Chapter 13

Organic psychiatric disorders

Organic psychiatric disorders are those with demonstrable pathology or aetiology, or which arise directly from a medical disorder. They are thereby distinguished from all other psychiatric disorders, which are traditionally called *functional*. This distinction is an oversimplification and conceptually flawed as all disorders have biological, psychological and social contributions (Chapter 1). The term causes both practical and semantic problems, but remains in widespread use and so is used here:

- The major organic disorders, *dementia* and *delirium*, are defined, like other psychiatric syndromes, by their characteristic clinical features. However, unlike other syndromes they are known to arise from different diseases with various aetiologies and pathologies. A complete diagnosis requires the disease as well as the dementia syndrome to be identified.
- Other organic disorders are simply psychiatric disorders of any type that appear, in a particular case, to be caused by an identifiable medical condition. Sometimes it is the psychiatric symptoms that first bring the person to medical attention.
- Substance misuse disorders are organic, in that there is a specific pharmacological cause. By convention they are classified separately (Chapter 14).
- Psychiatric disorders which are considered *psychological reactions* to illness—such as becoming depressed after being told you have cancer—are excluded from the organic category.

Dementia

Clinical features of dementia

- Review the core and module approach to assessment of dementia (Chapter 2 and 3).

Dementia is also known as *chronic brain syndrome*. Its cardinal feature is memory impairment (short-term worse than long-term) without impaired consciousness (cf. delirium). The overall clinical profile differs depending on the specific type of dementia, as outlined below, but other common features of dementia are:

- Symptoms present for 6 months, of sufficient severity to impair functioning.
- Personality and behavioural change—e.g. wandering, aggression, disinhibition.
- Dysphasias, dyspraxias and focal neurological signs may be present.
- Psychotic symptoms in half of cases at some stage.
- Unawareness of deficits (except early on).
- Nearly always progressive (though this is not a diagnostic criterion).

The incidence of dementia rises rapidly with age, affecting <5% of 65 year olds and 20% of 80 year olds. (Exact figures depend on severity threshold.) The risk of dementia is doubled in those with an affected first-degree relative. Other risk factors depend on the type of dementia concerned.

- The rapid rise with age is typified by Alzheimer's disease (Figure 13.1).
- Dementia in the under 65s is termed *presenile dementia*.

Differential diagnosis of dementia

Established dementia is usually unmistakeable, but mild dementia can be confused with (or coexist with) other conditions:

- *Depression*. Poor concentration and impaired memory are common in depression in the elderly, hence *pseudodementia*. Two factors which help distinguish depression from dementia are: Did low mood or poor memory come first? Is the failure to answer questions due to lack of ability or lack of motivation?
- *Delirium*. See next section.
- *Deafness*. Check the person can hear.
- *Dysphasia*. Check they can understand and speak.
- *Amnesic syndrome*. A purer short-term memory defect (see below).
- *Late-onset schizophrenia* (paraphrenia). Check for prominent symptoms of psychosis.

The causes of dementia

The common dementias are listed in Table 13.1. Alzheimer's disease accounts for over half of all cases, with the majority of the rest explained by vascular dementia and Lewy body dementia.

- The syndromes are not mutually exclusive, and the common ones often coexist.
- In presenile dementia, a higher percentage of cases are due to rare genetic disorders or severe head injury.

Clinical features differentiating the dementias

Careful clinical evaluation identifies the specific dementia in more than 80% of cases (Table 13.2).

- A distinction is sometimes made clinically between *cortical dementia* (e.g. Alzheimer's disease) and *subocortical dementia* (e.g. Huntington's

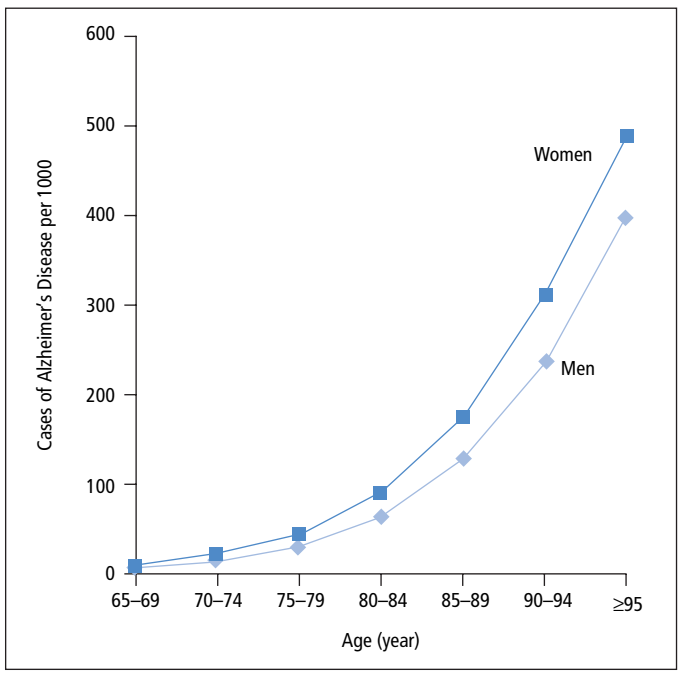

Figure 13.1 The prevalence of Alzheimer's disease with ageing. Adapted from Nussbaum RL, Ellis CE. *New Engl J Med* 2003, **348**, 1356–64.

Table 13.1 Causes of dementia.

Alzheimer's disease (50–60% of cases)
Vascular dementia (20–25%)
Dementia with Lewy bodies (10–15%)
Other causes (<10%)
Degenerative disorders
Frontotemporal dementia
Pick's disease
Huntington's disease
Prion disease
Progressive supranuclear palsy
Metabolic disorders
Alcoholic dementia
Vitamin B12 deficiency
Infections
HIV
Syphilis
Other causes
Normal pressure hydrocephalus
Intracranial tumours
Subdural haematoma
Head injury

disease) (Table 13.3). However, it is better to try and make a specific diagnosis (and the terms are misleading as most dementias affect both brain regions).

- The disorders themselves are discussed in the next section.

Assessment of dementia

In the history and examination, look for clues pointing towards one of the diagnoses in Table 13.2. For example, a history of a fall (?subdural haematoma) or incontinence (?normal pressure hydrocephalus), rapid fluctuations (?dementia with Lewy bodies); on examination you might find hypertension and a carotid bruit (?vascular dementia). Table 13.4 shows the baseline and specialized tests used in the investigation of dementia.

- Only the baseline blood tests would routinely be ordered, with further investigation depending on the age of the patient and the suggestion of a treatable aetiology (for example, a scan to exclude a subdural haematoma). In the absence of such clues, further tests in an elderly person rarely change the diagnosis, let alone reveal a reversible cause.
- Conversely, in a young person, every effort is made to make a diagnosis—not only in case it is treatable and to inform prognosis, but because the dementia may well be inherited and genetic testing available for the family. Presenile dementia is usually referred to neurologists.

Management of dementia

Occasionally, dementia is potentially reversible (e.g. space occupying lesion; B12 deficiency) and responds to appropriate treatment. Specific interventions to treat symptoms in Alzheimer's disease are also now available, as discussed below. However, the management of dementia generally has more modest goals, the main one being to optimize quality of life for patient and carer. Whatever the cause of the dementia, the following principles and strategies are important:

- Think about the patient, their environment, and their carers as potential targets for intervention.
- Characterize and treat the non-cognitive abnormalities. Simple interventions can improve problems such as wandering (e.g. by regular exercise or raising the door handle) and incontinence (e.g. by a toiletting routine). Non-pharmacological methods should always be employed first.
- Psychotic symptoms respond to low-dose antipsychotics. They should be used sparingly and for brief periods only, because psychosis in dementia is usually transient, and because they are associated with more rapid cognitive decline and with sudden death, especially in Lewy body dementia. Atypical antipsychotics may also increase risk of stroke.
- Antipsychotics should *not* be used for non-specific behavioural problems or to sedate people with dementia.
- Treat depression. SSRIs are better tolerated than tricyclics.
- A range of other interventions are also sometimes used, including reality orientation, lavender oil aromatherapy, carbamazepine, reminiscence therapy, music therapy, art therapy, and memory

Table 13.2 Clinical features distinguishing between the dementias.

	Prominent symptoms and signs	Other clinical features
Alzheimer's disease	Memory loss, especially short-term Dysphasia and dyspraxia Sense of smell impaired early Behavioural changes, e.g.wandering Psychotic symptoms at some stage	Relentlessly progressive Survival 5–8 years
Vascular dementia	Personality change Labile mood Preserved insight	Stepwise progression Signs of cerebrovascular disease History of hypertension Commoner in men, smokers
Dementia with Lewy bodies	Fluctuating dementia Delirium-like phases Parkinsonism Visual hallucinations	Antipsychotics worsen condition
Frontotemporal dementia	Stereotyped behaviours Personality change Early loss of insight Expressive dysphasia Memory relatively preserved Early primitive reflexes	Slowly progressive Family history common Commoner in women Onset usually before age 70
Huntington's disease	Schizophrenia-like psychosis Abnormal movements (choreiform) Depression and irritability Dementia occurs later	Presents in the 20s–40s Strong family history
Normal pressure hydrocephalus	Mental slowing, apathy, inattention Urinary incontinence Problems walking (gait apraxia)	Commonest in 50–70 year olds Commonest reversible dementia
Prion disease	Myoclonic jerks Seizures Cerebellar ataxia	Often presenile Rapid onset and progression Death within a year

Table 13.3 Clinical features of subcortical versus cortical dementias.

	Subcortical dementia	Cortical dementia
Memory loss	Moderate	Severe, early
Language	Normal	Affected (Dysphasia)
Personality	Apathetic, inert	Indifferent
Mood	Flat, depressed	Normal
Co-ordination	Impaired	Normal
Motor speed	Slowed	Normal

training. There is some evidence that the first three may be beneficial for behavioural problems.

- Social interventions such as meals-on-wheels, day care and respite admissions help carers cope.
- Investigate any sudden deterioration. It may be due to a treatable cause (e.g. superimposed delirium due to a UTI).
- In advanced dementia, palliative rather than active treatment is often indicated. Next-of-kin should be fully involved in these decisions (see vignette pp. 83–4). Advanced directives are becoming popular and should be respected.

Prognosis of dementia

Most dementias progress inexorably. Life expec-

Table 13.4 Investigation of dementia.

Test	What the test may show
Blood tests	
Full blood count	Macrocytosis, anaemia
Electrolytes	Hypercalcaemia, renal disease
Liver function tests	Alcoholic liver disease
Thyroid function	Hypothyroidism
Vitamin B12 and folate levels	Deficiencies can produce dementia
Syphilis serology	Now rare, but overlooked
HIV test	Dementia common in AIDS
Radiography	
Chest X-ray	Bronchial carcinoma with ?cerebral metastases
Brain imaging (CT or MRI)	Tumour; infarcts; haematoma; temporal lobe atrophy suggests Alzheimer's
EEG	Characteristic abnormality in prion disease (3 Hz 'spike and wave')
Other tests	
Lumbar puncture	Normal pressure hydrocephalus; herpes encephalitis
Cerebral blood flow studies	Parietal hypometabolism suggests Alzheimer's
Neuropsychological testing	Assess severity; profile of deficits may point to brain region most affected
Genetic testing	Available for some familial dementias—an area of rapid developments
Brain biopsy	Very rarely done; mainly for suspected prion disease

tancy is decreased, though the reasons are not clear. Death is usually within 5 to 8 years of onset, with some dementias, notably prion disease, progressing much more rapidly. Younger cases and those with focal neurological signs or psychotic symptoms have a worse prognosis.

- People with dementia should not be blood or tissue donors in view of the risk of transmission of prion disease.

Specific dementing disorders

This section summarizes the aetiological, pathological and therapeutic factors for the important dementias.

- Their main distinguishing clinical features are summarized in Table 13.2.

Alzheimer's disease

As the commonest dementia, it is fortunate that substantial progress has been made recently in understanding Alzheimer's disease, and the first specific treatments are now available.

It is a neuropathological diagnosis. The cardinal features are neurofibrillary tangles and senile (amyloid) plaques in the hippocampus and cerebral cortex. Loss of synapses is also important. Neurochemically, the main abnormality is a loss of acetylcholine due to degeneration of the cholinergic neurons of the basal forebrain. The cholinergic deficits and synaptic loss are proportional to the severity of the dementia.

A considerable amount is now known about the causes of the disease (Table 13.5).

- The major risk factor, apart from age, is the E4 (epsilon4) variant of the apolipoprotein E (apoE) gene. Three polymorphisms exist, apoE2, apoE3 and apoE4: 20% of the population carry one copy of apoE4 and have twice the risk of Alzheimer's disease; the 3% who are apoE4 homozygotes (i.e. two copies of apoE4) are at several-fold increased risk. Conversely, the risk is ~40% lower in people without an apoE4 allele, and those with apoE2 alleles may be at lower risk still. The effect of apoE4 is probably that it brings forward the age of onset of dementia, by about a decade. (Note that apoE4 is neither necessary nor sufficient for Alzheimer's disease—it is a risk factor.)

Table 13.5 Risk factors for Alzheimer's disease.

Factor	Comments
Genetic	
Apolipoprotein E (chromosome 19)	ApoE4 allele markedly increases risk
Amyloid precursor protein (chromosome 21); presenilins 1 and 2 (chromosomes 14 and 1)	Autosomal dominant mutations which cause early-onset familial Alzheimer's disease
Other genes (chromosomes 6, 10 and 11)	Identity and contribution uncertain
Down's syndrome (trisomy 21)	Alzheimer's disease occurs in middle age
Other risk factors	
Increasing age	Predominant risk factor (Fig. 13.1)
Females	Slightly higher risk than men (Fig. 13.1)
Homocysteinaemia	High plasma homocysteine doubles risk
Head injury	Doubles risk of Alzheimer's disease
Latent herpes simplex infection	In people with an apoE4 allele
History of depression	Risk of dementia may be increased
Aluminium exposure	Controversial
Environmental—protective factors	
High educational level	'Cerebral reserve'
Physically and mentally active lifestyle	'Use it or lose it'
Non-steroidal anti-inflammatories	?Retard a chronic brain inflammatory process
Statins	?Decrease atherosclerotic risk factors
Hormone replacement therapy	?Oestrogens are neuroprotective (but see text)

- Presenile Alzheimer's disease is often familial and (unlike most cases) caused by an autosomal dominant mutation. Three genes have been found so far, with several different mutations in each: amyloid precursor protein (APP), and presenilin (PS) 1 and 2. Though extremely rare, these cases helped identify the pathways involved in Alzheimer's disease generally (see below).
- A history of treatment with NSAIDs, HRT or statins has been associated with a lower risk of developing dementia and Alzheimer's disease. Plausible mechanisms exist to explain these effects. However, it is unclear if the association is genuine, or reflects other factors (e.g. women given HRT tend to be healthier and better educated).
- Prospective studies show that plasma homocysteine is an independent, graded risk factor for developing Alzheimer's disease several years later. This may be due to its role in atherosclerotic processes.

The key process leading to Alzheimer's disease is abnormal metabolism of β-amyloid, the main constituent of senile plaques, and a fragment of the amyloid precursor protein. The abnormality results in formation of insoluble forms of β-amyloid which accumulate and trigger a range of other harmful biochemical events. This 'amyloid cascade' is promoted in various ways by the causal factors mentioned (Figure 13.2). For example, NSAIDs and apoE4 both affect β-amyloid trafficking or processing.

- The central role of β-amyloid is supported by findings in transgenic mice with Alzheimer's disease-causing mutations in amyloid precursor protein gene. They become demented and get senile plaques, a fate which does not afflict normal rodents.
- Abnormalities in tau protein (which forms neurofibrillary tangles), oxidative stress and inflammatory processes may also be important. Their connection to β-amyloid is unclear.

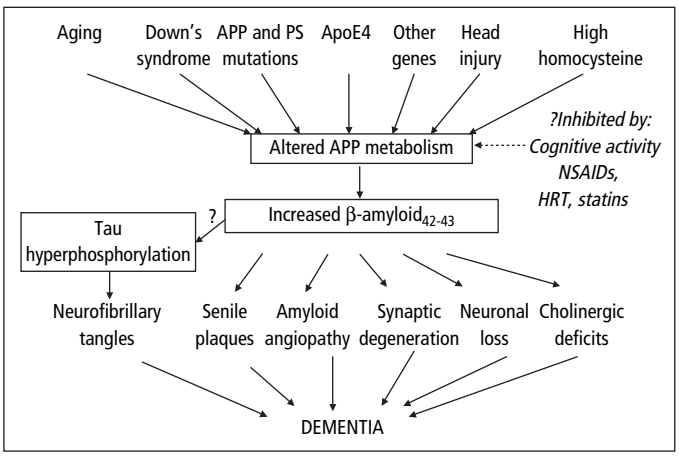

Figure 13.2 The pathogenesis of Alzheimer's disease.

Treatment of Alzheimer's disease

The first specific treatments for Alzheimer's disease are now available, and many more are in development.

- Cholinesterase inhibitors (e.g. donepezil) are licensed for mild and moderate Alzheimer's disease. They have modest effects against cognitive symptoms, global outcome and activities of daily living. The effect is roughly equivalent to a 6-month delay in cognitive decline. They may also have benefit for behavioural symptoms. In the UK, they must be prescribed by a specialist according to specified diagnostic and severity criteria.
- Memantine is an NMDA glutamate receptor antagonist. It has some efficacy in more severe Alzheimer's disease. Its therapeutic value in practice is not yet established.
- Various treatments directed at β-amyloid and its metabolism are being tested. These include the surprising finding that active and passive immunization against β-amyloid may be effective.
- Treatment trials with NSAIDs and folic acid (to lower homocysteine) are underway. Trials of HRT have been negative.
- CSF and MRI markers of the disease and its progression are being developed and may soon become useful either for diagnosis or for monitoring response to treatment.
- Any potential preventative or disease-retarding intervention must take into account the fact that the pathology begins in earnest at least a decade before the first symptoms.

Vascular dementia

An umbrella term for dementia thought to be vascular in origin. One type is caused by multiple small infarcts (hence the earlier name of *multi-infarct dementia*). Others include 'small vessel disease' and Binswanger's disease. It is associated with risk factors for atherosclerosis and cerebrovascular disease.

- There is no specific treatment, other than to attend to the cerebrovascular risk factors (e.g. aspirin, smoking).
- Vascular impairment acts synergistically with Alzheimer's disease to produce dementia.

Dementia with Lewy bodies

Also called *Lewy body disease*, it is the third commonest form of dementia. The overlap with Parkinson's disease reflects their common pathology, viz. Lewy bodies (intracellular inclusions made of α-synuclein), but in dementia with Lewy bodies these occur in the cerebral cortex. Its aetiology is unknown, and no genes have been identified.

- Cholinesterase inhibitors are valuable (and more effective than in Alzheimer's disease).
- Antipsychotics should be avoided because of

the risk of sensitivity reactions and increased mortality.

Parkinson's disease dementia

Dementia with Lewy bodies merges into *Parkinson's disease dementia*. The convention is that the latter category is used for dementia occurring more than 12 months after onset of parkinsonism. By this definition, dementia occurs in about 40% of cases of Parkinson's disease, especially later onset cases.

- L-DOPA does not improve the dementia; cholinesterase inhibitors may.
- Clozapine is useful for psychotic symptoms.

Frontotemporal dementia

A spectrum of conditions affecting the frontal and temporal lobes, including *Pick's disease* (characterized by Pick bodies and Pick cells), and dementias associated with motor neuron disease. Causes about ~5% of dementia, more so in younger subjects.

- The diseases are all thought to involve abnormalities in tau protein (see Alzheimer's disease). A few cases are caused by mutations in the tau gene on chromosome 17.
- There is no specific treatment. Patients are very sensitive to many psychotropic drugs, which should be used with caution to treat depressive or psychotic symptoms.

Huntington's disease

Also called *Huntington's chorea*. Unlike most psychiatric disorders, it is entirely genetic. It is autosomal dominant, caused by a trinucleotide repeat expansion ('molecular stutter') in the huntington gene on chromosome 4. Pathologically, there is marked atrophy of the caudate nucleus, and later in the frontal lobe. The abnormal huntingtin protein is though to be neurotoxic. Genetic testing is possible.

- The psychosis and depression can be treated symptomatically, but the dementia, and the disease itself, remain untreatable.

Normal pressure hydrocephalus

Accounts for up to 5% of dementia, and the commonest potentially reversible type. The term is a misnomer as CSF pressure is often increased. The clinical triad (Table 13.2) is characteristic, and MRI or CT scanning may help diagnostically.

- Treatment is by ventricular shunting; 50% respond well.

Prion disease

Includes *Creutzfeldt–Jakob disease (CJD)*. Prion disease is caused by an abnormal form of a protein called prion protein. The abnormality can be inherited or acquired, because abnormal prion protein is transmissible, via blood, diet and contaminated surgical instruments. Prion disease is exceedingly rare but notable, especially in the UK, because of 'variant CJD (vCJD)', acquired from cows with bovine spongiform encephalopathy (BSE)—prion disease in cattle.

- About 150 cases of vCJD have been reported (November 2004), and the current best predictions are for hundreds or thousands more. Unlike typical CJD, these have occurred in young adults and present with prominent psychiatric symptoms, especially depression and personality change.
- Any suspected case of prion disease should be referred to the National Surveillance Unit in Edinburgh.

Alcoholic dementia

True alcoholic dementia is rare. Visuospatial deficits are often prominent. It is associated with atrophy of the white matter and frontal lobes.

- Dementia in those with alcohol dependence is more often due to Alzheimer's disease or vascular dementia.
- Alcoholic dementia does not usually improve with abstinence.
- Alcohol dependence also causes amnesic syndrome (see below).

Delirium

Also known as *acute confusional state* or *acute brain syndrome*. It is common on medical and surgical wards—a third of elderly patients in hospital have an episode of delirium—so all doctors should be able to recognize and manage it.

- Review the unresponsive patient and cognition modules (Chapter 3).

Clinical features of delirium

Clouding of consciousness is the most important diagnostic sign. It refers to drowsiness, decreased awareness of surroundings, disorientation in time and place, and distractibility. At its most severe the patient may be unresponsive, but more commonly the impaired consciousness is quite subtle.

- Minor degrees of impaired consciousness can be detected by problems estimating the passage of time (e.g. how long the interview has been going on), and with concentration tasks (e.g. counting from 20 down to 1).

Because clouding of consciousness may not be apparent, the first clue to the presence of delirium is often one of its other features:

- Fluctuating course, worse at night.
- Visual hallucinations.
- Transient persecutory delusions.
- Irritability and agitation, or somnolence and decreased activity.
- Impaired concentration and memory.

The differential diagnosis includes dementia (Table 13.6), acute psychosis, and depression. Usually the clinical picture (especially the acute onset and rapid fluctuations), and its context, is sufficiently characteristic to make delirium a relatively simple diagnosis. However, differentiating delirium from Lewy body dementia can be difficult without a good history.

Table 13.6 Delirium versus dementia.

	Delirium	Dementia
Onset	Acute	Insidious
Short-term course	Fluctuating	Constant
Attention	Poor	Good
Delusions and hallucinations	Common, simple, fleeting	Less common, more stable

Table 13.7 Common causes of delirium.

Prescribed drugs
Tricyclic antidepressants
Benzodiazepines and other sedatives
Digoxin
Diuretics
Lithium
Steroids
Opiates
Other drugs
Alcohol intoxication
Alcohol withdrawal and delirium tremens
Medical conditions
Postoperative hypoxia
Febrile illness
Septicaemia
Organ failure (cardiac, renal, hepatic)
Hypoglycaemia
Dehydration
Constipation
Burns
Major trauma
Neurological conditions
Epilepsy (postictal)
Head injury
Space occupying lesion
Encephalitis

Aetiology of delirium

Recognition of delirium is followed by an urgent search for its cause (Table 13.7).

- Medication is implicated in a third of cases, mostly drugs with anticholinergic effects (e.g. tricyclics) or sedatives.

Several demographic factors predict those who are at high risk of developing delirium (Table 13.8). In these individuals, delirium can follow relatively trivial precipitants (e.g. constipation).

Table 13.8 Predisposing factors for delirium.

Elderly
Male
Pre-existing dementia
Pre-existing frailty or immobility
Previous episode of delirium
Sensory impairment

Table 13.9 Management of delirium.

Environmental components
Quiet surroundings (side room), constant lighting, clock, calendar
Regular routine
Clear simple communications
Limit numbers of staff (e.g. key nurse)
Involve family
Medical components
Monitor vital signs
Investigate and treat underlying cause (e.g. antibiotics, oxygen, stop drug)
Control agitation or psychotic symptoms with antipsychotics

Management of delirium

Delirium is managed where it occurs—usually in general hospitals. Psychiatrists may be asked to assess the patient, make the diagnosis and give advice. Treatment is directed both at the symptoms and at the cause, and includes both medical and environmental interventions (Table 13.9).

- In practice (and in an exam), emphasize both the need to search for a cause and for environmental steps whilst this is ongoing. The latter may avoid the need for medication, which can complicate the problem, and should only be used when necessary.
- Antipsychotics are the first-line pharmacological treatment. Haloperidol is often used, by intramuscular injection if it cannot be taken orally. It can be given intravenously but this is rarely required.
- The exception is for alcohol- or seizure-related delirium, when a benzodizaepine (e.g. lorazepam) is indicated (because of the epileptogenic potential of antipsychotics).
- A delirious person may occasionally be a risk to self, other patients, or staff. Call for help, ensure safety, and use physical restraint if essential (e.g. to allow drug to be administered).
- Patients with delirium are often incapable of giving informed consent. Treatment is therefore given under the common law. If continuing interventions without consent are anticipated, the Mental Health Act may be required.

Prognosis of delirium

Prognosis depends on the cause. Within a week the patient is usually better or has died. A quarter have died by 3 months. There is no good evidence that delirium progresses to dementia (although pre-existing dementia is a risk factor for delirium).

A 75-year-old lady was found lying on the floor and taken to hospital. She is drowsy, disorientated in time and place, distractible, and unable to give any history. She thinks you are trying to kill her. She is febrile and hypotensive, but has no neurological signs or injuries. Blood tests and X-rays are performed. She is given oxygen, and antibiotics for the clinical suspicion of septicaemia. Her agitation worsens but settles with haloperidol. The GP tells you that there is no past history of note. Blood cultures grow an organism sensitive to the antibiotic. Her condition improves over 72 hours. The haloperidol is tailed off and her cognitive function returns to normal.

Other organic disorders

Dementia and delirium have been described in detail because they are common. As mentioned at the start of the Chapter, there are many other 'organic' conditions which can together present with the whole range of psychiatric disorders. The important principles, and some specific examples, are summarized here.

Organic psychiatric disorders

The diagnostic rule is to preface the psychiatric label with 'organic' and state the aetiology (Table

Table 13.10 Organic psychiatric disorders.

Syndrome	Example of cause
Organic brain syndromes	Dementia, delirium, amnesic syndrome
Organic delusional (psychotic) disorders	Systemic lupus erythematosus
Organic mood disorders	Multiple sclerosis
Organic anxiety disorders	Thryotoxicosis
Organic personality disorders	Head injury

13.10). For example, 'Organic anxiety disorder due to thyrotoxicosis'.

Each organic syndrome is very rare compared to its 'functional' counterpart. In areas with good primary care, psychiatrists rarely see undiagnosed organic psychiatric disorders. However, when an organic syndrome does occur it is essential to recognize it. Detecting an organic disorder requires that you:

- Consider the possibility, with every patient. If this is done, it is easier not to forget to take a brief medical history and conduct a relevant physical examination and order investigations.
- Always include an organic disorder on your list of differential diagnoses in an exam situation.
- Be suspicious if aspects of the psychiatric presentation are unusual. For example, organic syndromes often produce abnormalities in unexpected functions—e.g. anosmia in depression due to a frontal meningioma.

There may or may not be an effective treatment for the organic disorder. Regardless, the psychiatric symptoms are still treated with the appropriate pharmacological, psychological and social interventions.

- As a rule, treatment response in an organic disorder is similar to that of its functional equivalent; for example, depression responds to antidepressants whether it is organic or not. The presence of the organic disorder may, however, affect the choice of drug (e.g. avoid TCAs in depression following myocardial infarction).

Amnesic syndrome

Amnesic (or *amnestic*) syndrome completes the triad of conditions (with dementia and delirium) which affect memory and which always have an organic cause. Its features are

- Selective loss of recent memory.
- Confabulation: the unconscious fabrication of recent events to cover gaps in memory.
- Time disorientation.
- Attention and immediate recall intact.
- Long-term memory and other intellectual faculties intact.

Amnesic syndrome is rare, and in practice difficult to distinguish from some dementias. It is due to damage to the mammillary bodies, hippocampus or thalamus. The usual cause is alcohol-induced thiamine deficiency (*Korsakov's syndrome*), which is treated with thiamine and abstinence. Other causes include herpes simplex encephalitis, severe hypoxia and head injury.

The memory deficits are often irreversible.

Epilepsy

There are several main types of epilepsy (Table 13.11). Epilepsy is usually managed by neurologists, but has many psychiatric aspects. Only the latter are covered here (Table 13.12).

- Consult a neurology text for general coverage of epilepsy.

Relationship of psychiatric symptoms with seizures

Complex partial epilepsy was called *psychomotor epilepsy* because of the frequency of psychiatric symptoms during seizures (Table 13.13). It has also been referred to as *temporal lobe epilepsy*, though this is not always the site of the seizure focus.

- The risk of schizophrenia is several-fold higher in people with complex partial epilepsy, more so if the focus is in the left temporal lobe, and due to an early developmental abnormality.
- The association with sexual dysfunction may be

Table 13.11 The main forms of epilepsy.

Type	Comments	Conscious level during seizure
Generalized seizures	No focal onset	
Tonic–clonic (Grand mal)	The 'classic' type of seizure	Unconscious
Absence (Petit mal)	Subtle and brief	Impaired
Partial seizures	Focal onset	
Complex partial (psychomotor)	Of most psychiatric significance	Impaired
Simple partial		Unaffected

Table 13.12 Psychiatric aspects of epilepsy.

Category	Example
Psychiatric symptoms related to seizures	
Due to shared aetiology	Temporal lobe tumour
At start of seizure	Hallucinations during aura
During seizure	Non-convulsive status presenting as a fugue state
After seizure	Postictal delirium
Between seizures	Psychosis of complex partial epilepsy
Psychiatric disorder masquerading as epilepsy	Pseudoseizures
Psychiatric disorder associated with epilepsy	
Depression	Common in people with epilepsy
Suicide	Several times more common in people with epilepsy
Psychiatric problems of treatment	
Side-effects of anticonvulsants	Depression with barbiturates
Seizures as medication side-effect	Antipsychotics and tricyclic antidepressants

due to the epilepsy, or the medication. Psychiatric presentations can occur with other epilepsies, but are much rarer.

- A*bsence seizures* in children produce transient lapses in concentration or simple automatisms and can be mistaken for a behavioural disorder.
- A *generalized seizure disorder* can present to a psychiatrist if, for example, the person was found wandering in a postictal delirium.

Psychiatric disorders masquerading as epilepsy

Pseudoseizures (hysterical seizures or *non-epileptic attack disorder)* is a form of dissociative disorder (p. 107). It can be hard to distinguish clinically from a true seizure. EEG monitoring during attacks may be required to make the diagnosis. Pseudoseizures are commoner in people who also have epilepsy.

- Other conditions which can be misdiagnosed as epilepsy include panic attacks, hypoglycaemia and schizophrenia. In children, consider temper tantrums and nightmares.

Psychological problems associated with having epilepsy

Historically, epilepsy has been attributed to demonic possession and its sufferers seen as irrita-

ble, self-centred people with criminal tendencies. Although entirely false, persisting negative attitudes, acting in concert with the real disabilities, probably explain the higher incidence of psychiatric disorders and suicide in epilepsy.

- The commonest psychiatric disorders are anxiety and depressive disorders.
- The suicide risk is increased 5-fold, and more so in those with complex partial seizures.
- Anticonvulsant treatments can compound the psychiatric problems—phenobarbitone causes hyperactivity and irritability in children; phenytoin can produce ataxia and delirium.

Table 13.13 Psychiatric symptoms of complex partial seizures.

During the seizure (ictal)
Impaired consciousness
Hallucinations and other distorted perceptions
olfactory
somatic—especially epigastric
Sense of ***déjà vu***
Depersonalization and derealization
Speech and memory affected
Automatisms and stereotyped behaviour
After the seizure (Postictal, hours to days)
Transient, florid psychosis
Between seizures (interictal)
Schizophrenia-like psychosis
Depression
Sexual dysfunction and lack of libido

Head injury

Head injury is a major cause of organic psychiatric syndromes in young adults. They can be difficult to treat and services are greatly under-resourced.

- The psychiatric consequences of a head injury depend partly on the nature and location of the injury, and partly on the person's premorbid characteristics. In blunt trauma, the brain suffers contusions at the point of impact, focal damage elsewhere as the brain reverberates in the skull and diffuse axonal damage due to the shearing forces.
- People with an apoE4 allele are at greater risk of persistent deficits following head injury.

A wide range of psychiatric problems can follow a head injury:

- A head injury with loss of consciousness may produce amnesia, either *anterograde* (post-traumatic), for events after the injury, or *retrograde*. Anterograde amnesia >24 hours predicts a poor

Table 13.14 Other medical disorders with psychiatric manifestations.

Medical disorder	Psychiatric manifestations
Cerebral tumour	Psychiatric symptoms in 50%, more so if tumour is in frontal or temporal lobes
Cerebral abscess	May present with psychiatric symptoms
Multiple sclerosis	Mood disturbance in 25%—bland euphoria or depression; cognitive deficits in 25%—can become severe; personality changes in 25%—apathy, irritability
Parkinson's disease	Depression in 50%, L-DOPA therapy may cause psychosis
Systemic lupus erythematosus	5% at presentation, 50% at some stage: mood disorder, psychosis, delirium and seizures
HIV	Dementia in 30% with AIDS; psychosis—prevalence uncertain
Cushing's disease	Severe depression common; occasionally psychosis and cognitive impairment
Addison's disease	Apathy and fatigue in 80%; depression in 50%; cognitive impairment in 50%; psychosis in 5%
Hyperthyroidism	Anxiety (common), depression, mania, delirium
Hypothyroidism	Mental slowing and depressive symptoms very common; rarely 'myxoedema madness'—delirium, depression, dementia; symptoms may persist despite thyroxine replacement
Hypercalcaemia	Psychosis, delirium and mood disorders in 50%; cognitive impairment in 25%

long-term outcome, including persistent cognitive deficits. The impairment ranges from a subtle slowing of thought and distractibility through to dementia. It tends to improve in the first year but thereafter deficits are likely to be permanent. There is no specific treatment.

- *Personality changes* are frequent. Typically the changes are indicative of frontal lobe damage: impaired ability to plan or persevere; emotional shallowness and lability; impulsivity and irritability. Altered sexual behaviour (in any direction) and long-winded speech also occur. Improvement is partial and slow.
- *Mood disorder, anxiety disorders* and *schizophrenia* are commoner than expected after head injury. Left- and right-sided frontal lobe damage are associated with depression and mania respectively.
- *Postconcussional syndrome* describes emotional, cognitive and bodily symptoms occurring after relatively minor head injury. The symptoms are sometimes thought to be 'psychological' or even feigned, related to hopes of compensation but may reflect subtle brain injury.

Other medical disorders associated with psychiatric disorders

Table 13.14 lists some medical disorders that often have psychiatric manifestations.

Key points

- Organic psychiatric disorders are those due to a recognized medical cause or pathology. Dementia and delirium are by definition organic disorders; all other psychiatric disorders can be. Hence consider an organic cause for every psychiatric presentation—epilepsy and endocrine disorders are classic culprits.
- Dementia is characterized by memory loss. It is usually insidious and progressive, sometimes reversible. The commonest causes are Alzheimer's disease, vascular dementia and dementia with Lewy bodies.
- Delirium is an acute, fluctuating confusional state, with clouding of consciousness. It has many causes (e.g. drugs, septicaemia). It is usually treatable.
- Treatment of organic disorders is aimed at the underlying cause ***and*** at the psychiatric symptoms.