • Previous history of depression or anxiety • Lack of social support • New diagnosis of a serious medical condition • Deterioration of, or failure of treatment for, a medical condition • Unpleasant, disabling or disfiguring treatment • Change in medical care, such as discharge from hospital • Impending death 28.9 Differential diagnosis of anxiety • Normal response to threat • Adjustment disorder • Generalised anxiety disorder • Panic disorder • Phobic disorder • Organic (medical) cause: Hyperthyroidism Paroxysmal arrhythmias Phaeochromocytoma Alcohol and benzodiazepine withdrawal Hypoglycaemia Temporal lobe epilepsy 28.8 Symptoms of anxiety disorder Psychological • Apprehension • Irritability • Worry • Poor concentration • Fear of impending disaster • Depersonalisation Somatic • Palpitations • Fatigue • Tremor • Dizziness • Sweating • Diarrhoea • Frequent desire to pass urine • Chest pain • Initial insomnia • Breathlessness • Headache

The incidence of SH varies over time and between countries. In the UK, the lifetime prevalence of suicidal ideation is 15% and that of acts of SH is 4%. SH is more common in women than men, and in young adults than the elderly. (In contrast, completed suicide is more common in men and the elderly; Box 28.12.) There is a higher incidence of SH among lower socioeconomic groups, particularly those living in crowded, socially deprived urban areas. There is also an association with alcohol misuse, child abuse, unemployment and recently broken relationships. Clinical assessment The main differential diagnosis is from accidental poisoning and so-called 'recreational' overdose in drug users. It must be remembered that SH is not a diagnosis but a presentation, and may be associated with any psychiatric diagnosis, the most common being adjustment disorder, substance and alcohol misuse, depressive disorder and personality disorder. In many cases, however, no psychiatric diagnosis can be made. Management A thorough psychiatric and social assessment should be attempted in all cases (Fig. 28.2), although some patients will discharge themselves before this can take place. The need for psychiatric assessment should not delay urgent medical or surgical treatment, though, and may need to be deferred until the patient is well enough for interview. The purpose of the psychiatric assessment is to: • establish the short-term risk of suicide • identify potentially treatable problems, whether medical, psychiatric or social. Topics to be covered when assessing a patient are listed in Box 28.13. The history should include events occurring immediately actual or anticipated losses, such as receiving a terminal diagnosis or undergoing disfiguring surgery. Treatment is by psychological and/or pharmacological therapies, as described on page 1189. Care is required when prescribing psychotropic drugs to the medically ill

in order to avoid exacerbation of the medical condition and harmful interactions with other prescribed drugs. Medically unexplained somatic symptoms Patients commonly present to doctors with physical symptoms. While these symptoms may be an expression of a medical condition, they often are not (see Fig. 28.6). They may then be referred to as ‘medically unexplained symptoms’ (MUS), which are very common in patients attending general medical outpatient clinics. Almost any symptom can be medically unexplained. They include: • pain (including back, chest, abdominal, pelvic and headache) • fatigue • fits, ‘funny turns’, dizziness and feelings of weakness. Patients with MUS may receive a medical diagnosis of a so-called ‘functional somatic syndrome’, such as irritable bowel syndrome (Box 28.11), and may also merit a psychiatric diagnosis on the basis of the same symptoms. The most frequent psychiatric diagnoses associated with MUS are anxiety or depressive disorders. When these are absent, a diagnosis of somatoform disorder may be appropriate. Somatoform disorders are discussed in more detail on page 1202. 28.12 Risk factors for suicide • Psychiatric illness (depressive illness, schizophrenia) • Older age • Male sex • Living alone • Unemployment • Recent bereavement, divorce or separation • Chronic physical ill health • Drug or alcohol misuse • Suicide note written • History of previous attempts (especially if a violent method was used) 28.11 Functional somatic syndromes Medical specialty Somatic syndromes Gastroenterology Irritable bowel syndrome, functional dyspepsia Gynaecology Pre-menstrual syndrome, chronic pelvic pain Rheumatology Fibromyalgia Cardiology Atypical or non-cardiac chest pain Respiratory medicine Hyperventilation syndrome Infectious diseases Chronic (post-viral) fatigue syndrome Neurology Tension headache, non-epileptic attacks, functional gait disorder Dentistry Temporomandibular joint dysfunction, atypical facial pain Ear, nose and throat Globus syndrome Allergy medicine Multiple chemical sensitivity 28.13 Assessment of patients after self-harm Current attempt • Patient’s account • Degree of intent at the time: preparations, plans, precautions against discovery, note • Method used, particularly whether violent • Degree of intent now • Symptoms of psychiatric illness Background • Previous attempts and their outcome • Family and personal history • Social support • Previous response to stress • Extent of drug and alcohol misuse Self-harm Self-harm (SH) is a common reason for presentation to medical services. The term ‘attempted suicide’ is potentially misleading, as most of these patients are not trying to kill themselves. Most cases of SH involve overdose, of either prescribed or non-prescribed drugs (Ch. 7). Less common methods include asphyxiation, drowning, hanging, jumping from a height or in front of a moving vehicle, and the use of firearms. Methods that carry a high chance of being fatal are more likely to be associated with serious psychiatric disorder. Self-cutting is common and often repetitive, but rarely leads to contact with medical services.

 ward is necessary only for persons who display one or more of the following: • an acute psychiatric disorder • high short-term risk of suicide • need for temporary respite from intolerable circumstances • requirement for further assessment of their mental state. Approximately 20% of SH patients make a repeat act during the following year and 1–2% kill themselves. Factors associated with suicide after an episode of SH are listed in Box 28.12. Disturbed and aggressive behaviour Disturbed and aggressive behaviour is common in general hospitals, especially in emergency departments. Most behavioural disturbance arises not from medical or psychiatric illness, but from alcohol intoxication, reaction to the situation and personality characteristics. Clinical assessment The key principles of management are, firstly, to establish control of the situation rapidly and thereby ensure the safety of the patient and others; and secondly, to try to determine the cause of the disturbance in order to remedy it. Establishing control requires the presence of an adequate number of trained staff, an appropriate physical

environment and sometimes sedation (Fig. 28.3). The assistance of hospital security staff and sometimes the police may be required. In all cases, the staff approach is important; a calm, non-threatening manner expressing understanding of the patient's concerns is often all that is required to defuse potential aggression (Box 28.14). An attempt should be made to try to identify the factors that are contributing to the disturbed behaviour. When the patient is cooperative, these are best determined at interview. Other sources of information about the patient include medical and psychiatric records, and discussion with nursing staff, family members and other informants, including the patient's GP. The following information should be sought: • psychiatric, medical (especially neurological) and criminal history • current psychiatric and medical treatment • alcohol and drug misuse • recent stressors • the time course and accompaniments of the current episode in terms of mood, belief and behaviour. Observation of the patient's behaviour may also yield useful clues. Do they appear to be responding to hallucinations? Are they alert or variably drowsy and confused? Are there physical features suggestive of drug or alcohol misuse or withdrawal? Are there new injuries or old scars, especially on the head? Do they smell of alcohol or solvents? Do they bear the marks of drug injection? Are they unwashed and unkempt, suggesting a gradual development of their condition? Investigations Depending on the results of clinical assessment, routine biochemistry, haematology and analysis of blood or urine for illicit drugs or alcohol may be required. Management Measures such as restraint and sedation may be required in patients with acute behavioural disturbance in order to Fig. 28.2 Assessment of patients admitted following self-harm. No Yes Patient admitted following deliberate self-harm Urgent medical treatment needed? Medical assessment to determine need for urgent medical treatment Medical treatment Psychiatric assessment Immediate and significant suicide risk No Yes Plan to minimise risk: • remove methods of self-harm • arrange for special support • consider use of mental health legislation Assess for significant psychiatric illness Suspected? No Yes Assess and arrange management for other problems Consider: • psychiatric admission (using mental health legislation if necessary) • referral to psychiatric service for outpatient treatment before and after the act, and especially any evidence of planning. The nature and severity of any current psychiatric symptoms must be assessed, along with the personal and social supports available to the patient outside hospital. Most SH patients have depressive and anxiety symptoms on a background of chronic social and personal difficulties (often complicated by use of alcohol or other substances), but no psychiatric disorder. They do not usually require psychotropic medication or specialised psychiatric treatment but may benefit from personal support and practical advice from a GP, social worker or community psychiatric nurse. Admission to a psychiatric

can be achieved with antipsychotic drugs (such as haloperidol) and/or benzodiazepines (such as lorazepam or diazepam). The choice of drug, dose, route and rate of administration depends on the patient's age, gender and physical health, as well as the likely cause of the disturbed behaviour. The benefits of sedation must always be balanced against the potential risks. When prescribing benzodiazepines, consider the risk of respiratory depression (particularly in patients with lung disease) and encephalopathy (in those with liver disease). When prescribing antipsychotic drugs for acute sedation, consider the risk of acute dystonias (such as 'oculogyric crisis') and acute arrhythmias (in patients with heart disease). Thus for a frail elderly woman with emphysema and delirium, sedation may be achieved with a low dose (0.5 mg) of oral haloperidol, while for a strong young man with an acute psychotic episode, 10 mg or more of intravenous diazepam and a similar

dose of haloperidol may be required. A parenterally administered anticholinergic agent, such as procyclidine, should be available to treat extrapyramidal effects from haloperidol if they arise. Flumazenil (p. 142) can be used to reverse respiratory depression caused by benzodiazepines. If the initial assessment suggests that the patient has an acute psychiatric disorder, then admission to a psychiatric facility may be indicated. If a medical cause is more likely, psychiatric transfer is usually inappropriate and the patient should be managed in a medical setting, with whatever nursing and security support is required. Where it is clear that there is no medical or psychiatric illness, the person should be removed from the hospital, to police custody if necessary. Many countries, such as the UK, also have specific mental health legislation that may be used to detain patients if necessary. Principles of management of psychiatric disorders The multifactorial origin of most psychiatric disorders means that there are multiple potential targets for treatment. It is useful to consider management strategies within a bio-psycho-social framework. This can help to address the biological factors that contribute to the illness with medication and other physical treatments such as electroconvulsive therapy, while also considering the potential role for psychological therapies and changes to the patient's social environment. Fig. 28.3 Acute management of disturbed behaviour. Yes No Yes No Yes No Disturbed behaviour Does it seem likely to be caused by mental disorder? Consider calling security/police Is the behaviour putting the patient or others at risk? Monitor and review Monitor and review

1. Ensure availability of adequate personnel to provide 'overwhelming force'
2. Try to attain a safe and quiet environment
3. Consider emergency sedation with haloperidol (0.5– 5 mg IM/orally) and/or benzodiazepine (diazepam 5–10 mg IV slowly in view of risk of respiratory depression or lorazepam 1–2 mg IM/orally) Consult with senior staff Consider repeating drug, increasing dose or using other agents such as midazolam or paraldehyde Are measures effective? 28.15 Medical psychiatry in old age • Organic psychiatric disorders: especially common, so cognitive function should always be assessed; if impaired, an associated medical condition or adverse drug effect should be suspected. • Disturbed behaviour: delirium is the most common cause. • Depression: common. Just because a person is old and frail does not mean that depression is 'to be expected' and that it should not be treated. • Self-harm: associated with an increased risk of completed suicide. • Medically unexplained symptoms: common and often associated with depressive disorder. • Loneliness, poverty and lack of social support: must be taken into consideration in management decisions. 28.14 Psychiatric emergencies • Intervene as necessary to reduce the risk of harm to the patient and to others • Adopt a calm, non-threatening approach • Arrange availability of other staff and parenteral medication • Consider diagnostic possibilities of drug intoxication, acute psychosis and delirium • Involve friends and relatives as appropriate identify the cause and to protect the patient and other people from harm. While this potentially raises legal issues, in most countries, including the UK, common law confers on doctors the right, and indeed the duty, to intervene against a patient's wishes if this is necessary. Sedation may be required and

 28.17 The negative cognitive triad associated with depression Cognitive error Example Negative view of self 'I am no good' Negative view of current life experiences 'The world is an awful place' Negative view of the future 'The future is hopeless' Pharmacological treatments These aim to relieve psychiatric disorder by modifying brain function.

The main biological treatments are psychotropic drugs. These are widely used for various purposes; a pragmatic classification is set out in Box 28.16. It should be noted that some drugs have applications to more than one condition; for example, antidepressants are also widely used in the treatment of anxiety and chronic pain. The specific subgroups of psychotropic drugs are discussed in the sections on the appropriate disorders below. Electroconvulsive therapy Electroconvulsive therapy (ECT) entails producing a convulsion by the brief administration of a high-voltage direct-current impulse to the head while the patient is anaesthetised and paralysed by muscle relaxant. If properly administered, it is remarkably safe, has few side-effects, and is of proven efficacy for severe depressive illness. There may be headaches and amnesia for events occurring a few hours before ECT (retrograde) and after it (anterograde). Pronounced amnesia can occur but is infrequent and difficult to distinguish from the effects of severe depression. Other forms of electromagnetic stimulation Clinical trials of transcranial magnetic stimulation (TMS) and vagal nerve stimulation (VNS) suggest they may have a limited role in patients with depression refractory to conventional treatments. Surgery Surgery to the brain (psychosurgery) has a very limited place and then only in the treatment of severe chronic psychiatric illness resistant to other measures. Frontal lobotomies are never done now, and pre-frontal leucotomies are very rare. Operations these days usually target specific sub-regions and tracts of the brain. Psychological therapies These treatments are useful in many psychiatric disorders and also in non-psychiatric conditions. They are based on talking with patients, either individually or in groups. Sometimes discussion is supplemented by ‘homework’ or tasks to complete between treatment sessions. Psychological treatments take a number of forms based on the duration and frequency of contact, the specific techniques applied and their underlying theory. General psychotherapy General psychotherapy should be part of all medical treatment. It involves empathic listening to the patient’s account of their symptoms and associated fears and concerns, followed by the sympathetic provision of accurate information that addresses these. Cognitive therapy This therapy is based on the observation that some psychiatric disorders are associated with systematic errors in the patient’s conscious thinking, such as a tendency to interpret events in a negative way or see them as unduly threatening. A triad of ‘cognitive errors’ has been described in depression (Box 28.17). Cognitive therapy aims to help patients to identify such cognitive errors and to learn how to challenge them. It is widely used for depression, anxiety and eating and somatoform disorders, and also increasingly in psychoses. Behaviour therapy This is a practically orientated form of treatment, in which patients are assisted in changing unhelpful behaviour, such as helping patients to implement carefully graded exposure to the feared stimulus in phobias. Cognitive behaviour therapy Cognitive behaviour therapy (CBT) combines the methods of behaviour therapy and cognitive therapy. It is the most widely available and extensively researched psychological treatment. Action Main groups Clinical use Antipsychotic Phenothiazines Butyrophenones Second-generation antipsychotics Schizophrenia Bipolar mania Delirium Antidepressant Tricyclics and related drugs Serotonin and noradrenergic re-uptake inhibitors Depression/anxiety Obsessive–compulsive disorder Monoamine oxidase inhibitors Depression/anxiety Mood-stabilising Lithium Valproate Lamotrigine Treatment and prophylaxis of bipolar disorder Adjunctive therapy in depression Anti-anxiety Benzodiazepines Anxiety/insomnia (short term) Alcohol withdrawal (short term) β-adrenoceptor antagonists Anxiety (somatic symptoms) } } } } 28.16 Classification of commonly used psychotropic drugs

Interpersonal psychotherapy Interpersonal psychotherapy (IPT) is a specific form of brief psychotherapy that focuses on patients' current interpersonal relationships and is an effective treatment for mild to moderate depression. Social interventions Some adverse social factors, such as unemployment, may not be readily amenable to intervention but others, such as access to benefits and poor housing, may be. Patients can be helped to address these problems themselves by being taught problem-solving. Befrienders and day centres can reduce social isolation, benefits advisers can ensure appropriate financial assistance, and medical recommendations can be made to local housing departments to help patients obtain more appropriate accommodation. Psychiatric disorders Dementia Dementia is a clinical syndrome characterised by a loss of previously acquired intellectual function in the absence of impairment of arousal. It affects 5% of those over 65 and 20% of those over 85. It is defined as a global impairment of cognitive function and is typically progressive and non-reversible. There are many subtypes (Box 28.19) but Alzheimer's disease and diffuse vascular dementia are the most common. Rarer causes of dementia should be actively sought in younger patients and those with short histories. Problem-solving therapy This is a simplified brief form of CBT, which helps patients actively tackle problems in a structured way (Box 28.18). It can be delivered by non-psychiatric doctors and nurses after appropriate training and is commonly used to help patients who self-harm in response to a situational crisis. Psychodynamic psychotherapy This treatment, also known as 'interpretive psychotherapy', was pioneered by Freud, Jung and Klein, among others. It is based on the theory that early life experience generates powerful but unconscious motivations. Psychotherapy aims to help the patient to become aware of these unconscious factors on the assumption that, once identified, their negative effects are reduced. The relationship between therapist and patient is used as a therapeutic tool to identify issues in patients' relationships with others, particularly parents, which may be replicated or transferred to their relationship with the therapist. Explicit discussion of this relationship (transference) is the basis for the treatment, which traditionally requires frequent sessions over a period of months or even years. 28.18 Stages of problem-solving therapy • Define and list problems • Choose one to work on • List possible solutions • Evaluate these and choose the best • Try it out • Evaluate the result • Repeat until problems are resolved 28.19 Subtypes and causes of dementia Type Common Less common Rare Vascular Diffuse small-vessel disease Amyloid angiopathy Multiple emboli Cerebral vasculitis Systemic lupus erythematosus Inherited Alzheimer's disease Fronto-temporal dementia Leukodystrophies Huntington's disease Wilson's disease Dystrophia myotonica Lewy body dementia Progressive supranuclear palsy Mitochondrial encephalopathies Cortico-basal degeneration Neoplastic (p. 1110) Secondary deposits Primary cerebral tumour Paraneoplastic syndrome (limbic encephalitis) Inflammatory – Multiple sclerosis Sarcoidosis Traumatic Chronic subdural haematoma Post-head injury Punch-drunk syndrome – Hydrocephalus (p. 1132) Communicating/non-communicating 'normal pressure' hydrocephalus – Toxic/nutritional Alcohol Thiamin deficiency Vitamin B12 deficiency Anoxia/carbon monoxide poisoning Heavy metal poisoning Infective – Syphilis HIV Post-encephalitis Whipple's disease Subacute sclerosing panencephalitis Prion diseases (p. 1126) – Sporadic Creutzfeldt–Jakob disease (CJD) Variant CJD Kuru Gerstmann–Sträussler–Scheinker disease

 invasive investigations, such as lumbar puncture or, very rarely, brain biopsy, may be indicated. Management This is mainly directed at addressing treatable causes and providing support for patients and carers. Tackling risk factors may slow deterioration, e.g. effective management of hypertension in vascular dementia, or abstinence and vitamin replacement in toxic/nutritional dementias. Psychotropic drugs may have a role in alleviating

symptoms, such as disturbance of sleep, perception or mood, but should be used with care because of an increased mortality in patients who have been treated long-term with these agents. Sedation is not a substitute for good care of patients and carers or, in the later stages, attentive residential nursing care. In the UK, incapacity and mental health legislation may be required to manage patients' financial and domestic affairs, as well as to determine their safe placement. If the diagnosis is Alzheimer-type dementia, cholinesterase inhibitors and memantine may slow progression for a time. Alzheimer's disease Alzheimer's disease is the most common form of dementia. It increases in prevalence with age and is rare in people under 45 years. Pathogenesis Genetic factors play an important role and about 15% of cases are familial. These cases fall into two main groups: early-onset disease with autosomal dominant inheritance and a later-onset group where the inheritance is polygenic. Mutations in several genes have been described but most are rare and/or of small effect. The inheritance of one of the alleles of apolipoprotein ε (apo ε4) is associated with an increased risk of developing the disease (2–4 times higher in heterozygotes and 6–8 times higher in homozygotes). Its presence is, however, neither necessary nor sufficient for the development of the disease and so genetic testing for ApoE4 is not clinically useful. The brain in Alzheimer's disease is macroscopically atrophic, particularly the cerebral cortex and hippocampus. Histologically, the disease is characterised by the presence of senile plaques and neurofibrillary tangles in the cerebral cortex. Histochemical staining demonstrates significant quantities of amyloid in the plaques (Fig. 28.4); these typically stain positive for the protein ubiquitin, which normally is involved in targeting unwanted or damaged proteins for degradation. This has led to the suggestion that the disease may be due to defects in the ability of neuronal cells to degrade unwanted proteins. Many different neurotransmitter abnormalities have also been described. In particular, there is impairment of cholinergic transmission, although abnormalities of noradrenaline (norepinephrine), 5-HT, glutamate and substance P have also been described. Clinical features The key clinical feature is impairment of the ability to remember new information. Hence, patients present with gradual impairment of memory, usually in association with disorders of other cortical functions. Short- and long-term memory are both affected but defects in the former are usually more obvious. Later in the course of the disease, typical features include apraxia, visuo-spatial impairment and aphasia. In the early stages of the disease, patients may notice these problems, but as the disease progresses it is common for patients to deny that there is anything wrong (anosognosia). In this situation, patients are Pathogenesis Dementia may be divided into 'cortical' and 'subcortical' types, depending on the clinical features. Clinical features The usual presentation is with a disturbance of personality or memory dysfunction. A careful history is essential and it is important to interview both the patient and a close family member. Simple bedside tests, such as the MoCA (p. 1182), are useful in assessing the nature and severity of the cognitive deficit, although a more intensive neuropsychological assessment may sometimes be required, especially if there is diagnostic uncertainty. It is important to exclude a focal brain lesion. This is done by determining that there is cognitive disturbance in more than one area. Mental state assessment is important to seek evidence of depression, which may coexist with or occasionally cause apparent cognitive impairment. Many of the primary degenerative diseases that cause dementia have characteristic features that may allow a specific diagnosis during life. Creutzfeldt–Jakob disease, for example, is usually quickly progressive (over months) and is associated with myoclonus. The more slowly progressive dementias are more difficult to distinguish during life, but fronto-temporal dementia typically presents with signs of temporal or frontal lobe dysfunction, whereas Lewy body dementia may present with visual hallucinations. The course may also help to distinguish types of dementia. Gradual worsening suggests Alzheimer's disease, whereas stepwise deterioration is

typical of vascular dementia. Investigations The aim is to seek treatable causes and to estimate prognosis. This is done using a standard set of investigations (Box 28.20). Imaging of the brain can exclude potentially treatable structural lesions, such as hydrocephalus, cerebral tumour or chronic subdural haematoma, though the only abnormality usually seen is that of generalised atrophy. An electroencephalogram (EEG) may be helpful if Creutzfeldt–Jakob disease is suspected, as characteristic abnormalities of generalised periodic sharp wave pattern are usually observed. If the initial tests are negative, more 28.20 Initial investigation of dementia In most patients • Imaging of head (computed tomography and/or magnetic resonance imaging) • Blood tests: Full blood count, erythrocyte sedimentation rate Urea and electrolytes, glucose Calcium, liver function tests Thyroid function tests Vitamin B12 Syphilis serology ANA, anti-dsDNA • Chest X-ray • Electroencephalography In selected patients • Lumbar puncture • HIV serology • Brain biopsy (ANA = antinuclear antibody; anti-dsDNA = anti-double-stranded DNA)

Many patients are depressed, and if this is confirmed, treatment with antidepressant medication may be helpful. Fronto-temporal dementia Fronto-temporal dementia encompasses a number of different syndromes characterised by behaviour abnormalities and impairment of language. Symptoms usually occur before the age of 60 and the prevalence has been estimated at 15 per 100 000 in the population aged between 45 and 65 years. The three major clinical subtypes are behavioural-variant fronto-temporal dementia, primary progressive aphasia and semantic dementia. Pick’s disease is a common cause of the first two in particular. Genetic factors play an important role and familial cases have been described caused by mutations in several genes, including MAPT, which encodes microtubule-associated protein tau, GRN, TPD43, FUS, VCP and C9orf72. The causal mutations trigger abnormal accumulation of tau and other proteins in brain tissue, which are seen as cytoplasmic inclusion bodies on histological examination (Fig. 28.5). It is of interest that many of the gene mutations that cause fronto-temporal dementia are also associated with amyotrophic lateral sclerosis (p. 1116), suggesting that these disorders share a similar pathogenic basis in which neuronal degeneration is caused by accumulation of abnormal proteins. The clinical presentation may be with personality change due to frontal lobe involvement or with language disturbance due to temporal lobe involvement. In contrast to Alzheimer’s disease, memory is relatively preserved in the early stages. There is no specific treatment. Disinhibition and compulsive behaviours can often brought to medical attention by their carers. Depression is commonly present. Occasionally, patients become aggressive, and the clinical features can be made acutely worse by intercurrent physical disease. Patients typically present with subjective memory loss, sometimes getting lost in familiar locations. A history of progressive memory loss and associated functional impairment, corroborated by an informant, is the key to making the diagnosis. Cognitive testing and neuroimaging can be helpful but in themselves are not diagnostic. Investigations Investigation is aimed at excluding treatable causes of dementia (see Box 28.19), as histological confirmation of the diagnosis usually occurs only after death. Management Treatment with anticholinesterases, such as donepezil, rivastigmine and galantamine, has been shown to be of some benefit at slowing progression of cognitive impairment in the early stages of the disease while post-synaptic cholinergic receptors are still available. The N-methyl-D-aspartate (NMDA) receptor antagonist memantine slightly enhances learning and memory in early disease and can also be useful in selected patients with more advanced disease. Novel treatments are under development to block amyloid plaque formation directly, by inhibiting the enzyme γ-secretase.

Non-pharmacological approaches include the provision of a familiar environment for the patient and support for the carers. Fig. 28.4 Alzheimer's disease. Section of neocortex stained with polyclonal antibody against βA4 peptide showing amyloid deposits in plaques in brain substance (arrow A) and in blood-vessel walls (arrow B). Courtesy of Dr J. Xuereb. A B Fig. 28.5 Fronto-temporal dementia. A Lateral view of formalin-fixed brain from a patient who died of Pick's disease, showing gyral atrophy of frontal and parietal lobes and a more severe degree of atrophy affecting the anterior half of the temporal lobe (arrow). B High power (× 200) view of hippocampal pyramidal layer, prepared with monoclonal anti-tau antibody. Many neuronal cell bodies contain sharply circumscribed, spherical cytoplasmic inclusion bodies (Pick bodies, arrows). A and B, Courtesy of Dr J. Xuereb. A B

1194 • MEDICAL PSYCHIATRY Anxiety People who are anxious may use alcohol as a means of relieving anxiety in the short term and this can develop into dependence. Conversely, alcohol withdrawal increases anxiety. Alcohol withdrawal syndrome The features are described in Box 28.22. Symptoms usually become maximal about 2–3 days after the last drink and can include seizures. The term 'delirium tremens' is used to describe severe alcohol withdrawal syndrome characterised by both delirium (characteristically, agitation and visual hallucinations) and physiological hyper-arousal (tremor, sweating and tachycardia). It has a significant mortality and morbidity (Box 28.22). Hallucinations Hallucinations (characteristically visual but sometimes in other modalities) are common in delirium tremens. Less common is the phenomenon called 'alcoholic hallucinosis', where a patient with alcohol dependence experiences auditory hallucination in clear consciousness at a time when they are not withdrawing from alcohol. be helped by selective serotonin re-uptake inhibitors (SSRIs). Although Alzheimer's and fronto-temporal dementia share certain symptoms, they cannot be treated with the same pharmacological agents because the cholinergic systems are not affected in the latter. Lewy body dementia This neurodegenerative disorder is clinically characterised by dementia and signs of Parkinson's disease. It is often inherited and mutations in the α-synuclein and β-synuclein genes have been identified in affected patients. These mutations result in accumulation of abnormal protein aggregates in neurons that contain the protein α-synuclein in association with other proteins, including ubiquitin (see Fig. 25.31, p. 1112). The cognitive state often fluctuates and there is a high incidence of visual hallucinations. Affected individuals are particularly sensitive to the side-effects of anti-parkinsonian medication and also to antipsychotic drugs. There is no curative treatment but anticholinesterase drugs can be helpful in slowing progression of cognitive impairment. Alcohol misuse and dependence Alcohol consumption associated with social, psychological and physical problems constitutes misuse. The criteria for alcohol dependence, a more restricted term, are shown in Box 28.21. Approximately one-quarter of male patients in general hospital medical wards in the UK have a current or previous alcohol problem. Pathogenesis Availability of alcohol and social patterns of use appear to be the most important factors. Genetic factors predispose to dependence. The majority of people who misuse alcohol do not have an associated psychiatric disorder, but a few drink heavily in an attempt to relieve anxiety or depression. Clinical features The modes of presentation of alcohol misuse and complications are summarised below. Social problems Common features include absenteeism from work, unemployment, marital tensions, child abuse, financial difficulties and problems with the law, such as violence and traffic offences. Low mood Low mood is common since alcohol has a direct depressant effect and heavy drinking creates numerous social problems. Attempted and completed suicide are associated with alcohol misuse. 28.22 Presentation and consequences of chronic alcohol misuse Acute intoxication • Emotional and

behavioural disturbance • Medical problems: hypoglycaemia, ketoacidosis, aspiration of vomit, respiratory depression • Accidents, injuries sustained in fights Withdrawal phenomena • Psychological symptoms: restlessness, anxiety, panic attacks • Autonomic symptoms: tachycardia, sweating, pupil dilatation, nausea, vomiting • Delirium: agitation, hallucinations (classically 'Lilliputian'), illusions, delusions • Seizures Consequences of harmful use Medical • Neurological: peripheral neuropathy, cerebellar degeneration, cerebral haemorrhage, dementia • Hepatic: fatty change and cirrhosis, liver cancer • Gastrointestinal: oesophagitis, gastritis, pancreatitis, oesophageal cancer, Mallory–Weiss syndrome, malabsorption, oesophageal varices • Respiratory: pulmonary tuberculosis, pneumonia • Skin: spider naevi, palmar erythema, Dupuytren's contractures, telangiectasias • Cardiac: cardiomyopathy, hypertension • Musculoskeletal: myopathy, fractures • Endocrine and metabolic: pseudo-Cushing's syndrome, hypoglycaemia, gout • Reproductive: hypogonadism, fetal alcohol syndrome, infertility Psychiatric and cerebral • Depression • Alcoholic hallucinosis • Alcoholic 'blackouts' • Wernicke's encephalopathy: nystagmus or ophthalmoplegia with ataxia and delirium • Korsakoff's syndrome: short-term memory deficits leading to confabulation 28.21 Criteria for alcohol dependence • Narrowing of the drinking repertoire • Priority of drinking over other activities (salience) • Tolerance of effects of alcohol • Repeated withdrawal symptoms • Relief of withdrawal symptoms by further drinking • Subjective compulsion to drink • Reinstatement of drinking behaviour after abstinence

arisen. The risk of side-effects, such as respiratory depression with benzodiazepines and anaphylaxis with Pabrinex, is small when weighed against the potential benefits of treatment. Acamprosate (666 mg 3 times daily) may help to maintain abstinence by reducing the craving for alcohol. Disulfiram (200–400 mg daily) can be given as a deterrent to patients who have difficulty resisting the impulse to drink after becoming abstinent. It blocks the metabolism of alcohol, causing acetaldehyde to accumulate. When alcohol is consumed, an unpleasant reaction follows, with headache, flushing and nausea. Disulfiram is always an adjunct to other treatments, especially supportive psychotherapy. Treatment with antidepressants may be required if depression is severe or does not resolve with abstinence. Antipsychotics, such as chlorpromazine (100 mg 3 times daily), are needed for alcoholic hallucinosis. Although such treatment may be successful, there is a high relapse rate. Prognosis Between 80% and 90% of patients with established alcohol dependence syndrome who embark on medically supervised detoxification will successfully complete detoxification without encountering significant complications. Sustaining abstinence is more challenging than achieving it, however. Studies indicate that 1 year after successful detoxification, only 20% of patients will remain abstinent. This figure rises to approximately 30% for patients who are engaged with alcohol services, and to over 40% if such specialist support is combined with supervised disulfiram treatment. Substance misuse disorder Dependence on and misuse of both illegal and prescribed drugs is a major problem worldwide. Drugs of misuse are described in detail in Chapter 7. They can be grouped as follows. Sedatives These commonly give rise to physical dependence, the manifestations of which are tolerance and a withdrawal syndrome. Drugs include benzodiazepines, opiates (including morphine, heroin, methadone and dihydrocodeine) and barbiturates (now rarely prescribed). Overdosage with sedatives can be fatal, primarily as a result of respiratory depression (Ch. 7). Withdrawal from opiates is notoriously unpleasant, and withdrawal from benzodiazepines and barbiturates can cause prolonged anxiety and even hallucinations and/or seizures. Intravenous opiate users are prone to bacterial infections,

hepatitis B (p. 873), hepatitis C (p. 877) and HIV infection (Ch. 12) through needle contamination. Accidental overdose is common, mainly because of the varied and uncertain potency of illicit supplies of the drug. The withdrawal syndrome, which can start within 12 hours of last use, presents with intense craving, rhinorrhoea, lacrimation, yawning, perspiration, shivering, piloerection, vomiting, diarrhoea and abdominal cramps. Examination reveals tachycardia, hypertension, mydriasis and facial flushing. Stimulants Stimulant drugs include amphetamines and cocaine. They are less dangerous than the sedatives in overdose, although they can cause cardiac and cerebrovascular problems through their pressor effects. Physical dependence syndromes do not arise, but withdrawal causes a rebound lowering in mood and can give rise to an intense craving for further use, especially in any form Wernicke–Korsakoff syndrome This is a rare but important indirect complication of chronic alcohol misuse. It is an organic brain disorder resulting from damage to the mamillary bodies, dorsomedial nuclei of the thalamus and adjacent areas of periventricular grey matter caused by a deficiency of thiamin (vitamin B1). The syndrome most commonly results from long-standing heavy drinking and an inadequate diet but can also arise from malabsorption or even protracted vomiting. Wernicke's encephalopathy (nystagmus or ophthalmoplegia with ataxia and delirium) often presents acutely and, without prompt treatment (see below), can progress and become irreversible. Korsakoff's syndrome (severe short-term memory deficits and confabulation) can develop chronically or acutely (with Wernicke's). Alcohol-related brain damage The term alcohol-related brain damage (ARBD) is often used as a collective description of the many brain pathologies associated with alcohol excess, which often coexist in the same patient. Acute alcohol intoxication causes ataxia, slurred speech, emotional incontinence and aggression. Very heavy drinkers may experience periods of amnesia for events that occurred during bouts of intoxication, termed 'alcoholic blackouts'. Established alcohol dependence may lead to 'alcoholic dementia', a global cognitive impairment resembling Alzheimer's disease, but which does not progress and may even improve if the patient becomes abstinent. Heavy alcohol use can damage the brain indirectly through Wernicke–Korsakoff syndrome (see above), head injury, hypoglycaemia and encephalopathy (p. 864). Effects on other organs These are protean and virtually any organ can be involved (Box 28.22). These effects are discussed in detail in other chapters in this book. Diagnosis The diagnosis of alcohol excess may emerge while taking the patient's history, but many patients do not tell the truth about their alcohol intake. Alcohol misuse may also present through its effects on one or more aspects of the patient's life, as listed above. Alcohol dependence commonly presents with withdrawal in those admitted to hospital, as they can no longer maintain their high alcohol intake in this setting. Management For the person misusing alcohol, provision of clear information from a doctor about the harmful effects of alcohol and safe levels of consumption is often all that is needed. In more serious cases, patients may have to be advised to alter leisure activities or change jobs to help them to reduce their consumption. Psychological treatment is used for people who have recurrent relapses and is usually available at specialised centres. Support to stop drinking is also provided by voluntary organisations, such as Alcoholics Anonymous (AA) in the UK. Alcohol withdrawal syndromes can be prevented, or treated once established, with long-acting benzodiazepines. Large doses may be required (such as diazepam 20 mg 4 times daily), tailed off over a period of 5–7 days as symptoms subside. Prevention of the Wernicke–Korsakoff syndrome requires the immediate use of high doses of thiamin, which is initially given parenterally in the form of Pabrinex (two vials 3 times daily for 48 hrs, longer if symptoms persist) and then orally (100 mg 3 times daily). There is no treatment for Wernicke–Korsakoff syndrome once it has

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association with ecstasy. A chronic psychosis has also been documented after regular LSD use. Organic solvents Solvent inhalation (glue sniffing) is popular in some adolescent groups. Solvents produce acute intoxication characterised by euphoria, excitement, dizziness and a floating sensation. Further inhalation leads to loss of consciousness; death can occur from the direct toxic effect of the solvent, or from asphyxiation if the substance is inhaled from a plastic bag. Pathogenesis Many of the causal factors for alcohol misuse also apply to substance misuse. The main factors are the psychological and behavioural vulnerabilities described above, cultural pressures, particularly within a peer group, and availability of a drug. In the case of some drugs such as opiates, medical over-prescribing has increased their availability, but there has also been a relative decline in the price of illegal drugs. Most drug users take a range of drugs – so-called polydrug misuse. Diagnosis As with alcohol, the diagnosis either may be apparent from the history and examination, or may be made only once the patient presents with a complication. Drug screening of samples of urine or blood can be valuable in confirming the diagnosis, especially if the patient persists in denial. Management The first step is to determine whether patients wish to stop using the drug. If they do not, they can still benefit from advice about how to minimise harm from their habit, such as how to obtain and use clean needles for those who inject. For those who are physically dependent on sedative drugs, substitute prescribing (using methadone, for example, in opiate dependence) may help stabilise their lives sufficiently to allow a gradual reduction in dosage until they reach abstinence. Some specialist units offer inpatient detoxification. For details of the medical management of overdose, see page 135. The drug lofexidine, a centrally acting α-agonist, can be useful in treating the autonomic symptoms of opiate withdrawal, as can clonidine, although this carries a risk of hypotension and is best used by specialists. Long-acting opiate antagonists, such as naltrexone, may also have a place, again in specialist hands, in blocking the euphoriant effects of the opiate, thereby reducing addiction.

Management First-episode schizophrenia usually requires admission to hospital because patients lack the insight that they are ill and are unwilling to accept treatment. In some cases, they may be at risk of harming themselves or others. Subsequent acute relapses and chronic schizophrenia are now usually managed in the community. Drug treatment Antipsychotic agents are effective against the positive symptoms of schizophrenia in the majority of cases. They take 2–4 weeks to be maximally effective but have some beneficial effects shortly after administration. Treatment is then ideally continued to prevent relapse. In a patient with a first episode of schizophrenia this will usually be for 1 or 2 years, but in patients with multiple episodes treatment may be required for many years. The benefits of prolonged treatment must be weighed against the adverse effects, which include extrapyramidal side-effects (EPSE) like acute dystonic reactions (which may require treatment with parenteral anticholinergics), akathisia and parkinsonism. For long-term use, antipsychotic agents are often given by slow-release (depot) injections to improve adherence. A number of antipsychotic agents are available (Box 28.25). These may be divided into conventional (first-generation) drugs such as chlorpromazine and haloperidol, and novel or secondgeneration drugs such as olanzapine and clozapine. All work by blocking D2 dopamine receptors in the brain. Patients who have not responded to conventional drugs may respond to newer agents, which are also less likely to produce unwanted EPSE but do tend to cause greater weight gain and metabolic disturbances, such as dyslipidaemia. Clozapine can be remarkably effective in those who do not respond to other antipsychotics but can cause agranulocytosis in about 1% of patients in the first

few months. Prescription therefore requires regular monitoring of white blood cell count, initially on a weekly basis, then fortnightly and monthly thereafter. Clozapine should not be stopped suddenly because of the likelihood of relapse. Adverse effects of antipsychotic drugs are listed in Box 28.26. Two serious adverse effects deserve special mention. Neuroleptic malignant syndrome This is a rare but serious condition characterised by fever, tremor and rigidity, autonomic instability Other characteristic symptoms are delusions of control: believing that one's emotions, impulses or acts are controlled by others. Another phenomenon is delusional perception, a delusion that arises suddenly alongside a normal perception, such as 'I saw the moon and I immediately knew he was evil.' Other, less common, symptoms may occur, including thought disorder, as manifest by incomprehensible speech, and abnormalities of movement, such as those in which the patient can become immobile or adopt awkward postures for prolonged periods (catatonia). Diagnosis The diagnosis is made primarily on clinical grounds but investigations may be required to rule out organic brain disease. The main differential diagnosis of schizophrenia (Box 28.24) includes: • Other functional psychoses, particularly psychotic depression and mania, in which delusions and hallucinations are congruent with a marked mood disturbance (negative in depression and grandiose in mania). Schizophrenia must also be differentiated from specific delusional disorders that are not associated with the other typical features of schizophrenia. • Organic psychoses, including delirium, in which there is impairment of consciousness and loss of orientation (not found in schizophrenia), typically with visual hallucinations; drug misuse, particularly in young people; and temporal lobe epilepsy with psychotic symptoms, in which olfactory and gustatory hallucinations may occur. Many of those who experience acute schizophrenia go on to develop a chronic state in which the acute, so-called positive symptoms resolve, or at least do not dominate the clinical picture, leaving so-called negative symptoms that include blunt affect, apathy, social isolation, poverty of speech and poor self-care. Patients with chronic schizophrenia may also manifest positive symptoms, particularly when under stress, and it can be difficult for those who do not know the patient to judge whether or not these are signs of an acute relapse. Investigations As in dementia, investigations are focused on excluding a treatable cause, such as a slow-growing brain tumour, temporal lobe epilepsy, neurosyphilis or various autoimmune conditions. These are required only in patients with neurological or other organic symptoms or signs. 28.23 Symptoms of schizophrenia First-rank symptoms of acute schizophrenia • A = Auditory hallucinations – second- or third-person/écho de la pensée • B = Broadcasting, insertion/withdrawal of thoughts • C = Controlled feelings, impulses or acts ('passivity' experiences/ phenomena) • D = Delusional perception (a particular experience is bizarrely interpreted) Symptoms of chronic schizophrenia (negative symptoms) • Flattened (blunted) affect • Apathy and loss of drive (avolition) • Social isolation/withdrawal (autism) • Poverty of speech (alogia) • Poor self-care 28.24 Differential diagnosis of schizophrenia Alternative diagnosis Distinguishing features Other functional psychoses Delusional disorders Absence of specific features of schizophrenia Psychotic depression Prominent depressive symptoms Manic episode Prominent manic symptoms Schizoaffective disorder Mood and schizophrenia symptoms both prominent Puerperal psychosis Acute onset after childbirth Organic disorders Drug-induced psychosis Evidence of drug or alcohol misuse Side-effects of prescribed drugs Levodopa, methyldopa, glucocorticoids, antimalarial drugs Temporal lobe epilepsy Other evidence of seizures Delirium Visual hallucinations, impaired consciousness Dementia Age, established cognitive impairment Huntington's disease Family history, choreiform movements, dementia

1198 • MEDICAL PSYCHIATRY given as part of an integrated treatment package, reduces the rate of relapse. Social treatment After an acute episode of schizophrenia has been controlled by drug therapy, social rehabilitation may be required. Recurrent illness is likely to cause disruption to patients' relationships and their ability to manage their accommodation and occupation; consequently, patients with schizophrenia often need help to obtain housing and employment. A graded return to employment and sometimes a period of supported accommodation are required. Patients with chronic schizophrenia have particular difficulties and may need long-term, supervised accommodation. This now tends to be in supported accommodation in the community. Patients may also benefit from sheltered employment if they are unable to participate effectively in the labour market. Ongoing contact with a health worker allows monitoring for signs of relapse, sometimes as part of a multidisciplinary team working to agreed plans (the 'care programme approach'). Partly because of a tendency to inactivity, smoking and a poor diet, patients with chronic schizophrenia are at increased risk of cardiovascular disease, diabetes and stroke, and require proactive medical as well as psychiatric care. Prognosis About one-third of those who develop an acute schizophrenic episode have a good outcome. One-third develop chronic, incapacitating schizophrenia, and the remainder largely recover after each episode but suffer relapses. Most affected patients cannot work or live independently. Schizophrenia is associated with suicide and up to 10% of patients take their own lives. Mood disorders Mood or affective disorders include: • unipolar depression: one or more episodes of low mood and associated symptoms • bipolar disorder: episodes of elevated mood interspersed with episodes of depression • dysthymia: chronic low-grade depressed mood without sufficient other symptoms to count as 'clinically significant' or 'major' depression. Depression Major depressive disorder has a prevalence of 5% in the general population and approximately 10–20% in chronically ill medical outpatients. It is a major cause of disability and suicide. If comorbid with a medical condition, depression magnifies disability, diminishes adherence to medical treatment and rehabilitation, and may even shorten life expectancy. Pathogenesis There is a genetic predisposition to depression, especially when of early onset. The genetic predisposition is mediated by variants in a large number of genes and loci of small effect rather than mutations in single genes. Adversity and emotional deprivation early in life also predispose to depression. Depressive episodes are often, but not always, triggered by stressful life events (especially those that involve loss or imposed change), including medical illnesses. Associated biological factors include and delirium. Characteristic laboratory findings are an elevated creatinine phosphokinase and leucocytosis. Antipsychotic medication must be stopped immediately and supportive therapy provided, often in an intensive care unit. Treatment includes ensuring hydration and reducing hyperthermia. Dantrolene sodium and bromocriptine may be helpful. Mortality is 20% untreated and 5% with treatment. Cardiac arrhythmias Antipsychotic medications cause prolongation of the QTc interval, which may be associated with ventricular tachycardia, torsades de pointes and sudden death. If this occurs, treatment should be stopped, with careful electrocardiographic monitoring and treatment of serious arrhythmias if necessary (p. 479). Psychological treatment Psychological treatment, including general support for the patient and family, is now seen as an essential component of management. CBT may help patients to cope with symptoms. There is evidence that personal and/or family education, when 28.26 Adverse effects of antipsychotic drugs Weight gain due to increased appetite Effects due to dopamine blockade* • Acute dystonia • Akathisia (motor restlessness) • Parkinsonism • Tardive dyskinesia • Gynaecomastia • Galactorrhoea Effects due to cholinergic blockade • Dry mouth • Blurred vision • Impotence • Constipation • Urinary retention Hypersensitivity reactions • Blood dyscrasias (neutropenia with clozapine) • Cholestatic jaundice • Photosensitive dermatitis Ocular

complications • Corneal and lens opacities (long-term use) *Less severe with clozapine, quetiapine and olanzapine, possibly because of strong 5-hydroxytrytamine-blocking effect and relatively weak dopamine blockade. 28.25 Antipsychotic drugs Group Drug Usual adult dose1 Phenothiazines Chlorpromazine 400–600 mg daily Butyrophenones Haloperidol 8–12 mg daily Thioxanthenes Flupentixol decanoate 40 mg fortnightly (depot injection) Diphenylbutylpiperidines Pimozide 8–10 mg daily Substituted benzamides Sulpiride 800–1200 mg daily Dibenzodiazepines2 Clozapine 300–600 mg daily Benzisoxazole2 Risperidone 4–6 mg daily Thienobenzodiazepines2 Olanzapine 10–15 mg daily Dibenzothiazepines2 Quetiapine 300–600 mg daily 1Lower or higher doses may be required in some patients. 2Second-generation antipsychotics.

an effective drug for an adequate period. For those who do not respond, a proportion will do so if changed to another class of antidepressant. The patient’s progress must be monitored and, after recovery, treatment should be continued for at least 6–12 months to reduce the high risk of relapse. The dose should then be tapered off over several weeks to avoid discontinuation symptoms. The Scottish Intercollegiate Guidelines Network (SIGN) and National Institute for Health and Clinical Excellence (NICE) have published treatment guidelines. Tricyclic antidepressants Tricyclic antidepressant (TCA) agents inhibit re-uptake of the amines noradrenaline (norepinephrine) and 5-HT at synaptic clefts. The therapeutic effect is noticeable within a week or two. Adverse effects, such as sedation, anticholinergic effects, postural hypotension, lowering of the seizure threshold and cardiotoxicity, can be troublesome during this period. TCAs may be dangerous in overdose and should be used with caution in people who have coexisting heart disease, glaucoma and prostatism. Selective serotonin re-uptake inhibitors Selective serotonin reuptake inhibitors (SSRIs) are less cardiotoxic and less sedative than TCAs, and have fewer anticholinergic effects. They are safer in overdose but can still cause QTc prolongation, headache, nausea, anorexia and sexual dysfunction. They can also interact with other drugs increasing serotonin (5-HT), to produce ‘serotonin syndrome’. This is a rare syndrome of neuromuscular hyperactivity, autonomic hyperactivity and agitation, and potentially seizures, hyperthermia, delirium and even death. Noradrenaline (norepinephrine) re-uptake inhibitors These agents inhibit noradrenaline uptake at the synaptic cleft but have additional pharmacological effects. Venlafaxine and duloxetine also act as serotonin re-uptake inhibitors, whereas mirtazapine also acts as an antagonist at 5-HT2a, 5-HT2c and 5-HT3 receptors. These drugs have similar efficacy to the agents listed above but a different adverse-effect profile. Monoamine oxidase inhibitors Monoamine oxidase inhibitors (MAOIs) increase the availability of neurotransmitters at synaptic clefts by inhibiting metabolism of noradrenaline (norepinephrine) and 5-HT. They are now rarely prescribed in the UK, since they can cause potentially dangerous interactions with drugs such as amphetamines and certain anaesthetic agents, and with foods rich in tyramine (such as cheese and red wine). This is due to accumulation of amines in the systemic circulation, causing a potentially fatal hypertensive crisis. Psychological treatment Both CBT and interpersonal therapy are as effective as antidepressants for mild to moderate depression. Antidepressant drugs are, however, preferred for severe depression. Drug and psychological treatments can be used in combination. Prognosis Over 50% of people who have had one depressive episode and over 90% of people who have had three or more episodes will have another. The risk of suicide in an individual who has had a depressive disorder is 10 times greater than in the general population. Bipolar disorder Bipolar disorder is an episodic disturbance with interspersed periods of depressed and

elevated mood; the latter is known hypofunction of monoamine neurotransmitter systems, including 5-HT and noradrenaline (norepinephrine), and abnormalities of the hypothalamic–pituitary–adrenal (HPA) axis, which results in elevated cortisol levels that do not suppress with dexamethasone. Diagnosis The symptoms are listed in Box 28.6. Depression may be mild, moderate or severe. It may also be recurrent or chronic. It can be both a complication of a medical condition and a cause of MUS (see below), so physical examination is essential; an associated medical condition should always be considered, particularly where there is no past history of depression and no apparent psychological precipitant. Investigations Investigations are not usually required unless there are clinical grounds for suspicion of an underlying medical disorder, such as Cushing's syndrome or hypothyroidism. Management Pharmacological and psychological treatments both work in depression. In practice, the choice is determined by patient preference and local availability. Severe depression complicated by psychotic symptoms, dehydration or suicide risk may require ECT. Drug treatment Antidepressant drugs are effective in moderate and severe depression, whether it is primary or secondary to a medical illness. The most suitable drug for an individual patient will depend on their previous response, likely side-effects, their concurrent illnesses and potential drug interactions. Commonly used antidepressants are shown in Box 28.27. The different classes of antidepressant have similar efficacy and about three-quarters of patients respond to treatment. Successful treatment requires the patient to take an appropriate dose of 28.27 Antidepressant drugs Group Drug Usual adult dose* Tricyclic antidepressants Amitriptyline 75–150 mg daily Imipramine 75–150 mg daily Dosulepin 75–150 mg daily Clomipramine 75–150 mg daily Selective serotonin re-uptake inhibitors (SSRIs) Citalopram 20–40 mg daily Escitalopram 10–20 mg daily Fluoxetine 20–60 mg daily Sertraline 50–100 mg daily Paroxetine 20–50 mg daily Monoamine oxidase inhibitors Phenelzine 45–90 mg daily Tranylcypromine 20–40 mg daily Moclobemide 300–600 mg daily Noradrenaline (norepinephrine) re-uptake inhibitors and SSRIs Venlafaxine 75–375 mg daily Duloxetine 60–120 mg daily Noradrenaline and specific serotonergic inhibitor Mirtazapine 15–45 mg daily *Higher doses may be required in some patients: see guidelines.

 Anxiety disorders These are characterised by the emotion of anxiety, worrisome thoughts, avoidance behaviours and the somatic symptoms of autonomic arousal. Anxiety disorders are divided into three main subtypes: phobic, paroxysmal (panic) and generalised (Box 28.28). The nature and prominence of the somatic symptoms often lead the patient to present initially to medical services. Anxiety may be stress-related and phobic anxiety may follow an unpleasant incident. Many patients with anxiety also have depression. Clinical features Phobic anxiety disorder A phobia is an abnormal or excessive fear of a specific object or situation, which leads to avoidance of it (such as excessive fear of dying in an air crash, leading to avoidance of flying). A generalised phobia of going out alone or being in crowded places is called 'agoraphobia'. Phobic responses can develop to medical procedures such as venepuncture. Panic disorder Panic disorder describes repeated attacks of severe anxiety, which are not restricted to any particular situation or circumstances. Somatic symptoms, such as chest pain, palpitations and paraesthesia in lips and fingers, are common. The symptoms are in part due to involuntary over-breathing (hyperventilation). Patients with panic attacks often fear that they are suffering from a serious illness, such as a heart attack or stroke, and seek emergency medical attention. Panic disorder may coexist with agoraphobia. Generalised anxiety disorder This is a chronic anxiety state associated with uncontrollable worry. The associated somatic symptoms of muscle tension and bowel disturbance often lead to a medical presentation. Diagnosis The diagnosis is made on the

basis of clinical history and typical symptoms, as described above. Where a diagnosis of panic disorder is suspected, it can be confirmed by asking the patient to hyperventilate deliberately for 1–2 minutes and observing whether the symptoms are reproduced. A finding of respiratory alkalosis on arterial blood gas measurement is indicative of chronic hyperventilation. Management Psychological treatment Explanation and reassurance are essential, especially when patients fear they have a serious medical condition. Specific as hypomania when mild or short-lived, or mania when severe or chronic. The lifetime risk of developing bipolar disorder is approximately 1–2%. Onset is usually in the twenties, and men and women are equally affected. Pathogenesis Bipolar disorder is strongly heritable (approximately 70%). Relatives of patients have an increased incidence of both bipolar and unipolar affective disorder. A number of genetic variants of small effect have been identified by genome-wide association studies. Life events, such as physical illness, sleep deprivation and medication, may also play a role in triggering episodes. Diagnosis The diagnosis is based on clear evidence of episodes of depression and mania. Isolated episodes of hypomania or mania do occur but they are usually preceded or followed by an episode of depression. Psychotic symptoms may occur in both the depressive and the manic phases, with delusions and hallucinations that are usually in keeping with the mood disturbance. This is described as an affective psychosis. Patients who present with symptoms of both bipolar disorder and schizophrenia in equal measure may be given a diagnosis of schizoaffective disorder. Management Depression should be treated as described above. If antidepressants are prescribed, however, they should be combined with a mood-stabilising drug (see below) to avoid ‘switching’ the patients into (hypo)mania. Manic episodes and psychotic symptoms usually respond well to antipsychotic drugs (see Box 28.25). Prophylaxis to prevent recurrent episodes of depression and mania with mood-stabilising agents is important. The main drugs used are lithium and sodium valproate but lamotrigine, olanzapine, quetiapine and risperidone are increasingly employed. Caution must be exercised when stopping these drugs, as a relapse may follow. Lithium carbonate is the drug of first choice. It is also used for acute mania, and in combination with a tricyclic as an adjuvant treatment for resistant depression. It has a narrow therapeutic range, so regular blood monitoring is required to maintain a serum level of 0.5–1.0 mmol/L. Toxic effects include nausea, vomiting, tremor and convulsions. With long-term treatment, weight gain, hypothyroidism, increased calcium and parathyroid hormone (PTH), nephrogenic diabetes insipidus (p. 687) and renal failure can occur. Thyroid and renal function should be checked before treatment is started and regularly thereafter. Lithium may be teratogenic and should not be prescribed during the first trimester of pregnancy. Anticonvulsants, such as sodium valproate and lamotrigine, and the antipsychotic drug olanzapine can all be used as prophylaxis in bipolar disorder, usually as a second-line alternative to lithium. Valproate conveys a high risk of birth defects and should not be used in women of child-bearing age. Olanzapine can cause significant weight gain. (For a list of the adverse effects of antipsychotic drugs, see Box 28.26.) Prognosis The relapse rate of bipolar disorder is high, although patients may be perfectly well between episodes. After one episode, the annual average risk of relapse is about 10–15%, which doubles after more than three episodes. There is a substantially increased lifetime risk of suicide of 5–10%. 28.28 Classification of anxiety disorders Phobic anxiety disorder Panic disorder Generalised anxiety disorder Occurrence Situational Paroxysmal Persistent Behaviour Avoidance Escape Agitation Cognitions Fear of situation Fear of symptoms Worry Symptoms On exposure Episodic Persistent

The symptoms are transient and usually resolve completely within a few days. The lay media often describes this as ‘shock’. Adjustment disorder A more common psychological response to a major stressor is a less severe but more prolonged emotional reaction. Clinical features The predominant symptom is usually depression and/or anxiety, which is insufficiently persistent or intense to merit a diagnosis of depressive or anxiety disorder. There may also be anger, aggressive behaviour and associated excessive alcohol use. Symptoms develop within a month of the onset of the stress, and their duration and severity reflect the course of the underlying stressor. Grief reactions following bereavement are a particular type of adjustment disorder. They manifest as a brief period of emotional numbing, followed by a period of distress lasting several weeks, during which sorrow, tearfulness, sleep disturbance, a sense of futility, anger and ‘bargaining’ are common. Perceptual distortions may occur, including misinterpreting sounds as the dead person’s voice or ‘seeing’ the dead person. ‘Pathological grief’ describes a grief reaction that is abnormally intense or persistent. Diagnosis The diagnosis is made on the basis of the typical history following a stressful life event, as described above. Management Ongoing contact with and support from a doctor or another person who can listen, reassure, explain and advise are often all that is needed. Most patients do not require psychotropic medication, although benzodiazepines reduce arousal in acute stress reactions and can aid sleep in adjustment disorders. Post-traumatic stress disorder Post-traumatic stress disorder (PTSD) is a delayed and/or protracted response to a stressful event of an exceptionally threatening or catastrophic nature. Examples of such events include natural disasters, terrorist activity, serious accidents and witnessing violent deaths. PTSD may also sometimes occur after distressing medical treatments or intensive care. Clinical features The development of PTSD is usually delayed from a few days to several months between the traumatic event and the onset of symptoms. Typical symptoms are recurrent intrusive memories (flashbacks) of the trauma; sleep disturbance, especially nightmares (usually of the traumatic event) from which the patient awakes in a state of anxiety; symptoms of autonomic arousal (anxiety, palpitations, enhanced startle); emotional blunting; and avoidance of situations that evoke memories of the trauma. Anxiety and depression are often associated and excessive use of alcohol or drugs frequently complicates the clinical picture. Diagnosis The diagnosis is made on the basis of the typical clinical features following a traumatic life event. Management In the immediate aftermath of a significant trauma, the main aim is to provide support, direct advice and the opportunity treatment may be needed. Treatments include relaxation, graded exposure (desensitisation) to feared situations for phobic disorders, and CBT. Drug treatment Antidepressants are the drugs of first choice (p. 1199). The therapeutic dose is usually higher for anxiety disorders than for depression and there is some evidence that, within their respective classes, paroxetine (SSRI) and clomipramine (TCA) have greatest efficacy against anxiety disorders. Early side-effects of antidepressants can lead to a worsening of anxiety symptoms in the first 2 weeks and patients should be warned of this. Benzodiazepines are useful in the short term but regular (> 3 doses per week) long-term use carries a very high risk of dependence. Regular prescriptions should therefore be limited to 3 weeks; beyond that, prescriptions should be restricted to occasional use as required, with periodic review to guard against dose escalation. Short-acting benzodiazepines, such as lorazepam, have a rapid onset and provide symptomatic relief for up to 2 hours but have the greatest potential for dependence. Longer-acting drugs, such as diazepam, can take an hour to take effect when given orally but provide symptomatic relief for up to 12 hours. A β-blocker, such as propranolol, can help when somatic symptoms are prominent. Obsessive–compulsive disorder Obsessive–compulsive disorder (OCD) is characterised by ‘obsessions’ – thoughts, images or impulses that are recurrent, unwanted and usually anxiety-provoking, but recognised as one’s own.

In many cases, the obsessions give rise to ‘compulsions, which are repeated acts performed to relieve the anxiety. Unlike the anxiety disorders discussed above, which are more common in women, OCD is equally common in men and women. Clinical features Common examples include thoughts of contamination, giving rise to repeated and ritualised hand-washing, and thoughts of having forgotten something, giving rise to time-consuming repeated checking. The differential diagnoses include normal checking behaviour and delusional beliefs about thought possession. Diagnosis The diagnosis is made on the basis of the typical history, as described above. Management OCD usually responds to some degree to antidepressant drugs (high-dose clomipramine or SSRI; see Box 28.27) and to ‘exposure response prevention’ – a form of CBT in which patients are encouraged to expose themselves to the feared thought or situation without performing the anxiety-relieving compulsions. Relapses are common, however, and the condition often becomes chronic. Stress-related disorders Acute stress reaction Following a stressful event, such as a serious medical diagnosis or a major accident, some people develop a characteristic pattern of symptoms: an initial state of ‘daze’ or bewilderment is followed by altered activity (withdrawal or agitation), often with anxiety.

 Dissociative conversion disorders Dissociative conversion disorders are characterised by a loss or distortion of neurological functioning that is not fully explained by organic disease. These may be psychological functions such as memory (‘dissociative amnesia’), sensory functions such as vision (‘dissociative blindness’), or motor functions (‘functional gait disorder’) (Box 28.29). The cause is unknown but there is an association with recent stress and with adverse childhood experiences, including physical and sexual abuse. Organic disease may precipitate dissociation and provide a model for symptoms. For example, non-epileptic seizures often occur in those with epilepsy. Treatment with CBT may be of benefit. Somatisation disorder This is defined as the occurrence of multiple medically unexplained physical symptoms affecting several bodily systems. It is also known as Briquet’s syndrome after the physician who first described the presentation. Symptoms often start in early adult life but somatisation disorder can arise later, usually following an episode of physical illness. The disorder is much more common in women. Patients may undergo a multitude of negative investigations and unhelpful operations, particularly hysterectomy and cholecystectomy. There is no proven treatment except to try to ensure that unnecessary investigations and surgical procedures are avoided to minimise iatrogenic harm. Hypochondriacal disorder Patients with this condition have a strong fear or belief that they have a serious, often fatal, disease (such as cancer), and that fear persists despite appropriate medical reassurance. They are typically highly anxious and seek many medical opinions and investigations in futile but repeated attempts to relieve their fears. Hypochondriacal disorder often resembles OCD, but in a small proportion of cases the conviction that disease is present reaches delusional intensity. The best-known example is that of parasitic infestation (‘delusional parasitosis’), which leads patients to consult dermatologists. Treatment with CBT can be helpful. Patients who suffer delusions may benefit from antipsychotic medication. The condition may become chronic. Body dysmorphic disorder This is defined as a preoccupation with bodily shape or appearance, with the belief that one is disfigured in some way (previously known as ‘dysmorphophobia’). People with this condition may make inappropriate requests for cosmetic surgery. Treatment with CBT or antidepressants may be helpful. The belief in disfigurement may sometimes be delusional, in which case antipsychotic drugs can help. Management The management of the various syndromes of medically unexplained complaints described above is based on the general principles outlined in Box 28.30 and discussed in more detail below. for

emotional catharsis (debriefing may actually be harmful). In established PTSD, structured psychological approaches (CBT, eye movement desensitisation and reprocessing (EMDR), and stress management) are effective. Antidepressant drugs are moderately effective. Prognosis The condition runs a fluctuating course, with most patients recovering within 2 years. In a small proportion, the symptoms become chronic. Somatoform disorders The essential feature of these disorders is that the somatic symptoms are not explained by a medical condition (medically unexplained symptoms), nor better diagnosed as part of a depressive or anxiety disorder. The derivation of the term 'somatoform' is 'body-like'. Several syndromes are described within this category; there is considerable overlap between them, both in the underlying causes and in the clinical presentation. Pathogenesis The cause of somatoform disorders is incompletely understood but contributory factors include depression and anxiety, the erroneous interpretation of somatic symptoms as evidence of disease, excessive concern with physical illness and a tendency to seek medical care. A family history or previous history of a particular condition may have shaped the patient's beliefs about illness. Doctors may exacerbate the problem, either by dismissing the complaints as non-existent or by over-emphasising and investigating the possibility of disease. Clinical features Somatoform disorders can present in several different ways, as described below. Somatoform autonomic dysfunction This describes somatic symptoms referable to bodily organs that are largely under the control of the autonomic nervous system. The most common examples involve the cardiovascular system ('cardiac neurosis'), respiratory system ('psychogenic hyperventilation') and gut ('psychogenic vomiting' and 'irritable bowel syndrome'). Antidepressant drugs and CBT may be helpful. Somatoform pain disorder This describes severe, persistent pain that cannot be adequately explained by a medical condition. Antidepressant drugs (especially tricyclics and dual action drugs such as duloxetine) are helpful, as are some of the anticonvulsant drugs, particularly carbamazepine, gabapentin and pregabalin. CBT and multidisciplinary pain management teams are also useful. Chronic fatigue syndrome Chronic fatigue syndrome (CFS) is characterised by excessive fatigue after minimal physical or mental exertion, poor concentration, dizziness, muscular aches and sleep disturbance. This pattern of symptoms may follow a viral infection such as infectious mononucleosis, influenza or hepatitis. Symptoms overlap with those of depression and anxiety. There is good evidence that many patients improve with carefully graded exercise and with CBT, as long as the benefits of such treatment are carefully explained. 28.29 Common presentations of dissociative (conversion) disorder • Gait disturbance • Loss of function in limbs • Aphonia • Non-epileptic seizures • Sensory loss • Blindness

re-referral for investigation, to ensure that treatable aspects of the patient's problems, such as depression, are actively managed and to prevent the GP from becoming demoralised. Eating disorders There are two well-defined eating disorders, anorexia nervosa (AN) and bulimia nervosa (BN); they share some overlapping features. Ninety per cent of people affected are female. There is a much higher prevalence of abnormal eating behaviour in the population that does not meet diagnostic criteria for AN or BN but may attract a diagnostic label such as 'binge eating disorder'. In developed societies, obesity is arguably a much greater problem but is usually considered to be more a disorder of lifestyle or physiology than a psychiatric disorder. Anorexia nervosa The lifetime risk of anorexia nervosa for women living in Europe is approximately 1–2% (for men it is < 0.5%) with a peak age of onset of 15–19 years. Predisposing factors include familiality (both genetic and shared environmental factors appear to play a role) and 'neurotic' personality traits. The illness is

often precipitated by weight loss, whether due to non-pathological dieting/increased exercise or physical illness such as gastrointestinal disorders or diabetes mellitus. Many sufferers do not engage with specialist services and it is not uncommon for the first presentation to be with a medical problem (Box 28.31) rather than to psychiatric services. Clinical features There is marked weight loss, arising from food avoidance, often in combination with bingeing, purging, excessive exercise and/ or the use of diuretics and laxatives. Body image is profoundly disturbed so that, despite emaciation, patients still feel overweight and are terrified of weight gain. These preoccupations are intense and pervasive, and the false beliefs may be held with a conviction approaching the delusional. Anxiety and depressive symptoms are common accompaniments. Downy hair (lanugo) may develop on the back, forearms and cheeks. Extreme starvation is associated with a wide range of physiological and pathological bodily changes. All organ systems may be affected, although the most serious problems are cardiac and skeletal (Box 28.31). Pathogenesis The underlying cause is unclear but probably includes personality (high neuroticism), genetic (twin studies indicate heritability of 0.3–0.5) and environmental factors, including, in many societies, the social pressure on women to be thin. Diagnosis Diagnostic criteria are shown in Box 28.32. Differential diagnosis is from other causes of weight loss, including psychiatric disorders such as depression, and medical conditions such as inflammatory bowel disease, malabsorption, hypopituitarism and cancer, although it is important to remember that AN can coexist with any of these. The diagnosis is based on a pronounced fear of fatness despite being thin, and on the absence of an adequate alternative explanation for weight loss. Management The aims of management are to ensure patients' physical wellbeing while helping them to gain weight by addressing the Reassurance Patients should be asked what they are most worried about. Clearly, it may be unwise to state categorically that the patient does not have any disease, as that is difficult to establish with certainty. However, it can be emphasised that the probability of having a disease is low and that doctors often see patients with physical symptoms but no physical disease. If patients repeatedly ask for reassurance about the same health concern despite reassurance, they may have hypochondriasis. Explanation Patients need a positive explanation for their symptoms. It is unhelpful to say that symptoms are psychological or 'all in the mind'. Rather, a term such as 'functional' (meaning that the symptoms represent a reversible disturbance of bodily function) may be more acceptable. When possible, it is useful to describe a plausible physiological mechanism that is linked to psychological factors such as stress and implies that the symptoms are reversible. For example, in irritable bowel syndrome, psychological stress results in increased activation of the autonomic nervous system, which leads to constriction of smooth muscle in the gut wall, which in turn causes pain and bowel disturbance. Advice This should focus on how to overcome factors perpetuating the symptoms: for example, by resolving stressful social problems or by practising relaxation. The doctor can offer to review progress, to prescribe (for example) an antidepressant drug and, if appropriate, to refer for physiotherapy or psychological treatments such as CBT. The attitudes of relatives may need to be addressed if they have adopted an over-protective role, unwittingly reinforcing the patient's disability. Drug treatment Antidepressant drugs are often helpful, even if the patient is not depressed. Psychological treatment There is evidence for the effectiveness of CBT. Other psychological treatments such as IPT may also have a role. Rehabilitation Where there is chronic disability, particularly in dissociative (conversion) disorder, conventional physical rehabilitation may be the best approach. Shared care Ongoing planned care is required for patients with chronic intractable symptoms, especially those of somatisation disorder. Review by the same specialist, interspersed with visits to the same GP, is probably the best way to avoid unnecessary multiple 28.30 General management principles for medically

unexplained symptoms • Take a full sympathetic history • Exclude disease but avoid unnecessary investigation or referral • Seek specific treatable psychiatric syndromes • Demonstrate to patients that you believe their complaints • Establish a collaborative relationship • Give a positive explanation for the symptoms, including but not over-emphasising psychological factors • Encourage a return to normal functioning

1204 • MEDICAL PSYCHIATRY that minimum lifetime BMI is the strongest prognostic indicator (BMI < 11.5 is associated with an standardised mortality ratio of 4–5). Other indicators of poor prognosis are comorbid BN and atypical demographics (very early or relatively late onset, male gender). Forty per cent of additional deaths are due to suicide, the remainder being due to complications of starvation. Bulimia nervosa The prevalence of BN is difficult to determine with precision, as only a small proportion of sufferers come to medical attention. It is believed to be more common than AN, with a similar gender ratio. Peak age of onset is slightly later than for AN, typically late adolescence or early adult life. Clinical features Patients with BN are usually at or near normal weight (unlike in AN), but display a morbid fear of fatness associated with disordered eating behaviour. They recurrently embark on eating binges, often followed by corrective measures such as self-induced vomiting. Diagnosis Diagnostic criteria are shown in Box 28.32. Physical signs of repeated self-induced vomiting include pitted teeth (from gastric acid), calluses on knuckles (‘Russell’s sign’) and parotid gland enlargement. There are many associated physical complications, including the dental and oesophageal consequences of repeated vomiting, as well as electrolyte abnormalities, cardiac arrhythmias and renal problems (see Box 28.31). Investigations Self-induced vomiting and/or abuse of laxatives and diuretics can lead to clinically significant electrolyte disturbances, including hypokalaemia leading to cardiac arrhythmias. Hence it is good practice to measure urea and electrolytes and obtain an ECG whenever these behaviours are prominent in any patient and when BN is suspected in any medical inpatient. Repeated vomiting can also give rise to Mallory–Weiss tears and even oesophageal rupture; if symptoms are suggestive of these, an endoscopy should be performed. Management Treatment of bulimia with CBT achieves both short-term and long-term improvements. Guided self-help and IPT may also be of value. There is also evidence for benefit from the SSRI fluoxetine, but high doses of up to 60 mg daily may be required for a prolonged period of up to 1 year; this appears to be independent of the antidepressant effect. Prognosis Bulimia is not associated with increased mortality but a proportion of sufferers go on to develop anorexia. At 10-year follow-up, approximately 10% are still unwell, 20% have a subclinical degree of bulimia, and the remainder have recovered. Personality disorders Personality refers to the set of characteristics and behavioural traits that best describes an individual’s patterns of interaction with the world. The intensity of particular traits varies from person to person, although certain ones, such as shyness or irritability, are displayed to some degree by most people. A personality disorder (PD) is diagnosed when an individual’s personality causes persistent and severe problems for the person or for others. beliefs and behaviours that maintain the low weight. Treatment is usually given on an outpatient basis. Inpatient treatment should be reserved for those at risk of death from medical complications or from suicide. There is a limited evidence base for CBT-based psychological treatments. Family behaviour therapy (FBT) has efficacy among adolescent but not adult patients. Psychotropic drugs are of no proven benefit in AN but antidepressant medication may be indicated in those with clear-cut comorbid depressive disorder. Weight gain is best achieved in a collaborative fashion. Compulsory admission and refeeding (including tube feeding) are very occasionally resorted to when patients are at risk of death and other measures have failed. While this may produce a short-term improvement in weight, it rarely changes long-term

prognosis. Prognosis Two-thirds of patients with AN no longer meet diagnostic criteria at 5-year follow-up. However, long-term follow-up studies suggest that many sufferers continue to have a relatively low body mass index (BMI), suggesting that the symptoms do not completely resolve. Approximately 20% of patients develop a chronic, intractable disorder. Long-term follow-up studies demonstrate 28.32 Diagnostic criteria for eating disorders Anorexia nervosa • Weight loss of at least 15% of total body weight (or body mass index ≤ 17.5) • Avoidance of high-calorie foods • Distortion of body image so that patients regard themselves as fat even when grossly underweight • Amenorrhoea for at least 3 months Bulimia nervosa • Recurrent bouts of binge eating • Lack of self-control over eating during binges • Self-induced vomiting, purgation or dieting after binges • Weight maintained within normal limits 28.31 Medical consequences of eating disorders Cardiac • ECG abnormalities: T-wave inversion, ST depression and prolonged QTc interval • Arrhythmias, including profound sinus bradycardia and ventricular tachycardia Haematological • Anaemia, thrombocytopenia and leucopenia Endocrine • Pubertal delay or arrest • Growth retardation and short stature • Amenorrhoea • Sick euthyroid state Metabolic • Uraemia • Renal calculi • Osteoporosis Gastrointestinal • Constipation • Abnormal liver function tests

Management PDs usually persist throughout life and are not readily treated. They typically become less extreme with age but can re-emerge in the context of cognitive decline. Treatment options are limited but there is some evidence that emotionally unstable PD may respond to dialectical behavioural therapy (DBT). Anxious (avoidant) and obsessional (anankastic) PD may benefit from prescription of anxiolytic drugs, while paranoid/schizotypal PD may be improved by treatment with low doses of antipsychotic agents. The problematic and inflexible patterns of interaction that characterise a PD are often apparent in the patient’s interaction with health services and can present a challenge to both the service and the patient. Clear clinical communication supported by robust documentation can help to minimise any potential disruption. Factitious disorder and malingering Factitious disorder describes the repeated and deliberate production of the signs or symptoms of disease to obtain medical care. Pathogenesis It is difficult to understand what motivates a person to act in this way. Several theories have been proposed but the deception that lies at the heart of the condition makes it impossible to gather accurate data from which to draw reliable conclusions. Clinical features The disorder feigned is usually medical but can be a psychiatric illness (for example false reports of hallucinations or symptoms of depression). An example of a medical factitious disorder is dipping of a thermometer into a hot drink to fake a fever. Factitious disorder is uncommon and is important to distinguish from somatoform disorders. A suggested diagnostic algorithm is shown in Figure 28.6. Pathogenesis Some PDs appear to have an inherited aspect (especially schizotypal and paranoid subtypes) but most are more clearly related to an unsatisfactory upbringing and adverse childhood experiences. Clinical features PD can present in various ways. For example, anxiety may be so pronounced that the individual rarely ventures into any situation where they fear scrutiny. Dissocial traits, such as disregard for the well-being of others and a lack of guilt concerning the adverse effects of one’s actions on others, may occur. If pronounced, they may lead to damage to others, to criminal acts or to successful careers, such as in politics. Diagnosis It is possible to classify PD into several subtypes (such as emotionally unstable, antisocial or dependent), depending on the particular behavioural traits in question. A patient who meets diagnostic criteria for one subtype may also meet criteria for others. As allocation to one particular subtype gives little guidance to management or prognosis,

classification is of limited value. Diagnosis requires a longitudinal perspective, with clear evidence that the patient's behavioural traits and pattern of interaction with the world have been present throughout their adult life, have been evident across a range of settings and have caused repeated and persistent problems. It can be difficult to achieve this during a single interview, and most psychiatrists warn against making a diagnosis of personality disorder until the patient has been seen several times and corroborative accounts have been obtained. It is common for PD to accompany other psychiatric conditions, making treatment of the latter more difficult and therefore affecting their prognosis. Fig. 28.6 Diagnosis of medically unexplained symptoms (MUS). Are symptoms fully explained by organic disease? Organic disease Yes 'Medically unexplained' Is patient consciously feigning? No 'Factitious disorder' No 'Malingering' Yes 'Functional somatic syndrome' (see Box 28.11) No 'Somatisation disorder' Yes Is there a preoccupation with symptoms or diagnosis? No Is there an identifiable gain? Yes Diagnosis 'Hypochondriasis' Symptoms 'Somatisation' Multiple presentations to various departments?

 mother and to remain vigilant for development of post-partum depression. Post-partum depression This occurs in 10–15% of women, with onset typically within a month of delivery (although women often suffer for some time before presenting). It can usually be differentiated from postpartum blues by the duration and severity of the symptoms, in particular anhedonia (loss of capacity for pleasure) and negative thoughts. Risk factors include a previous history of depression, a previous history of post-partum depression, antenatal depression and antenatal anxiety. Unlike depression arising at other times, post-partum depression is not more common in lower socioeconomic groups; the prevalence is similar across all social backgrounds. Diagnosis, explanation and reassurance are important. The usual psychological and drug treatments for depression should be considered (p. 1199) to minimise the impact on the mother and child at what is a very important time for both. A number of helpful guidelines are available to inform prescribing decisions. The potential risks to both mother and child should be considered and, if hospital admission is required, it should ideally be to a mother and baby unit. Puerperal psychosis This has its peak onset in the first 2 weeks after childbirth but can arise several weeks later. It is a rare but serious complication affecting approximately 1 in 500 women. There is a strong association with a personal or familial history of bipolar disorder. It usually takes the form of a manic or depressive psychosis but with sudden onset and fluctuation in severity. Delirium is rare with modern obstetric management but should still be considered in the differential diagnosis. Suspiciousness, concealment and impulsivity are common features of puerperal psychosis; hence the risks to both mother and baby are considerable. The clinical priority is to ensure the safety of both mother and baby and so psychiatric admission, ideally to a psychiatric mother and baby unit, is usually necessary. Pharmacological treatment reflects the clinical picture; antipsychotic medication is almost always indicated, augmented by antidepressants if the picture is of psychotic depression and/or by mood stabilisers if the picture is bipolar. Most women recover but the risk of recurrence following subsequent deliveries is 50% and some women will progress to psychotic episodes not associated with childbirth, usually bipolar disorder. Psychiatric disorders during pregnancy Pregnancy can affect the course of psychiatric illnesses and of bipolar affective disorder in particular. Mood-stabilising drugs such as lithium and valproate, which are prescribed for prophylaxis in bipolar disorder (p. 1200), are teratogenic and should be avoided whenever possible. Most guidelines recommend deferring conception until mood-stabilising medication is not required, or replacing the mood stabiliser with an antipsychotic such as chlorpromazine. Furthermore, the immediate post-partum period is associated with a dramatically increased risk of relapse in bipolar

disorder: studies report relapse rates of up to 60% in the first 3 months after delivery in the absence of prophylactic medication. When relapse occurs following childbirth, not only are the stakes higher than at other times but also the onset of illness is more rapid, the symptoms more severe and concealment more pronounced. Post-partum relapse of bipolar affective Münchausen's syndrome This refers to a severe chronic form of factitious disorder. Patients characteristically travel widely, sometimes visiting several hospitals in one day. Although the condition is rare, such patients are memorable because they present so dramatically. The history can be convincing enough to persuade doctors to undertake investigations or initiate treatment, including exploratory surgery. It may be possible to trace the patient's history and show that they have presented similarly elsewhere, often changing name several times. Some emergency departments hold lists of such patients. Malingering Malingering is a description of behaviour, not a psychiatric diagnosis. It refers to the deliberate and conscious simulation of signs of disease and disability for an identifiable gain (patients have motives that are clear to them but which they initially conceal from doctors). Examples include the avoidance of burdensome responsibilities (such as work or court appearances) or the pursuit of financial gain (fraudulent claims for benefits or compensation). Malingering can be hard to detect at clinical assessment but is suggested by evasion or inconsistency in the history. Management Management is by gentle but firm confrontation with clear evidence of the fabrication of illness, together with an offer of psychological support. Treatment is usually declined but recognition of the condition may help to avoid further iatrogenic harm. Puerperal psychiatric disorders There are three important psychiatric presentations following childbirth. When managing these conditions, it is important always to consider both the mother and the baby, and their relationship (Box 28.33). Post-partum blues This is characterised by irritability, labile mood and tearfulness. About 80% of women are affected to some degree. Symptoms begin soon after childbirth, typically peak on about the fourth day and then resolve spontaneously within a few weeks. While the aetiology of baby blues is not fully understood, it is likely to be related to hormonal or physiological changes associated with childbirth. No treatment is required, other than to reassure the 28.33 Psychiatric illness and pregnancy • Psychiatric disorder and pregnancy: always consider effects on mother, fetus and child. • Bipolar disorder: women should have pre-conceptual advice because there is a very high risk of relapse following delivery and some mood stabilisers are teratogenic. • Psychiatric treatments and pregnancy: always make an individual assessment of the risks and benefits taking into consideration effects on mother, fetus and child. • Post-partum low mood ('blues'): weeks 1–3; most cases are transient. • Persistent low mood and anhedonia: may indicate depressive illness. • Puerperal psychosis: progresses rapidly and is an indication for psychiatric admission.

that balances the restrictions imposed. There should also be provisions for appeals and oversight. Further information Books and journal articles Hofer H, Pozzi A, Joray M, et al. Safe refeeding management of anorexia nervosa inpatients: an evidence-based protocol. Nutrition 2014; 30:524–30. Steel RM. Factitious disorder (Munchausen's syndrome). J R Coll Physicians Edinb 2009; 39:343–7. Taylor D, Meader N, Bird V, et al. Pharmacological interventions for people with depression and chronic physical health problems: systematic review and meta-analyses of safety and efficacy. Br J Psychiatry 2011; 198:179–88. Whiteford HA, Degenhardt L, Rehm J, et al. Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. Lancet 2013; 382: 1575–86. Websites cebmh.com Centre for

Evidence-based Mental Health. dementia.ie/images/uploads/site-images/ACE-III_Scoring_(UK).pdf Addenbrooke's Cognitive Examination – 3rd edn (ACE-III): administration and scoring guide. mind.org.uk Information on depression. mocatest.org Montreal Cognitive Assessment. neurosymptoms.org A guide to medically unexplained neurological symptoms. niaaa.nih.gov/ Information on alcoholism. nice.org.uk National Institute for Health and Care Excellence: treatment guidelines for depression. ocdaction.org.uk Useful information about obsessive–compulsive disorder. rcpsych.ac.uk/info/index.htm Royal College of Psychiatrists: mental health information. sign.ac.uk Scottish Intercollegiate Guidelines Network: treatment guidelines for depression, including Guideline 127 – Management of perinatal mood disorders. who.int/mental_health/ World Health Organisation: mental health and brain disorders. www4.parinc.com/ Mini-Mental State Examination. disorder requires urgent specialist treatment, usually comprising admission to a psychiatric mother and baby unit. Ideally, women with major mental disorders such as bipolar affective disorder should be offered expert pre-conception advice to help them make informed decisions about medication and other aspects of their psychiatric care. A comprehensive post-partum risk management plan should be agreed during pregnancy. Psychiatry and the law Medicine takes place in a legal framework, made up of legislation (statute law) drafted by parliament or other governing bodies, precedent built up from court judgements over time (case law), and established tradition (common law). Psychiatry is similar to other branches of medicine in the applicability of common and case law but differs in that patients with psychiatric disorders can also be subject to legislative requirements to remain in hospital or to undergo treatments they refuse, such as the administration of antipsychotic drugs to a patient with acute schizophrenia who lacks insight and whose symptoms and/or behaviour pose a risk to himself/herself or to others. The UK has three different Mental Health Acts, covering England and Wales, Scotland, and Northern Ireland, and all of these have recently been revised. Other countries may have very different provisions. It is important for practitioners to be familiar with the relevant provisions that apply in their jurisdictions and are likely to arise in the clinical settings in which they work. All the countries that make up the UK have also introduced Incapacity Acts in recent years, with detailed provisions covering medical treatments for patients incapable of consenting, whether this incapacity arises from physical or mental illness. In general, the guiding principle in British law is that people should be free to make their own decision about any proposed medical treatment, except where their ability to make and/or communicate that decision is demonstrably impaired (by mental illness or physical incapacity). Any restrictions or compulsions applied should be the minimum necessary, they should be applied only for as long as is necessary, and there should be a benefit to the patient

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