

occlusion of a retinal artery. It is painless and lasts less than 60 minutes. It is usually caused by an embolus from an atheromatous carotid artery in the neck. The most common emboli are cholesterol emboli, which usually arise from an ulcerated plaque.<sup>7</sup> Other causes include emboli from the heart, temporal arteritis and benign intracranial hypertension. Other symptoms or signs of cerebral ischaemia, such as transient hemiparesis, may accompany the symptom. The source of the problem should be investigated. The risk of stroke after an episode of amaurosis fugax appears to be about 2% per year.<sup>7</sup>

## Transient ocular ischaemia

Unilateral loss of vision provoked by activities such as walking, bending or looking upwards is suggestive of ocular ischaemia.<sup>7</sup> It occurs in the presence of severe extracranial vascular disease and may be triggered by postural hypotension and ‘stealing’ blood from the retinal circulation.

## Retinal detachment<sup>9</sup>

Retinal detachment may be caused by ocular trauma, thin retina (myopic people), previous surgery (e.g. cataract operation), choroidal tumours, vitreous degeneration or diabetic retinopathy.

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### Clinical features

- Sudden onset of floaters, flashes or black spots
- Blurred vision in one eye becoming worse
- A dark shadow peripherally, progressing centrally over days/weeks
- Partial or total loss of visual field (total if macula detached)

Ophthalmoscopy may show detached retinal fold as large grey shadow in vitreous cavity.

### Management

- Immediate referral for sealing of retinal tears
- Small holes treated with laser or freezing probe
- Pneumatic retinopexy is an option
- True detachments usually require surgery

## Vitreous haemorrhage

Haemorrhage may occur from spontaneous rupture of vessels, avulsion of vessels during retinal

traction or bleeding from abnormal new vessels.<sup>6</sup> Associations include ocular trauma, diabetic retinopathy, tumour and retinal detachment.

## Clinical features

- Sudden onset of floaters or ‘blobs’ in vision
- May be sudden loss of vision
- Visual acuity depends on the extent of the haemorrhage; if small, visual acuity may be normal

Ophthalmoscopy may show reduced light reflex: there may be clots of blood that move with the vitreous (a black swirling cloud).

## Management

- Urgent referral to exclude retinal detachment
- Exclude underlying causes such as diabetes
- Ultrasound helps diagnosis
- May resolve spontaneously
- Bed rest encourages resolution
- Surgical vitrectomy for persistent haemorrhage

## ⌚ Central retinal artery occlusion

The cause is usually arterial obstruction by atherosclerosis, thrombi or emboli. There may be a history of TIAs. Exclude temporal arteritis (immediately measure ESR).

## Clinical features

- Sudden loss of vision like a ‘curtain descending’ in one eye (same as amaurosis fugax, but doesn’t resolve)
- Vision not improved with 1 mm pinhole
- Usually no light perception

## Ophthalmoscopy

- Initially normal
- May see retinal emboli or pale swelling

- Classic ‘red cherry spot’ at macula

## Management

Urgent referral to an ophthalmologist but if seen early, or ophthalmologist delayed, use this procedure within 30 minutes:

- massage globe digitally through closed eyelids (use rhythmic direct digital pressure for at least 5 minutes)—may dislodge embolus distally
- rebreathe carbon dioxide (paper bag) or inhale special CO<sub>2</sub> mixture (carbogen)
- intravenous acetazolamide (Diamox) 500 mg
- refer urgently (less than 6 hours)—exclude temporal arteritis

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Prognosis is poor. Significant recovery is unlikely unless treated immediately (within 30 minutes).

## ⌚ Central retinal vein thrombosis

Thrombosis is associated with several possible factors, such as hypertension, diabetes, thrombocytopenia, glaucoma and hyperlipidaemia. It usually occurs in the elderly.

### Clinical features

- Sudden loss of central vision in one eye (if macula involved): can be gradual over days
- Vision not improved with 1 mm pinhole

Ophthalmoscopy shows swollen disc and multiple retinal haemorrhages, ‘stormy sunset’ appearance.

## Management

Refer to an ophthalmologist. No immediate treatment is effective. The cause needs to be found first and treated accordingly. Some cases respond to fibrinolysis treatment. Laser photocoagulation may be necessary in later stages if neovascularisation develops, to prevent thrombotic glaucoma. Intravitreal injection of a monoclonal antibody is also an option.

## ⌚ Macular degeneration

There are two age-related types: exudative or ‘wet’ (acute), and pigmentary or ‘dry’ (slow onset).

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- ‘Wet’ MD is caused by choroidal neovascular membranes that develop under the retina of the macular area and leak fluid or bleed. It is a serious disorder.

- ‘Dry’ MD (9 out of 10 cases of MD) develops more slowly and is always painless.
- More common with increasing age (usually over 60), when it is termed ‘age-related MD’, and in those with myopia (relatively common).
- May be familial.

## Clinical features

- Sudden fading of central vision (see FIG. 66.6 )
- Distortion of vision
- Straight lines may seem wavy and objects distorted
- Use a grid pattern (Amsler chart): shows distorted lines
- Central vision eventually completely lost
- Peripheral fields normal



**FIGURE 66.6** Appearance of a subject through the eyes of a person with age-related macular degeneration

*Photo courtesy Allergan Pharmaceuticals*

## Ophthalmoscopy

- White exudates, haemorrhage in retina
- Macula may look normal or raised

## Management

No treatment is available to reverse MD. However ‘wet’ MD should be referred urgently for treatment to slow its progression: regular intravitreal injection of antivascular endothelial growth factor drugs (e.g. ranibizumab, bevacizumab) into the vitreous humour.<sup>10</sup> The Age-Related Eye Disease Study provided confirmatory evidence that the chronic pigmentation type responds to free-radical treatment with the antioxidants vitamins A, C, E and zinc using beta-carotene, 15 mg; vitamin C, 500 mg; vitamin E, 400 IU; and 80 mg zinc oxide.<sup>11</sup> Advise patient to cease smoking if applicable.<sup>12</sup> Low vision aids may be beneficial.

## Drusen

Drusen are small yellow deposits under the retina composed of lipids, a fatty protein. They are part of ageing and harmless per se, but having drusen increases a person’s risk of developing dry MD.

## Temporal arteritis

With temporal arteritis (giant cell arteritis) there is a risk of sudden and often bilateral occlusion of the short ciliary arteries supplying the optic nerves, with or without central retinal artery involvement.<sup>13</sup>

### Clinical features<sup>14</sup>

- Usually older person: over 65 years
- Sudden loss of central vision in one eye (central scotoma)
- Can rapidly become bilateral
- Associated temporal headache or jaw claudication (around 60%)
- Temporal arteries tender, thickened and non-pulsatile (around 30%)
- Visual acuity—blurred, diplopia; severely impaired (around 20%)
- Afferent pupil defect on affected side
- Usually elevated ESR >40 mm

Ophthalmoscopy shows optic disc swollen at first, then atrophic. The disc may appear quite normal.

### Management

- Other eye must be tested
- Immediate corticosteroids (60–100 mg prednisolone daily for at least 1 week)

- Biopsy temporal artery (if there is a localised tender area)

## Retinal migraine

Migraine may present with symptoms of visual loss ('aura'). Associated headache and nausea may be absent.

### Clinical features

- Zigzag lines or lights
- Multicoloured flashing lights
- Unilateral or bilateral field deficit
- Resolution within a few hours

## ⌚ Posterior vitreous detachment

The vitreous body collapses and detaches from the retina. It may lead to retinal detachment.

[Page 806](#)

### Clinical features

- Sudden onset of floaters
- Visual acuity usually normal
- Flashing lights indicate traction on the retina

### Management

- Refer to an ophthalmologist urgently.
- An associated retinal hole or detachment needs exclusion.

## ⌚ Optic (retrobulbar) neuritis

Causes include multiple sclerosis, neurosyphilis and toxins. A significant number of cases eventually develop multiple sclerosis.

### Clinical features

- Usually a woman 20–40 years (multiple sclerosis)
- Loss of vision in one eye over a few days

- Retro-ocular discomfort with eye movements
- Variable visual acuity
- Usually a central field loss (central scotoma)
- Afferent pupil defect on affected side

## Ophthalmoscopy

- Optic disc swollen if ‘inflammation’ anterior in nerve
- Optic atrophy appears later
- Disc pallor is an invariable sequel

## Management

- Test visual field of other eye
- MRI
- Most patients recover spontaneously but are left with diminished acuity
- Intravenous steroids hasten recovery and have a protective effect against the development of further demyelinating episodes

## Corneal disorders

People with corneal conditions typically suffer from ocular pain or discomfort and reduced vision. The common condition of dry eye may involve the cornea while contact lens disorders, abrasions/ulcers and infections are common serious problems that threaten eye sight.

Inflammation of the cornea—keratitis—is caused by factors such as ultraviolet light, e.g. ‘arc eye’, herpes simplex, herpes zoster ophthalmicus and the dangerous ‘microbial keratitis’.

Bacterial keratitis is an ophthalmological emergency that should be considered in the contact lens wearer presenting with pain and reduced vision. Topical corticosteroids should be avoided in the undiagnosed red eye.

Refer to [CHAPTER 40](#) for corneal lesions.

## Pitfalls

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- Mistaking the coloured haloes of glaucoma for migraine.
- Failing to appreciate the presence of retinal detachment in the presence of minimal visual impairment.

- Omitting to consider temporal arteritis as a cause of sudden visual failure in the elderly.
- Using eyedrops to dilate the pupil (for fundal examination) in the presence of glaucoma.

## When to refer

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- Most problems outlined need urgent referral to an ophthalmologist, especially retinal detachment.
- Acute visual disturbance of unknown cause requires urgent referral.
- Any blurred vision—sudden or gradual, painful or painless—especially if 1 mm pinhole fails to alter visual acuity.
- Refer all suspicious optic discs.
- Cataracts when visual impairment seriously affects everyday activities.

### Practice tips

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- Tonometry is advised routinely for all people over 40 years; those over 60 years should have tests every 2 years.
- Any family history of glaucoma requires tonometry from 40 years onwards.
- Sudden loss of vision in the elderly suggests temporal arteritis (check the ESR and temporal arteries). It requires immediate institution of high-dose steroids to prevent blindness in the other eye. A time-scale guide showing the rate of visual loss is presented in [TABLE 66.6](#).
- Temporal arteritis is an important cause of retinal artery occlusion.
- Suspect field defect due to chiasmal compression if people are misjudging when driving.
- Pupillary reactions are normal in cortical blindness.
- Central retinal artery occlusion may be overcome by early rapid lowering of intraocular pressure.
- Retinal detachment and vitreous haemorrhage may require early surgical repair.
- Keep in mind antioxidant therapy (vitamins and minerals) for chronic macular degeneration.
- Consider multiple sclerosis foremost if there is a past history of transient visual

failure, especially with eye pain.

- If the person develops an eye symptom after using a hammer, always X-ray if no metal fragment can be seen on examination.

**Table 66.6** Time-scale guide for rate of visual loss<sup>3,7</sup>

**Sudden: less than 1 hour**

Amaurosis fugax  
Central retinal artery occlusion  
Hemianopias from ischaemia (emboli)  
Migraine  
Vitreous haemorrhage  
Acute angle glaucoma  
Papilloedema

**Within 24 hours**

Central retinal vein occlusion  
Functional (hysteria)

**Less than 7 days**

Retinal detachment  
Optic neuritis  
Acute macular problems

**Up to several weeks (variable)**

Choroiditis  
Malignant hypertension

**Gradual**

Compression of visual pathways  
Chronic glaucoma  
Cataracts  
Diabetic maculopathy  
Retinitis pigmentosa  
Macular degeneration  
Refractive errors

# Patient education resources

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Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Cataracts
- Colour blindness
- Floaters and flashes
- Glaucoma
- Macular degeneration

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# 67 Weight change

*Everything in excess is opposed to nature.*

HIPPOCRATES, 460–370 BC

In Australia, as in most other developed nations, the population health issue of having too much body weight has far outstripped the health issue of being underweight. The vexing problem of obesity is covered in the chronic disease section (see [CHAPTER 80](#) ). However, the relatively short-term gain or loss of weight presents a significant diagnostic challenge in general practice, particularly as so many underlying conditions can result in weight change.

## Weight gain

### Key facts and figures

- Two-thirds of the Australian population are overweight or obese and only 2–4% underweight.<sup>1</sup>
- Less than 1% of obese people have an identifiable secondary cause of obesity.<sup>2</sup>
- Two conditions causing unexplained weight gain that can be diagnosed by the physical examination are Cushing syndrome and hypothyroidism.
- After pregnancy, obesity may result from a failure to return to prepartum energy requirements.
- Even small weight losses are effective in preventing diabetes and improving the cardiovascular risk profile.<sup>3</sup>

## A diagnostic approach

A summary of the diagnostic strategy model is presented in [TABLE 67.1](#) .

**Table 67.1** Weight gain: diagnostic strategy model

**Probability diagnosis**

Exogenous obesity

Alcohol excess

Drugs

**Serious disorders not to be missed**

Cardiovascular:

- cardiac failure

Hypothalamic disorders (hyperphagia):

- craniopharyngiomas
- optic gliomas

Liver failure

Nephrotic syndrome

**Pitfalls (often missed)**

Pregnancy (early)

Endocrine disorders:

- hypothyroidism
- Cushing syndrome
- insulinoma
- acromegaly
- hypogonadism
- hyperprolactinaemia
- polycystic ovarian syndrome

Idiopathic oedema syndrome

Klinefelter syndrome

Congenital disorders:

- Prader–Willi syndrome
- Laurence–Moon–Bardet–Biedl syndrome

**Seven masquerades checklist**

Depression

Drugs

Thyroid disorder (hypothyroidism)

**Is the patient trying to tell me something?**

Yes: the reasons for obesity should be explored.

# Probability diagnosis

The outstanding cause of weight gain in exogenous obesity is excessive calorie intake coupled with lack of exercise, and influenced heavily by socio-environmental factors (see [CHAPTER 80](#) ).

## Serious causes not to be missed

It is important not to misdiagnose hypothalamic disorders, which may result in hyperphagia and obesity. Injury to the hypothalamus may occur following trauma and encephalitis and with a variety of tumours, including craniopharyngiomas, optic gliomas and pituitary neoplasms. Some of these tumours may cause headaches and visual disturbances.

It is also important not to overlook major organ failure and kidney disorders as a cause [Page 809](#) of increased body weight, especially cardiac failure, liver failure and the nephrotic syndrome. The associated increase in body water needs to be distinguished from increased body fat. Consider the obesity hypoventilation syndrome (Pickwickian syndrome) in those with BMI  $>40 \text{ kg/m}^2$ .

## Pitfalls

### Endocrine disorders

The endocrine disorders that cause obesity include Cushing syndrome, hypothyroidism, insulin-secreting tumours and hypogonadism. They should not represent difficult diagnostic problems.

An insulin-secreting tumour (insulinoma) is a very rare adenoma of the B cells of the islets of Langerhans. The main features are symptoms of hypoglycaemia and obesity.

### Congenital disorders

The rare congenital disorders that cause obesity, such as Prader–Willi and Laurence–Moon–Bardet–Biedl syndromes, should be easy to recognise in children (see [CHAPTER 23](#) and later in this chapter).

### Chromosomal abnormalities

An important abnormality to bear in mind is Klinefelter syndrome (XXY karyotype), which affects one out of every 400–500 males. The boys show excessive growth of long bones and are tall and slim. Without testosterone treatment they become obese as adults.

Some girls with Turner syndrome (XO karyotype) may be short and overweight.

### Some gender pointers

Consider polycystic ovarian syndrome in women and obstructive sleep apnoea in obese men.

## Seven masquerades checklist

The important masquerades include hypothyroidism and drug ingestion. Hypothyroidism is usually not associated with marked obesity. Drugs that can be an important contributing factor include tricyclic (and other) antidepressants, corticosteroids, pizotifen, the antipsychotics, depot progesterone and insulin. Obesity (overeating) may be a feature of depression, especially in the early stages.

## Psychogenic considerations

An underlying emotional crisis may be the reason for the overweight person to seek medical advice. It is important to explore diplomatically any hidden agenda and help them to resolve any conflict.

## The clinical approach

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A careful history is very valuable in ascertaining food and beverage intake and perhaps giving people insight into their calorie intake, since some deny overeating or will underestimate their food intake.<sup>3</sup> Enquire about gynaecological and family history, e.g. diabetes, cardiac disease.

### Relevant questions

- Do you feel that you have an excessive appetite?
- Tell me in detail what you ate yesterday.
- Give me an outline of a typical daily meal.
- Tell me about snacks, soft drink and alcohol that you have.
- What exercise do you get?
- Do you have any special problems, such as getting bored, tense and upset or depressed?
- What drugs are you taking?

### Examination

In the physical examination it is very important to measure body weight and height (and calculate BMI), waist circumference and assess the degree and distribution of body fat and the overall nutritional status. For a discussion of anthropometric measurements and their interpretation, see [CHAPTER 80](#). Record blood pressure and test the urine with dipsticks. Keep in mind that a standard blood pressure cuff on a large arm may give falsely elevated values.

Remember the rare possibilities of Cushing syndrome, acromegaly and hypothyroidism. Search for evidence of atherosclerosis and diabetes, and for signs of alcohol abuse.

An extensive working up of the CNS is not indicated in obesity without the presence of suspicious symptoms such as visual difficulties.

## Important investigations

- Lipid profile
- Glucose (fasting) and/or HbA1c if significant weight gain
- EUC, LFTs

## Investigations to consider

- Thyroid function tests
- Cortisol (if hypertensive)
- Testosterone (suspected sleep apnoea)
- ECG and chest X-ray (older than 40)

## Weight gain in children

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Various studies have found that approximately 10% of prepubertal and 15% of adolescent age groups are obese.<sup>4</sup>

Obesity in children is a BMI for age >95th percentile while overweight is >85th percentile. There is a risk of obesity-associated diseases and carrying the problem into adulthood, with a greater risk of obesity, premature death and disability.

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Raising the issue with parents and child requires sensitivity and discretion. Parents often blame obesity in children on their 'glands', but endocrine or metabolic causes are rare and can be readily differentiated from exogenous obesity by a simple physical examination and an assessment of linear growth. Children with exogenous obesity tend to have an accelerated linear growth whereas children with secondary causes are usually short.

## Congenital or inherited disorders

### Prader–Willi syndrome

The characteristic features are bizarre eating habits (e.g. binge eating), obesity, hypotonia, hypogonadism, intellectual disability, small hands and feet and a characteristic facial appearance (narrow bifrontal diameter, 'almond-shaped' eyes and a 'tented' upper lip). Progressive obesity

results from excessive intake in addition to decreased caloric requirements (see CHAPTER 23 ).

### Laurence–Moon–Bardet–Biedl syndrome

The characteristic features are obesity, intellectual disability, polydactyly and syndactyly, retinitis pigmentosa and hypogonadism.

### Beckwith–Wiedemann syndrome

Characteristics include excessive growth, macrosomia, macroglossia, umbilical hernia and neonatal hypoglycaemia. Children appear obese as they are above the 95th percentile by 18 months of age. Intelligence is usually in the normal range.

## Endocrine disorders

Endocrine disorders in children that can rarely cause obesity include hypothyroidism (often blamed as the cause but seldom is), Cushing syndrome, insulinomas, hypothalamic lesions, Fröhlich syndrome (adiposogenital dystrophy) and Stein–Leventhal syndrome (PCOS) in girls.

## Managing obesity in children

Childhood obesity usually reflects an underlying problem in the family system. It can be a very difficult emotional problem in adolescents, who develop a poor body image. An important strategy is to meet with family members, determine whether they perceive the child's obesity as a problem and whether they are prepared to solve the problem. The family dynamics will have to be assessed and strategies outlined. This may involve referral for expert counselling. It is worth pointing out that children eat between one-third and two-thirds of their meals at school, so schools should be approached to promote special programs for children who need weight reduction.

Conventional therapy by dietary modification, increasing energy expenditure by increasing activity, reducing sedentary behaviour, behaviour modification and family involvement is recommended (see CHAPTER 80 ). The best outcomes are achieved with a specialist team working with the whole family.<sup>5</sup> Some authorities emphasise that weight maintenance rather than weight loss is appropriate since many children will 'grow into their weight'.<sup>6</sup>

## Weight gain in adults

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### Cushing syndrome

Cushing syndrome is the term used to describe the chemical features of increased free circulating glucocorticoid. The most common cause is iatrogenic with the prescribing of synthetic corticosteroids. The spontaneous primary forms such as Cushing disease (pituitary-dependent hyperadrenalinism) are rare. As the disorder progresses the body contour tends to assume the often

quoted configuration of a lemon with matchsticks (see [CHAPTER 14](#) ).

## Clinical features

- Change in appearance
- Central weight gain (truncal obesity)
- Hair growth and acne in females
- Muscle weakness
- Amenorrhoea/oligomenorrhoea (females)
- Thin skin/spontaneous bruising
- Polymyalgia/polydipsia (diabetes mellitus)
- Insomnia
- Depression

## Signs

- Moon face
- ‘Buffalo hump’
- Purple striae
- Large trunk and thin limbs: the ‘lemon with matchsticks’ sign

Refer for diagnostic evaluation, including plasma cortisol and overnight dexamethasone suppression tests.

Untreated Cushing syndrome has a very poor prognosis, with premature death from myocardial infarction, cardiac failure and infection; hence, early diagnosis and referral is essential.

## Oedema

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Oedema (dropsy) is an excessive accumulation of fluid in tissue spaces. It may be generalised or localised—peri-orbital, peripheral or an arm (lymphoedema, refer to [CHAPTER 58](#) ).

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### Generalised oedema

The site of generalised oedema is largely determined by gravity. It is due to an abnormal excess

of sodium in the body, which leads to accumulation of water. The causes can be generally divided into two groups—oedema associated with a decreased plasma volume and oedema associated with an increased plasma volume (see TABLE 67.2 ).

**Table 67.2** Causes of generalised oedema

Decreased plasma volume
Hypoalbuminaemia (e.g. nephrotic syndrome, chronic liver disease, malnutrition)
Increased plasma volume
Congestive cardiac failure
Chronic kidney failure
Drugs (e.g. corticosteroids, NSAIDs, certain antihypertensives, oestrogens, lithium, others)
Idiopathic oedema

## Diagnosis

Clinical examination, including urinalysis, is usually sufficient to establish the cause of the oedema. In other cases, investigation of kidney or liver function may be required.

## Treatment

- Treat the cause where known
- Salt (sodium) restriction
- Diuretics:
  - a loop diuretic (e.g. frusemide)
  - a potassium-sparing diuretic (e.g. spironolactone)

## Idiopathic oedema

Idiopathic oedema, also known as cyclical or periodic oedema, is a common problem and the diagnosis is made on a characteristic history:

- exclusive to women
- may be cyclical or persistent
- usually unrelated to menstrual cycle

- excessive diurnal weight gain (worse on prolonged standing)
- abdominal bloating
- may affect hands and face as well as feet
- often made worse by diuretics
- may be associated with headache, depression, tension

Treatment of this condition is difficult. Most diuretics can aggravate the problem. Supportive stockings and a nutritious diet (with restricted sodium intake) are recommended as first-line treatment. A trial of spironolactone is often recommended.

## Swelling (puffiness) of the face and eyelids

The causes are similar to those for generalised oedema. Important specific causes to consider are:

- kidney disease (e.g. nephrotic syndrome, acute nephritis)
- hypothyroidism
- Cushing syndrome and corticosteroid treatment
- mediastinal obstruction/superior vena cava syndrome
- angio-oedema
- skin sensitivity (e.g. drugs, cosmetics, hair dryers)
- carotico-cavernous fistula

## Swelling of the legs

Refer to [CHAPTER 58](#) .

### ‘Cellulite’

‘Cellulite’ refers to a characteristic form of dimpling seen in the subcutaneous tissues of hips, buttocks and thighs of females. The dimpling pattern is related to the manner of attachment of fibrous septae that contain the fat. Many patients seek advice about ‘cellulite’ in the buttocks and thighs in particular. Explain that the best way to overcome it is to maintain an ideal weight. If overweight, lose it slowly and exercise to improve the muscle tone in the buttocks and thighs.

## Weight loss

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In family practice complaints of loss of weight are more frequent than complaints about being too thin. Of great significance is the problem of recent loss of weight. A very analytical history is required to determine the patient's perception of weight loss. The equivalent problem in children is failure to gain weight or thrive.

Weight loss is an important symptom because it usually implies a serious underlying disorder, either organic or functional. It may or may not be associated with anorexia and thus diminished food intake.

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## Key facts and checkpoints

- Any loss of more than 5% of normal body weight is significant.
- The most common cause in adults of recent weight loss is stress and anxiety.<sup>7</sup>
- Serious organic diseases to consider are:
  - malignant disease
  - diabetes
  - chronic infections (e.g. tuberculosis)
  - thyrotoxicosis
- The most important variable to consider in evaluating weight loss is appetite. Eating and weight go hand in glove.
- Two conditions commonly associated with weight loss are anaemia and fever; they must be excluded.
- Early detection of eating disorders improves outcome.

## A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 67.3 .

**Table 67.3** Weight loss: diagnostic strategy model (other than deliberate dieting or malnutrition)

### Probability diagnosis

Stress and anxiety (e.g. redundancy, relationship breakdown)

Depressive illness

Non-coping elderly/dementia

Eating disorders: anorexia nervosa/bulimia

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Chronic congestive heart failure

Malignant disease, including:

- stomach
- pancreas
- lung
- myeloma
- caecum
- lymphoma

Chronic infection:

- HIV infections (AIDS, AIRC)
- tuberculosis
- hidden abscess
- infective endocarditis
- brucellosis
- others, e.g. overseas acquired infection

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### Pitfalls (often missed)

Drug dependence, esp. alcohol

Malabsorption states:

- intestinal parasites/infestations
- coeliac disease

Other GIT problems

Chronic kidney failure

Connective tissue disorders (e.g. SLE, RA)

Dementia

*Rarities:*

- malnutrition
- Addison disease
- hypopituitarism

---

### Seven masquerades checklist

Depression

Diabetes

Drugs

Anaemia

Thyroid disorder (hyperthyroidism)

UTI

### Is the patient trying to tell me something?

A possibility. Consider stress, anxiety and depression.

Anorexia nervosa and bulimia are special considerations.

## Probability diagnosis

Excluding planned dietary restriction, psychological factors are the most common cause, particularly recent stress and anxiety.<sup>7</sup> Elderly people with adverse psychological factors, neglect and possibly drug effects can present with wasting.

## Serious disorders not to be missed

Many of the problems causing weight loss are very serious, especially malignant disease.

### Malignant disease

Weight loss may be a manifestation of any malignancy. With cancer of the stomach, pancreas and caecum, malignant lymphomas and myeloma, weight loss may be the only symptom. Occult malignancy must be regarded as the most common cause of unexplained weight loss in the absence of major symptoms and signs. The mechanisms may be multiple, with anorexia and increased metabolism being important factors.

### Chronic infections

These are now less common but tuberculosis must be considered, especially in people from less developed countries. Some cases of infective endocarditis may progress only very slowly with general debility, weight loss and fever as major features.<sup>8</sup>

Other infections to consider are brucellosis, and protozoal and systemic fungal infection. Infection with HIV virus must be considered, especially in high-risk groups.

## Pitfalls

Drug dependency, including alcohol and narcotic drugs, must be considered, especially Page 813 when the problem may result in inappropriate nutrition. Apart from malignant disease there is a whole variety of gastrointestinal disorders that require consideration—these include malabsorption states, gastric ulceration, and intestinal infestations, especially in people returning from a significant stay in tropical and under-developed countries.

Addison disease (see [CHAPTER 14](#)) can be very difficult to diagnose. Symptoms include

excessive fatigue, anorexia, nausea and postural dizziness. Hyperpigmentation is a late sign.

## Seven masquerades checklist

Depression and the endocrine disorders---diabetes and hyperthyroidism---are important causes.

### Diabetes

Unintended weight loss is a particular issue with type 1 diabetes; always be aware of the dangerous ketoacidosis as a first presentation. The triad is thirst + polyuria + weight loss.

### Hyperthyroidism

This is usually associated with weight loss although in some, such as an elderly male, it may not be obvious. An important clue will be weight loss in the presence of an excellent appetite, which helps distinguish it from a psychoneurotic disturbance.

### Depression

Weight loss is a common feature of depression and is usually proportional to the severity of the disease. In the early stages of depression, weight gain may be present but when the classic loss of the four basic drives (appetite, energy, sleep and sex) becomes manifest, weight loss is a feature.

### Drugs

Any prescribed drugs causing anorexia can cause weight loss. Important drugs include digoxin, narcotics, cytotoxics, NSAIDs, some antihypertensives and theophylline. Be mindful of inappropriate use of thyroxine and laxatives.

### Red flag pointers for weight loss

Weight loss per se is a big red flag.

- Rapid weight loss with malaise
- Acid dental erosion on surfaces of upper teeth: think bulimia
- Weakness and malaise in young females: consider eating disorder and hypokalaemia
- Evidence of abuse in a child

## Psychogenic considerations

Weight loss is a feature of anxiety as well as depression. Some patients with psychotic disturbances, including schizophrenia and mania, may present with weight loss.

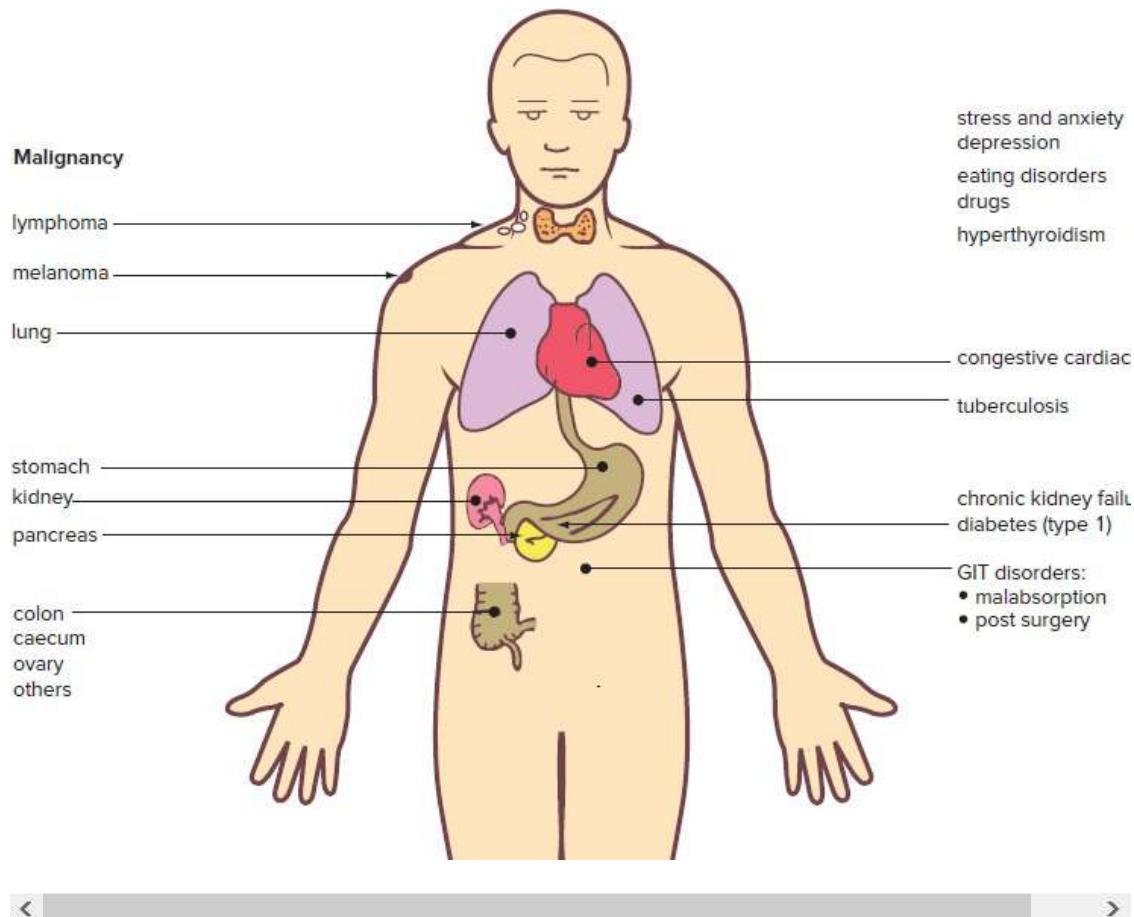
Anorexia nervosa is quite common, particularly in females between the ages of 12 and 20 years. The main differential diagnosis is hypopituitarism, although anorexia nervosa can cause endocrine disturbances through the hypothalamic pituitary axis.

## The clinical approach

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### History

It is important to document the weight loss carefully and evaluate the patient's recordings. The same set of scales should be used. It is also important to determine the food intake. However, in the absence of an independent witness such as a spouse or parent, this can be difficult. Food intake may be diminished with psychogenic disorders and cancer but increased or steady with endocrine disorders, such as diabetes and hyperthyroidism, and with steatorrhoea. FIGURE 67.1 shows the possible causes of weight loss.



**FIGURE 67.1** Weight loss: causes to consider

## General questions

- Exactly how much weight have you lost and over how long?
- Have you changed your diet in any way?
- Has your appetite changed? Do you feel like eating?
- Have your clothes become looser?
- What is your general health like?
- Do you feel uptight (tense), worried or anxious?
- Do you get very irritable or tremulous?
- Do you feel depressed?
- Do you ever force yourself to vomit?
- Are you thirsty?
- Do you pass a lot of urine?
- Do you have excessive sweating?
- Do you experience a lot of night sweats?
- What are your motions like?
- Are they difficult to flush down the toilet?
- Do you have a cough or bring up sputum?
- Do you get short of breath?
- Do you have any abdominal pain?
- Are your periods normal (for females)?
- What drugs are you taking?
- How many cigarettes do you smoke?

## Examination

A careful general examination is essential with special attention to:

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- vital parameters, e.g. BMI, temperature, BP

- the thyroid and signs of hyperthyroidism
- the abdomen (check liver, any masses and tenderness)
- rectal examination (test stool for occult blood)
- reflexes
- look for acid dental erosion on surface of upper teeth (bulimia)

## Investigations

Basic investigations include:

- haemoglobin, red cell indices and film
- white cell count
- ESR/CRP
- thyroid function test
- random blood sugar
- EUC, LFTs
- chest X-ray
- urine analysis
- faecal occult blood

Others to consider:

- upper GIT (endoscopy or barium meal)
- HIV serology
- ultrasound of abdomen (or CT if suspected abnormality not found)
- colonoscopy
- tumour markers, e.g. CA-125, CEA

## Weight loss in children

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Weight loss in children can be considered as:

- 1. failure to thrive (FTT): the child up to 2 years below 3rd percentile (refer to CHAPTER 84 )
- 2. weight loss in a child after normal development

## Loss of weight in the older child

Acute or chronic infections are the most common causes of weight loss in children beyond infancy.<sup>9</sup> In acute infections the weight loss is transient, and once the infection clears the child generally regains the lost weight. In chronic infections signs may be more difficult to detect (e.g. urinary tract infection, pulmonary infection, osteomyelitis, chronic hepatitis). In common with the younger child who fails to thrive, the older child may be suffering from malabsorption syndrome, chronic infection of the urinary tract or a rare chromosomal or metabolic disorder.<sup>10</sup> Tuberculosis, diabetes and malignant disease may present as weight loss and it is necessary to exclude organic disease before considering the more common emotional disorders.

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## Eating disorders in the adolescent

Concerns about body image and dieting are very common among young women in modern society, and to an increasing extent in young men. Among these dieters 5–10% become abnormally preoccupied with dieting and slimness and progress to the eating disorders of anorexia nervosa and bulimia. They often have extremely low self-esteem and feel ineffective. They tend to be perfectionists with obsessive-compulsive traits. A history of childhood sexual abuse may be relevant. Media images alone do not cause eating disorders but have a role—genetic vulnerability, temperament, psychological and environmental factors also mediate these illnesses: ‘Genes load the gun—the environment pulls the trigger’.

The DSM-5 criteria for diagnosing these disorders, which have serious physical and psychological consequences, are presented in TABLE 67.4 . The differential diagnosis of anorexia nervosa includes most of the problems listed in TABLE 67.4.

**Table 67.4** DSM-5 criteria for diagnosing anorexia nervosa and bulimia

### Anorexia nervosa

- A Restriction of energy intake relative to requirements leading to significantly low body weight in context of age, sex and physical health. That weight is less than minimally normal or expected
  - B Intense fear of gaining weight or becoming fat, despite current underweight status
  - C Disturbance of body image (body size or shape) or persistent lack of recognition of seriousness of low body weight
- |       |   |
|-------|---|
| Types | Restricting type—no binge eating or purging binge eating/purging type |
|-------|---|

### Bulimia nervosa

- A      Recurrent episodes of binge eating, that is:
1. eating in a discrete period of time an abnormal quantity of food compared with the average person
  2. a sense of lack of control during the binge
- B      Recurrent inappropriate compensatory behaviour to prevent weight gain (e.g. self-induced vomiting; misuse of laxatives, diuretics, enemas etc.); fasting or excessive exercise
- C      A and B both occur, on average, at least twice a week for 3 months
- D      Self-evaluation is unduly influenced by body shape and weight
- E      Does not occur exclusively during periods of anorexia nervosa
- Types    Purging, non-purging (e.g. fasting, excessive exercise)
- 

### The history<sup>11</sup>

These patients are often secretive, tend to minimise their symptoms and may be in denial of their problem. GPs are encouraged to engage these patients and keep in close contact with them, referring them as necessary. The validated screening tool SCOFF is recommended.

### The SCOFF screening tool

- S** Do you make yourself **Sick** because you feel uncomfortably full?
- C** Do you worry you have lost **Control** over how much you eat?
- O** Have you recently lost more than 6 kg (**One stone**) in a 3-month period?
- F** Do you believe yourself to be **Fat** when others say you are too thin?
- F** Would you say **Food** dominates your life?

If the patient answers Yes to two or more questions, there is a high index of suspicion for an eating disorder, warranting a more detailed assessment.

## Anorexia nervosa

See FIGURE 67.2.



**FIGURE 67.2** 18-year-old adolescent with severe anorexia nervosa (BMI 7.7). This patient survived after care by her GP.

*Photo and history courtesy Dr MM O'Brien*

Anorexia nervosa is a syndrome characterised by the obsessive pursuit of thinness through dieting with extreme weight loss and disturbance of body image.<sup>12</sup> The main symptoms are anorexia and weight loss. The mortality rate may be as high as 18%. It has the highest mortality and suicide rate of any psychiatric disorder.<sup>11</sup>

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## Typical features

- Up to 1% incidence among schoolgirls aged 16<sup>13</sup>
- Bimodal age of onset: 13–14 and 17–18 years<sup>11</sup>
- Unknown cause
- Poor insight
- Severe emaciation

- Amenorrhoea
- Loss of body fat
- Sallow, dry and scaly skin, hair loss
- Increased lanugo body hair
- BMI <17.5
- Continuing behaviour directed at weight loss

## **Bulimia nervosa**

Bulimia is episodic secretive binge eating followed by self-induced vomiting, fasting or the use of laxatives or diuretics. This binge–purge syndrome is also referred to as bulimarexia. It is more difficult to detect than anorexia nervosa but has a higher incidence. There are two types—the purging type and the non-purging type where fasting or excessive exercise are the compensatory behaviours. The purging type is the most life-threatening behaviour because of the danger of hypokalaemia.

### **Typical features<sup>11</sup>**

- Young females (F:M ratio 10:1)
- Begins at later age, usually 17–25 years
- Associated psychoneurotic disorders
- Family history
- Fluctuations in body weight without extreme loss or gain
- Menstrual history usually normal but periods may be irregular—amenorrhoea rare
- Physical complications of frequent vomiting (e.g. dental decay, effects of hypokalaemia)
- Recurrent laxative, stimulant or enema abuse
- Preoccupation with food
- Exaggerated weight/body shape concern
- Impulse control disorders (e.g. gambling, substance abuse)
- Depressed mood with guilt after a binge

Laboratory evaluation should include electrolytes, FBE, iron studies, TFTs, kidney function

tests, coeliac disease screening and LFTs, ESR.

## Avoidant/restrictive food intake disorder

Previously known as ‘selective eating disorder’, this is an eating disorder whereby people eat only within a very narrow repertoire of foods. These people may appear healthy but can also present with weight loss, growth failure or nutritional deficiency.<sup>14</sup>

*Note:* The other main eating disorder is ‘binge eating disorder’, which is defined as recurrent episodes of binge eating in the absence of regular use of inappropriate compensatory behaviours characteristic of bulimia nervosa. Most people who binge eat are obese.

## Management of eating disorders

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Early detection and intervention are essential to reduce the risk of chronicity. Treatment [Page 817](#) can be conducted on an outpatient basis but if there are marked trends, such as severe weight loss, a family crisis, severe depression and a suicide risk, the person requires hospital admission. The burden on caregivers of people with eating disorders is high. There are often problematic family interrelationships that require exploration.

Important goals are:

- establish a good and caring relationship with the patient
- resolve underlying psychological difficulties
- restore weight to a level between ideal and the patient’s concept of optimal weight
- provide a balanced diet of at least 3000 calories per day (anorexia nervosa)

Structured behavioural therapy, intensive psychotherapy and family therapy may be tried but supportive care by physicians and allied health staff appears to be the most important feature of therapy.<sup>15</sup> Psychotherapy may be arranged by referral to a psychologist or psychiatrist. The patient may need admission to hospital, especially if dehydration and hypokalaemia (from purging) and also suicide are concerns. Rapid weight loss, vomiting, especially with poor insight, are also ‘red flags’ for hospital admission. Antidepressants, especially of the SSRI group, may be helpful for selected patients with a comorbid depressive illness. Fluoxetine is the preferred agent for bulimia. It is important to provide ongoing support for both patient and family.

## Weight loss in the elderly

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General weight loss is a relatively common physiological feature of many elderly people. However, abnormal weight loss is commonly encountered in the socially disadvantaged elderly, especially those who live alone and lack drive and interest in adequate food preparation. Other factors include relative poverty and poor dentition, including ill-fitting and painful false teeth. An

important cause that should always be considered is malignant disease. Consider depression, dementia and drug interactions as potential causes of weight loss. Depression is the most common reversible cause of weight loss in elderly people, occurring in up to 30% of all medical outpatients presenting with undernutrition.<sup>15</sup> Weight loss of more than 5% body weight in 6 months is significant and suggests undernutrition.<sup>15</sup>

Congestive cardiac failure, especially secondary to ischaemic heart disease, is a common cause of weight loss. This is due to visceral congestion.

## Gastrointestinal causes of weight loss

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The following conditions may lead to weight loss:

- coeliac disease
- poor oral hygiene
- chronic vomiting or diarrhoea (e.g. pyloric stenosis)
- gastric ulcer
- cancer of the stomach, oesophagus, large bowel
- problem alcohol drinking
- partial or total gastrectomy
- other GIT surgery
- inflammatory bowel disease (e.g. Crohn disease, ulcerative colitis)
- steatorrhoea
- lymphoma of the gut
- parasitic infestation
- cirrhosis of the liver

The mechanisms of weight loss include anorexia, malabsorption, obstruction with vomiting and inflammation.

## When to refer

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- Those with BMI >35 who are resistant to simple weight control measures.<sup>5</sup>
- Those with obesity and associated medical problems such as angina or severe osteoarthritis

who require relatively rapid weight reduction.

- Possibility of endocrine or congenital cause of weight gain or loss.
- Any unexplained weight loss, especially if an endocrine cause or malignancy is suspected and can't be identified.
- Weight loss related to a serious psychological illness or eating disorder.

### Practice tips

- Ask patients what they really believe is the cause of their weight gain or loss.
- An anxiety state and hyperthyroidism can be difficult to differentiate as causes of weight loss. Perform thyroid function tests.
- Laboratory tests are rarely needed to establish the diagnosis of an eating disorder. Hormonal levels return to normal following weight gain.
- A high index of suspicion by the family doctor is required to diagnose eating disorders. Think of it in a mid-teen female; weight loss through dieting; wide fluctuation in weight; amenorrhoea and hyperactivity.

## Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

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- Eating disorders
- Obesity: how to lose weight wisely
- BMI calculator

## Resources

NHMRC Obesity guidelines. Available from:

[www.health.gov.au/internet/main/publishing.nsf/Content/obesityguidelines-index.htm](http://www.health.gov.au/internet/main/publishing.nsf/Content/obesityguidelines-index.htm).

RACGP Red book: the 5 As approach. Available from: [www.racgp.org.au/your-practice/guidelines/snap/2-an-approach-to-preventive-care-in-general-practice/21-the-5as/](http://www.racgp.org.au/your-practice/guidelines/snap/2-an-approach-to-preventive-care-in-general-practice/21-the-5as/).

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# 68 Depression and other mood disorders

*Though I am often in the depths of misery, there is still calmness, pure harmony and music inside me. I see paintings or drawings in the poorest cottages and the dirtiest corners. And my mind is driven towards these things with an irresistible momentum.*

VINCENT VAN GOGH, BIPOLAR SUFFERER (1853–1890)

A mood disorder, also referred to as an affective disorder, is an emotional condition that significantly impacts on a person's mood and related functions. The combination of involved symptoms leads to a predominant mood state that is abnormal in quality, duration or both.

### Classification:

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A classification of the types of mood disorders is presented in TABLE 68.1 . There are two basic groups ranging from the very low mood of depression to the elevated mood of mania.

**Table 68.1** Classification of mood disorders DSM-5

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#### Mood disorders:

- major depressive illness
- hypomanic episode
- manic episode

#### Depressive disorders:

- major depressive disorder

- persistent depressive episode
- disruptive mood regulation, e.g. temper tantrums
- premenstrual dysphoric disorder
- depressive disorder due to another medical condition
- substance-induced mood disorder
- other specified or non-specified disorder

**Bipolar and related disorders:**

- bipolar 1 disorder
- bipolar 2 disorder
- cyclothymic disorder

- The DSM-5 classification<sup>1</sup> divides depressive disorders into major depressive disorder (MDD), disruptive mood dysregulation disorder, persistent depressive disorder (PDD) and premenstrual dysphoric disorder. Other 'specified' and 'unspecified' disorder categories allow for diagnosis of those patients who fall short of the various diagnostic criteria.<sup>2</sup> Refer to [CHAPTER 10](#) on depression.<sup>3</sup>
- MDD is subclassified with coded course or severity specifiers. These include mild, moderate or severe (see [TABLE 68.2](#))—with psychotic features, in partial remission and in full remission.
- Non-coded specifiers can also be used, including 'with anxious distress', 'with mixed features', 'with melancholic features', 'with atypical features', 'with mood-congruent psychotic features', 'with catatonia', 'with peripartum onset' and 'with seasonal pattern'.

**Table 68.2** Classification of severity of depressive illness, based on clinical features

Symptom cluster	Mild	Moderate	Severe
Mood	<ul style="list-style-type: none"> <li>• Lowered mood</li> <li>• Reduced joy</li> <li>• Crying</li> <li>• Anxiety</li> <li>• Irritability</li> </ul>	<ul style="list-style-type: none"> <li>• Reduced interest in things</li> <li>• Reduced pleasure in things</li> <li>• Reduced reactivity</li> </ul>	<ul style="list-style-type: none"> <li>• No interest in things</li> <li>• No pleasure in things</li> <li>• No reactivity</li> </ul>

Depressive thought	<ul style="list-style-type: none"> <li>• Loss of confidence</li> </ul>	<ul style="list-style-type: none"> <li>• Pessimistic about the future</li> <li>• Feeling worthless or a failure</li> <li>• Paranoid ideas</li> </ul>	<ul style="list-style-type: none"> <li>• Hopeless, see no future, self-reproach, guilt, shame</li> <li>• Consider illness a punishment</li> <li>• Paranoid or nihilistic delusions</li> </ul>
Cognition	<ul style="list-style-type: none"> <li>• Minor forgetfulness or lack of concentration</li> </ul>	<ul style="list-style-type: none"> <li>• Indecisiveness</li> <li>• Forgetfulness</li> </ul>	<ul style="list-style-type: none"> <li>• Unable to make decisions</li> <li>• Slowed mentation, seems cognitively impaired (pseudodementia)</li> </ul>
Somatic	<ul style="list-style-type: none"> <li>• Low drive</li> <li>• Loss of interest in food</li> <li>• Lowered libido</li> <li>• Mild initial insomnia; wake 1–2 times a night</li> </ul>	<ul style="list-style-type: none"> <li>• Low energy, drive</li> <li>• Eat with encouragement; mild weight loss</li> <li>• Loss of libido</li> <li>• Initial insomnia; wake several times a night</li> </ul>	<ul style="list-style-type: none"> <li>• No energy, drive</li> <li>• Unable to eat; severe weight loss</li> <li>• No libido</li> <li>• Psychomotor retardation or agitation</li> <li>• Sleep only a few hours</li> </ul>
Social	<ul style="list-style-type: none"> <li>• Mild social withdrawal</li> </ul>	<ul style="list-style-type: none"> <li>• Apathy and social withdrawal</li> <li>• Work impairment</li> </ul>	<ul style="list-style-type: none"> <li>• Apathy and social withdrawal</li> <li>• Marked work impairment</li> <li>• Poor self-care</li> </ul>
Suicidality	<ul style="list-style-type: none"> <li>• Life not enjoyable, not worth living</li> </ul>	<ul style="list-style-type: none"> <li>• Thoughts of death or suicide</li> </ul>	<ul style="list-style-type: none"> <li>• Evidence of intent to suicide (plans, attempts, etc.)</li> </ul>

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In a general practice setting, having a checklist of symptoms to work through with a patient can be a useful part of the assessment of the depressed patient. However, the DSM criteria can be too rigid for general practice, with many patients whom GPs recognise as having psychological issues not meeting DSM-5 criteria.<sup>4</sup> As Ian Hickie explains, ‘Primary care psychiatry is not specialist psychiatry in general practice’.<sup>5</sup>

## Types of mood disorders

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# Depressive disorders

## Major depressive episode

Major depressive episode (MDE) or unipolar depression which manifests with at least five of the features described in the DSM-5 diagnostic criteria (see [CHAPTER 10](#) where the clinical features and management of depression is covered in more detail).

## Major depressive disorder (MDD)

Major depressive disorder (MDD) or unipolar depression is also referred to as major depression or clinical depression. It manifests as the presence of a single major depressive episode without a manic or psychotic presentation at any one point in time. The two key criteria for MDD in the DSM-5 are a pervasive depressed mood and marked loss of interest or pleasure (otherwise referred to as anhedonia) persisting for at least two weeks along with other criteria (see [CHAPTER 10](#) ).

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## Major depression with psychotic features

Major depression with psychotic features, where there is both psychotic and depressive features at one point in time.

## Adjustment disorder with depressed mood

Adjustment disorder with depressed mood is a period of distress and emotional disturbance following a significant stressful life event (reactive depression, e.g. loss of employment). It is a less severe form of depression without sufficient criteria for major depression, and is very common. The symptoms may resolve spontaneously or benefit from short-term counselling. Its duration is usually no longer than 6 months. There is no evidence that antidepressants are helpful.

## Melancholic depression (melancholia)

This is basically general MDD but is a more severe form where symptoms such as anhedonia and psychomotor retardation are prominent. The term is not currently used often by psychiatrists and other therapists.

## Recurrent brief depression

There is a high prevalence in general practice of patients with recurrent episodes of short duration, about 3 to 7 days, as often as monthly. Premenstrual dysphoria may be a factor. As a rule antidepressants are ineffective. Lithium is an alternative medication for long-term use. Management is based on psychotherapy, especially CBT.

## Bereavement

This is a reactive depression to the death of a loved one. The expression and duration vary considerably among cultural groups. The diagnosis of MDD is usually not given until symptoms are present for 2 months after the loss. See [CHAPTER 4](#) .

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## Perinatal depression

This term recognises depressive symptoms throughout pregnancy and in the postnatal phase, where the disorder ranges from normal ‘baby blues’ to postnatal adjustment disorder, and finally to the severe postnatal (or postpartum) depression. It affects about 10–20% of mothers.

### Postnatal blues

The ‘blues’ are a very common problem occurring in up to 80% of women that arises in the first 2 weeks (usually 3–10 days after childbirth) but lasts only about 4–14 days. Clinical features are feeling flat or depressed, mood swings, irritability, feeling emotional (e.g. crying easily) and inadequate, and lacking confidence.

### Postnatal adjustment disorder

Occurs in the first 6 months; similar symptoms to the ‘blues’; anxiety with handling baby; psychosomatic complaints; fearful of criticism.

### Postnatal depression

Some women develop a very severe depression after childbirth. Always consider it in the frequent attender. Symptoms are present for at least 2 consecutive weeks, with onset in the first few days postpartum.

This occurs in 10–30% of women in the first 6–12 months (usually first 6 months, peaks about 12th week); anxiety and agitation common; marked mood swings; poor memory and concentration; typical features of depression.

*Note:* Beware of puerperal psychosis with onset usually within the first 2 weeks.

### Postpartum psychosis

The most common postpartum psychosis is an affective disorder: mania or agitation depression. It demands urgent attention. Symptoms that appear within the first month include unusual behaviour, agitation, delirium, hallucinations, mania and suicidal ideation. It is rare, occurring in about 1:500 births.

### Dysthymic disorder or persistent depressive disorder

## (PDD)

Refers to longstanding chronic depressed mood of relatively mild severity for at least 2 years. The depressed mood is accompanied by two or more of the symptoms outlined in the DSM-5 criteria. Antidepressants are less predictably effective than in major depression. *Note:* Included in DSM-5.

## Double depression

This is defined as a moderate depressed mood (dysthymia) that lasts for at least 2 years.<sup>6</sup>

## Disruptive mood dysregulation disorder

This is a depressive disorder of children up to 18 years of age who exhibit persistent irritability and frequent episodes of extreme behavioural control and uncontrollable social behaviour without any significant provocation. *Note:* Included in DSM-5.

## Premenstrual dysphoric disorder

This diagnosis is based on the presence of a cluster of affective behavioural and emotional symptoms in the week preceding the onset of menstruation, followed by the resolution of these symptoms after onset. The symptoms must include at least five depressive symptoms (see [CHAPTER 10](#)). *Note:* Included in DSM-5.

## Dysphoria

This is a profound state of unease or generalised dissatisfaction with life (as opposed to euphoria). It may accompany depression, anxiety, physical discomfort, menstruation or unhappiness with one's biological sex or usual gender role (gender dysphoria).

## Atypical depression

This is different to the persistent sadness of typical depression, where the individual's mood improves with a pleasurable event.

## Catatonic depression

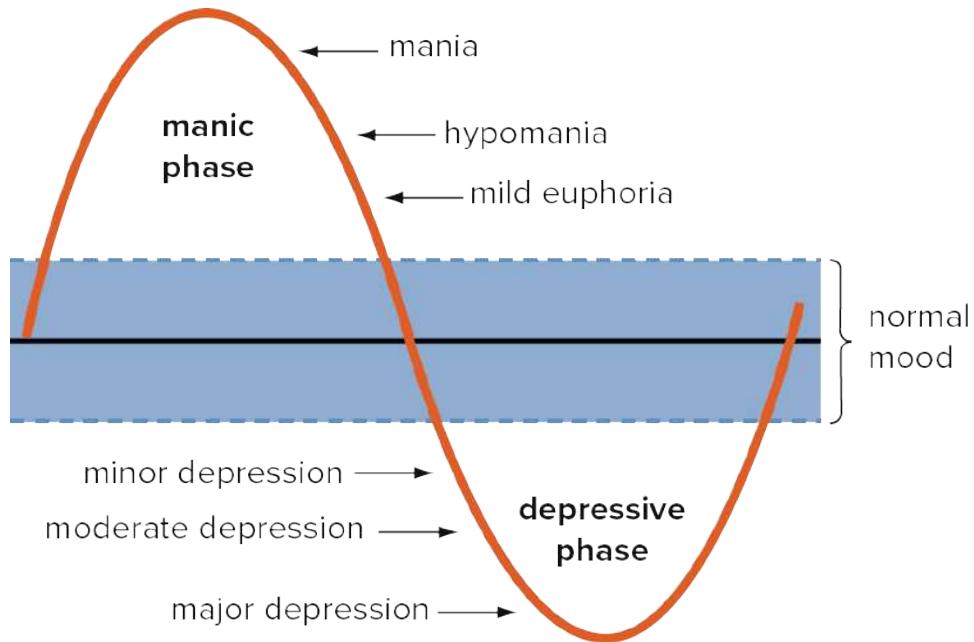
An uncommon and severe form of major depressive episode involving disturbance of motor behaviour where the person is mute and has severe psychomotor retardation with purposeless or bizarre movements. Grimacing, echolalia and echopraxia may feature. Catatonic symptoms can also appear in schizophrenia or a manic episode.

## Seasonal affective disorder

Seasonal affective disorder (SAD), or ‘winter blues’ or winter depression, is a recurrent Page 823 depressive disorder seen in people living in cold climates where winters are bleak and dark. It appears in the autumn or winter and usually resolves in spring. The diagnosis is based on at least two episodes occurring in colder months and none at other times over a 2-year period or longer. Features of depression include sleeping difficulty, sadness, lethargy, irritability and anxiety, while atypical symptoms include somnolence and increased appetite (carbohydrate craving). Treatment is based on psychotherapy, phototherapy and medication such as the SSRIs. Refer to: [www.sada.org.uk](http://www.sada.org.uk).

## Bipolar disorders

Bipolar is a broad term to describe a recurrent illness with episodes of either abnormal high mood (mania) or low mood (depression), with return to normal function in between. The swing in moods in bipolar disorders (formerly manic depression disorders) is illustrated in **FIGURE 68.1**. It affects 1–2% of the population, while BP-NOS may affect between 2–5% (refer to **CHAPTER 69** for features and management).



**FIGURE 68.1** Bipolar disorder (manic depression): possible mood swings

### **Bipolar I disorder**

Bipolar I disorder has one fully fledged manic or mixed episode and usually depressive episodes.

### **Bipolar II disorder**

Bipolar II disorder is defined as a major depressive episode with at least one hypomanic episode

lasting a minimum of 4 days but no classic manic episodes.

## Hypomania

Hypomania is the term used to describe the symptoms of mania that are similar to but less severe (without criterion C) and of shortened duration. The subsequent major depressive (cyclothymic) phase is associated with a high risk of suicide.

## Cyclothymic disorder

Cyclothymic disorder is a form of bipolar disorder consisting of recurrent hypomanic and dysthymic episodes without full manic or major depressive episodes.

## Dysphoric mania

Dysphoric mania or mixed episode during manic episodes patients may also experience depressive symptoms.<sup>3</sup>

## Rapid cyclic bipolar disorder

Where 4 or more episodes of depression, mania or mixed episodes occur in a 12-month period.<sup>3</sup>

## Bipolar disorder not otherwise specified (BP-NOS)

Also known as ‘subthreshold’ bipolar, this is where the patient has some symptoms of the bipolar spectrum but does not conform to the DSM-5 diagnosis (see [CHAPTER 69](#) ).

## Community mood disorders prevalence study

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An interesting study conducted over 9 years commencing in 1985 among young American adults involved a selection of demographic and health characteristics.<sup>7</sup> Lifetime prevalence based on six mood measures were estimated and showed the following distribution.

- 1. Major depressive episode (MDE): 8.6%
  - 2. Major depressive disorder with severity (MDS-s): 7.7%
  - 3. Dysthymia: 6.2%
  - 4. MDE with dysthymia: 3.4%
  - 5. Any bipolar disorder: 1.6%
  - 6. Any mood disorder: 11.5%
-

## Key points

- Mood disorders are marked emotional disturbances consisting of prolonged periods of profound sadness, excessive joyousness or both, or variations of both, especially with depression.
- Diagnosis is based on analysis of a requisite number of mood disorder symptoms as presented by the DSM-5 classification.
- These disorders tend to have a hereditary basis which should be addressed in the patient's history.
- With all overt disorders, depression or mania, we must be very mindful of the importance of being alert for the risk of suicide.

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## 69 The disturbed patient

*There is not a sight in nature so mortifying as that of a Distracted Person, when his imagination is troubled, and his whole soul is disordered and confused.*

JOSEPH ADDISON (1672–1719)

The disturbed and confused patient is a complex management problem in general practice. The cause may be a single one or a combination of several abnormal mental states (see

TABLE 69.1 ).<sup>1</sup> The cause may be an organic mental disorder, which may be a long-term insidious problem such as dementia, or an acute disorder (delirium), often dramatic in onset. On the other hand, the cause of the disturbance may be a psychiatric disorder such as panic disorder, mania, major depression or schizophrenia.

**Table 69.1** A general classification of psychiatric disorders<sup>1</sup>

Organic mental disorders:

- acute organic brain syndrome (delirium)
- chronic organic brain syndrome (dementia)

Psychoactive and substance use disorders:

- toxic states
- drug dependency
- withdrawal states

Schizophrenic disorders

Mood disorders:

- major depression
- bipolar (manic depressive) disorder
- adjustment disorders with depressed mood
- dysthymia

Anxiety disorders:

- generalised anxiety disorder
- panic disorder
- obsessive-compulsive disorder
- phobic disorders
- post-traumatic stress disorder

Disorders specific to children

Other disorders:

- postpartum psychiatric illness
  - eating disorders
  - personality disorders
  - body dysmorphic disorder
- 

## Key facts and checkpoints

- Depression affects 15% of people over 65 and can mimic or complicate any other illness, including delirium and dementia.<sup>1</sup>
- Elderly patients with depression are at a high risk of suicide.
- Always search vigorously for the cause or causes of delirium.
- Seeing patients in their home is the best way to evaluate their problem and support systems. It allows opportunities for a history from close contacts and for checking medication, alcohol intake and other factors.
- The diagnosis of dementia can be overlooked: a Scottish study showed that 80% of demented patients were not diagnosed by their GP.<sup>2</sup>
- Patients with a chronic brain syndrome (dementia) are at special risk of an acute brain syndrome (delirium) in the presence of infections and many prescribed drugs.<sup>1</sup>
- Consider prescribed and illicit substances, including the severe anticholinergic delirium syndrome.
- The key feature of dementia is impaired memory.
- The two key features of delirium are disorganised thought and attention.

- Hallucination guidelines:
  - auditory: psychoses, e.g. schizophrenia
  - visual: almost always organic disorder
  - olfactory: temporal lobe epilepsy
  - tactile: cocaine abuse, alcohol withdrawal

The manifestations of the disturbance are many and include perceptual changes and hallucinations, disorientation, changes in consciousness, changes in mood from abnormally elevated to gross depression, agitation and disturbed thinking, including delusions.

## A diagnostic approach

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A summary of the diagnostic strategy model for the disturbed or confused patient is presented in [TABLE 69.2](#).

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**Table 69.2** The disturbed mind: diagnostic strategy model

### Probability diagnosis

The 4 Ds:

- dementia
- delirium (look for cause)
- depression
- drugs: toxicity, withdrawal

### Serious disorders not to be missed

Cardiovascular:

- CVAs
- cardiac failure
- arrhythmia
- acute coronary syndromes

Neoplasia:

- cerebral
- cancer (e.g. lung)

Severe infections:

- septicaemia
- HIV infection
- infective endocarditis

Hypoglycaemia

Bipolar disorder/mania

Schizophrenia states

Anxiety/panic

Subdural haematoma

---

### Pitfalls (often missed)

Illicit drug withdrawal

Fluid and electrolyte disturbances

Faecal impaction (elderly)

Urinary retention (elderly)

Hypoxia

Pain syndromes (elderly)

Rarities:

- autoimmune encephalitis
- hypocalcaemia/hypercalcaemia
- kidney failure
- hepatic failure
- prion diseases (e.g. Creutzfeldt–Jakob disease)

---

### Seven masquerades checklist (all possible)

- Depression
- Diabetes
- Drugs
- Anaemia
- Thyroid disorder
- Spinal dysfunction (severe pain in elderly)
- UTI

---

### Is the patient trying to tell me something?

Consider anxiety, depression, emotional deprivation or upset, change in environment, borderline personality disorder, serious personal loss.

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## Glossary of terms

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**Alzheimer disease** A term used for both senile and presenile dementia, which has characteristic pathological degenerative changes in the brain.

**Cognition** The mental functions of perception, thinking and memory. It is the process of 'knowing'.

**Compulsions** Repeated, stereotyped and seemingly purposeful actions that the person feels compelled to carry out but resists, realising they are irrational (most are associated with obsessions).

**Confusion** Disorientation in time, place and person. It may be accompanied by a disturbed conscious state (see [TABLE 64.1](#), [CHAPTER 64](#)).

**Conversion** The process by which thoughts or experiences unacceptable to the mind are repressed and converted into physical symptoms.

**Delirium** (also termed 'toxic confusional state') A relatively acute disorder in which impaired consciousness is associated with abnormalities of perception or mood.

**Delusions** Abnormal, illogical or false beliefs that are held with absolute conviction despite evidence to the contrary.

**Dementia** An acquired, chronic and gradually progressive deterioration of memory, intellect and personality. Presenile dementia or early-onset dementia is dementia under 65 years of age. Senile dementia refers to older patients (usually over 80 years).

**Dissociation** A psychological disorder in which unpleasant memories or emotions are split off from consciousness and the personality and buried into the unconsciousness.

**Depersonalisation** An alteration in the awareness of the self—the person feels unreal.

**Hallucinations** Disorders of perception quite divorced from reality. Features:

- mostly auditory or visual
- a false perception—not a distortion
- perceived as normal perceptions
- independent of the person's will

**Illusions** False interpretations of sensory stimuli such as mistaking people or familiar things.

**Obsessions** Recurrent or persistent thoughts, images or impulses that enter the mind despite efforts to exclude them.

**Somatisation** The conversion of mental experiences or states into bodily symptoms, with no physical causation.

## Probability diagnosis

The diagnosis depends on the age and presentation of the patient. In a teenager the probable causes of acute confusion or irrational behaviour include drug toxicity or withdrawal, schizophrenia, severe depression or a behavioural disorder.

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It is the elderly who commonly present with confusion. The questions that must be asked are:

- Is the problem one of the 4 Ds—dementia, delirium, depression or drugs—or something else?
- If delirium is the problem, what is the cause?

Depression affects 15% of people over 65 and can mimic other causes of confusion and behavioural disturbance.

Significant prescribed drugs include hypnotics, sedatives, oral hypoglycaemics, antihypertensives, digoxin, antihistamines, anticholinergic drugs and antipsychotics.

## Serious disorders not to be missed

There are many serious underlying disorders that must be considered, especially with delirium (see TABLE 69.3). Cerebral organic lesions, including space-occupying lesions (e.g. cerebral tumour, subdural haematoma), severe infection (systemic or intracerebral) and cancer at any site, especially lung, breast, bowel or lymphoma, must be ruled out.

**Table 69.3** Important causes of delirium (typical examples of each group)

### Drug intoxication and drug sensitivity

Anticholinergics  
Antidepressants  
Sedatives  
Alcohol, opioids, etc.

### Withdrawal from substances of abuse and prescribed drugs

Alcohol  
Opioids  
Amphetamines  
Cannabis

## Sedatives and anxiolytics

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### Infections

- Specific:
    - Urinary tract
    - Lower respiratory (e.g. pneumonia)
    - Otitis media
    - Cellulitis
  - Intracranial:
    - Meningitis
    - Encephalitis
  - Systemic:
    - Infective endocarditis
    - Septicaemia
    - HIV
    - Other viral infections
    - Malaria
- 

### Metabolic disturbances

- Uraemia, hepatic failure
  - Electrolyte disturbances
  - Dehydration
- 

### Endocrine disturbances

- Diabetic ketoacidosis, hypoglycaemia
  - Hypothyroidism/hyperthyroidism
- 

### Nutritional and vitamin deficits

- Vitamin B complex deficiency (esp. B6, B12)
  - Wernicke encephalopathy
- 

### Hypoxia

- Respiratory failure, cardiac failure, anaemia
- 

### Vascular

- CVA
  - Acute coronary syndromes
- 

### Head injury and other intracranial problems

- Seizures
  - Complex partial seizures
- 

### 'Subtle' causes

- Pain (e.g. herpes zoster)
- Emotional upset

- Environmental change
  - Peri-operative/anaesthetic effect
  - Faecal impaction
  - Urinary retention
- 

The sudden onset of delirium may suggest angina, myocardial infarction or a cerebrovascular accident. Twenty per cent of patients with delirium also have underlying heart failure.<sup>3</sup>

## Pitfalls

There are many pitfalls, especially with drug toxicity or withdrawal from the so-called illicit drugs. In the elderly in particular, fluid and electrolyte disturbances, such as dehydration, hypokalaemia, hyponatraemia and hypocalcaemia, can cause delirium. Bowel disturbances such as faecal impaction or constipation can cause delirium and incontinence of both faeces and urine.

## Seven masquerades checklist

All the following disorders can cause disturbed or confused behaviour, particularly in the elderly:

- depression: a very important cause of ‘pseudodementia’
- drugs: toxicity or withdrawal (see TABLE 69.4 )
- diabetes: especially hypoglycaemia, which can occur with type 2
- anaemia: often from self-neglect or chronic blood loss
- thyroid disorders: both hyperthyroidism and hypothyroidism can present with disturbed behaviour; ‘myxoedemic madness’ may be precipitated by atropine compounds
- urinary tract infection: causes or contributes to 20% of cases of hallucinations or illusions<sup>2</sup>
- spinal dysfunction: with its many severe pain syndromes, such as sciatica, can be a significant factor

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**Table 69.4** Prescribed drugs that can cause delirium

Anticholinergic:

- antiparkinsonian (e.g. benzotropine)
- tricyclic antidepressants

Tranquillisers and hypnotics:

- major tranquillisers (e.g. chlorpromazine)
- minor tranquillisers (e.g. diazepam)
- hypnotics
- lithium

Anti-epileptics

Antihistamines 1 and 2

Antihypertensives

Corticosteroids

Cardiac drugs:

- digoxin
- diuretics
- beta blockers

Opioids

Sympathomimetics

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## Psychogenic factors

Apart from the primary psychiatric disorders of anxiety, depression, mania and schizophrenia, relatively simple and subtle social problems, such as loneliness, boredom, a domestic upset, financial problem or similar issues, can trigger a confusional state.

## The clinical approach

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### History

Developing rapport with the disturbed or confused patient is essential and can be helped by a warm handshake or a reassuring pat on the shoulder. The basis of the history is a careful account from relatives or witnesses about the patient's behaviour.

When communicating with the patient, speak slowly and simply (avoid shouting), face the patient and maintain eye contact. Important features are the past history and recent psychosocial history, including recent bereavement, family upsets and changes in environment. Search for evidence of depression and note any organic symptoms such as cough, constipation and so on.

### Mental status examination

The most practical bedside screening test of mental function is the Mental Status Questionnaire of Kahn and colleagues,<sup>4</sup> which includes 10 simple questions.

1. What is the name of this place?
2. What city are you in now?
3. What year is it?
4. What month is it?
5. What is the date today?
6. What year were you born?
7. When is your birthday?
8. How old are you?
9. Who is the prime minister/president?
10. Who was the prime minister/president before him?

(Interpretation: normal 9–10; mildly impaired 8–9; confused/demented 7 or less.)

Other MMSEs are presented in [CHAPTER 125](#) .

## Examination

The patient's general demeanour, dress and physical characteristics should be noted at all times. Assess the patient's ability to hear, see, speak, reason, obey commands, stand and walk. Any problems related to the special senses can cause confusion.

Look for features of alcohol abuse, Parkinson disease and hypothyroidism.

Examine the neurological system and keep in mind the possibility of a subdural haematoma, which may have followed a forgotten fall.

Don't omit the rectal examination to exclude faecal impaction, melaena, cancer and prostatomegaly (in males), and also check the bladder for evidence of chronic retention.

## Investigations

Investigations to consider for delirious or demented patients (unknown cause):

- urinalysis and microscopy
- cultures of blood and urine
- total and differential blood count; ESR

- blood glucose
- urea and creatinine and electrolytes
- calcium and phosphate
- vitamin D
- thyroid function tests
- liver function tests
- serum vitamin B12 and folate levels
- ECG/troponin (?acute coronary syndrome)
- chest X-ray
- cerebral CT scan, especially non-contrast CT
- syphilis serology
- HIV
- arterial blood gases

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## Behavioural emergencies: management of the acutely disturbed patient<sup>1</sup>

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Delirious or psychotic patients can be paranoid and respond defensively to the world around them. This behaviour can include aggressive and violent behaviour, resulting in danger to themselves, their friends and family and to their medical attendants.

Dangerousness should be assessed from features such as the patient's past history (especially previous dangerous behaviour), age, sex, recent stress, victim behaviour, muscle bulk, presence of weapons, degree of overactivity and the manner of handling of the present distress by others. The patient may be in a state of acute panic and trying to flee a situation or in an agitated psychotic state prepared to confront the situation. It should be emphasised that most violent individuals are not mentally ill.

Most cases require an injection (the ideal intravenous administration can be extremely difficult and hazardous), which is often interpreted as a physical attack. It may not be possible to diagnose the cause of the problem before giving the injection.

### Approach to management

- Assess the environment and don't move into the patient's space until in a position of control.
- React calmly. Communicate calmly and simply.
- State your task firmly and simply.
- Try to control the disturbed patient gently.
- Ensure the safety of all staff and make certain that heroics are not attempted in dangerous circumstances.
- An adequate number of staff to accompany the doctor is essential—six is ideal (one for immobilisation of each limb, one for the head and one to assist with drugs).<sup>1</sup>
- Patients should be placed on the floor in the prone position.

## Principles of sedative administration<sup>1</sup>

- Use the safest possible route of administration whenever possible (i.e. oral in preference to parenteral but often impractical). Intravenous administration has the lowest margin of safety.
- Parenteral administration should be restricted to severely disturbed patients.
- Closely monitor vital signs during and after sedative administration.
- Avoid intramuscular diazepam because of poor absorption.
- Be cautious of intravenous midazolam in such patients because of the risk of respiratory depression.
- Avoid benzodiazepines in patients with respiratory insufficiency. Haloperidol is an alternative.
- Patients have died from cardiopulmonary arrest after repeated sedative administration (especially benzodiazepines), so intensive monitoring is essential.

Monitor the following adverse effects:

- respiratory depression
- hypotension
- dystonic reactions, including choking
- neuroleptic malignant syndrome

## Treatment options<sup>1</sup>

The treatment in acute medical and psychiatric settings depends on the appropriate mode of

administration. Benzodiazepines are generally the drugs of first choice over antipsychotics in tranquillisation.<sup>5</sup> However, the IV route allows titration to the desired degree of sedation and has a more immediate effect.

## Intravenous medication

diazepam or midazolam 5 mg IV initially

*then*

2.5–5 mg increments IV, repeated every 3–4 minutes until required level of sedation (rousable drowsiness) is reached—up to a maximum of 20–30 mg, when specialist advice is needed, especially if further boluses are necessary

*and/or* droperidol 5–10 mg IV or olanzapine

## Intramuscular medication

If this route considered appropriate:

midazolam 5–10 mg IM

*or* (if history of benzodiazepine tolerance)

droperidol 5–10 mg IM

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*or*

olanzapine 5–10 mg repeated every 2–4 hours

*or*

combination midazolam/droperidol

(These first two injections can be repeated in 20 minutes if required. Droperidol is similar to haloperidol but more sedating. Keep in mind the rare but potentially fatal laryngeal dystonia with high doses—cover with benztrapine 2 mg IM.)

## Oral medication (if considered appropriate)

diazepam 5–20 mg (o), repeated every 2–6 hours (max. 120 mg/24 hours)

*or*

lorazepam 1–2 mg (o), repeated every 2–6 hours (max. 10 mg/24 hours)

If sedation is not achieved, add an antipsychotic medication, e.g. olanzapine 5–10 mg initially or risperidone 0.5–1 mg initially.

## Postdisturbance evaluation

Determine the likely cause, such as:

- acute organic brain syndrome: toxic causes, infection
- alcohol or drugs (illicit or prescribed): intoxication, withdrawal
- manic illness
- severe depression
- schizophrenic syndrome
- severe panic

## Postoperative cognitive dysfunction and dementia

This occurs in up to 12% of apparently previously cognitively normal patients who show a decline of cognitive function, especially in memory and executive function. The study<sup>6</sup> shows that it can occur in surgery other than cardiac surgery, even with regional anaesthesia. It is more common in those over 65 years. It is usually self-limiting and lasts 1–12 months.

### Acute organic brain syndrome (delirium)

The many labels of acute organic brain syndrome include:

- delirium
- acute confusional state
- toxic confusional state
- confusional episode
- acute brain syndrome

### Main clinical features

- Clouding of conscious state
- Disorientation
- Impaired attention
- Impaired memory

- Global cognitive defect—onset over days/hours

Refer to the box.

## DSM-5 criteria for delirium<sup>5</sup>

Diagnosis of delirium requires evidence of:

- A** Disturbance of consciousness, attention and awareness
- B** Clinical features appearing over a short period
- C** A change in cognition:
  - perceptual disturbance
  - incoherent speech
  - disorientation
  - memory impairment/deficit
- D** A & C not better explained by another disorder
- E** Evidence of a cause

## Other clinical features<sup>1</sup>

- The patients are usually elderly.
- Anxiety and agitation can be severe but in hypoactive deliria (usually due to metabolic disturbance) the conscious state can vary from drowsiness to coma.
- Odd behaviour with mood swings can occur.
- Psychotic symptoms can occur.
- Delusions are usually fleeting.
- The disturbance is usually worse at night and may be aggravated by sedation.
- Visual hallucinations are a feature of alcohol withdrawal.
- Attacks on bystanders may result (uncommon).

Always seek a cause.<sup>1</sup> A list of causes is presented in TABLE 69.3 . The most important causes are:

- infections (usually in urinary tract, lungs or ear, or systemic in young or elderly)

- prescribed drugs

### Anticholinergic delirium

Consider this cause (from drugs with anticholinergic properties or illicit substances). Features include hyperactivity, marked thought disorder, vivid visual hallucinations and very disturbed behaviour.

### Differential diagnosis of delirium

In the earlier stages it may mimic the various psychiatric disorders, including anxiety, depression, various hallucinatory states, particularly agitated schizophrenia (rarely), extreme manic states, complex partial seizures, dementia. Consider deafness. Delirium is common in the hospital setting, especially in patients  $\geq 65$  years of age.

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### Investigations

Investigations are those listed under ‘The clinical approach’ earlier in the chapter.

### Treatment

Principles:

- Acute delirium is a medical emergency.
- Establish normal hydration, electrolyte balance and nutrition.
- Consider alcohol withdrawal and give a trial of thiamine when the cause of delirium is unknown.
- Attend to helpful environmental factors (e.g. calm atmosphere, a night-light, orientation clues, presence of friends and relatives).
- Give oxygen if hypoxic, e.g. respiratory distress.

### Medication

Medication<sup>1</sup> may not be needed, but it will be if there are symptoms of anxiety, aggression or psychosis (doses for a fit adult). A single dose is usually adequate.<sup>1</sup>

For anxiety and depression:

midazolam 1.25–5 mg IM

For psychotic behaviour:

haloperidol 0.5 mg (o) as a single dose

*or*

olanzapine 2.5 mg (o) daily as a single dose

If oral administration is not possible or when parenteral medication is required (cover with benztropine 2 mg (o) or IM):

haloperidol 0.5 mg IM as a single dose

*or*

olanzapine 2.5 mg IM as a single dose

For anticholinergic delirium:

tacrine hydrochloride 15–30 mg with caution by slow IV injection (an antidote)

*Note:*

- For hypoxia, give oxygen.
- Avoid benzodiazepines, especially in children and in patients with respiratory insufficiency.
- Consider necessity for pain relief.
- Use lower doses of parenteral medications in the very old and frail.



## Dementia (chronic organic brain syndrome)

Dementia or neurocognitive disorder is an important diagnosis to consider in the elderly patient. The DSM criteria for dementia are presented in [CHAPTER 125](#).

The main feature of dementia is impairment of memory, especially recent memory, when the person cannot remember what has happened a few hours (or even moments) earlier but may clearly remember the events of the past.

The more serious behavioural changes encountered with dementia tend to occur in the advanced stages. However, these disturbances may be precipitated by illness such as infections, emotional upset and drugs. These serious disturbances include:

- uninhibited behaviour
- hallucinations (generally uncommon)
- paranoid delusions

If a stable patient becomes acutely disturbed, delirium should be suspected.

## Presenile dementia—Alzheimer type

The main features are:

- onset in late 50s and early 60s
- insidious onset
- early loss of short-term memory
- progressive decline in intellect
- death in 5–10 years
- more common in Down syndrome

### Differential diagnosis of dementia

There are two approaches to the differential diagnosis, including consideration of the classic causes of disturbed behaviour as summarised in the mnemonic in TABLE 69.5 .<sup>7</sup>

**Table 69.5** Differential diagnosis of dementias

<b>D</b>	=	<b>Delirium</b> drugs (see toxic)
<b>E</b>	=	<b>Emotional disorder</b> = depression endocrine = thyroid
<b>M</b>	=	<b>Memory</b> = benign forgetfulness
<b>E</b>	=	<b>Elective</b> = anxiety disorders/neuroses
<b>N</b>	=	<b>Neurological:</b> <ul style="list-style-type: none"><li>• CVA</li><li>• head trauma</li></ul>
<b>T</b>	=	<b>Toxic:</b> <ul style="list-style-type: none"><li>• drugs/medication</li><li>• metabolic disease</li></ul>
<b>I</b>	=	<b>Intellect</b> —low or retarded
<b>A</b>	=	<b>Amnesic disorders</b> —Korsakoff syndrome
<b>S</b>	=	<b>Schizophrenia</b> (chronic)

However, the foremost differential diagnosis should be ‘pseudodementia’ caused by severe depression.

A simple comparison between schizophrenia and dementia is shown in TABLE 69.6 .

**Table 69.6** Comparison of schizophrenia and dementia

	Dementia	Schizophrenia
<b>Onset</b>	Middle-aged or elderly	Young
<b>Memory</b>	Always impaired	Usually unaffected
<b>Delusions</b>	Rare	Frequent
<b>Hallucinations</b>	Uncommon	Frequent
<b>Thought broadcasting</b>	Never	Frequent

A vigorous search for a possible cause of dementia is warranted since there are a significant number of reversible causes. In particular, it is important to exclude the psychiatric conditions that may mimic dementia.

## Treatment<sup>1</sup>

- To control psychotic symptoms or disturbed behaviour:

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risperidone 0.5–2 mg (o) daily

or

olanzapine 2.5–10 mg (o) daily in 1 or 2 doses

- To control symptoms of anxiety and agitation:

oxazepam 7.5 mg (o) 1 to 4 times daily. Avoid benzodiazepines for more than 2 weeks

- For depression—administer antidepressants

- Treat any folate and vitamin D deficiency

## The acute psychotic patient

Acute psychosis is the presence of the mental state where appreciation of reality is impaired as

evidenced by the presence of typical psychotic symptoms such as delusions, hallucinations, mood disturbance and bizarre behaviour.<sup>8</sup> Refer to the diagnostic strategy for hallucinations in TABLE 69.7 .

**Table 69.7** Hallucinations: diagnostic strategy model

**Probability diagnosis**

- Drugs (illicit or prescribed)
- Alcohol (acute or chronic)
- Schizophrenia
- Febrile delirium
- Affective (mood) disorders
- Drug withdrawal (incl. alcohol, hypnotics)
- Dementias (esp. Lewy body)

**Serious disorders not to be missed**

Vascular:

- cerebrovascular disease
- migraine (luminous)

Infections:

- encephalitis/meningitis
- septicaemia
- any serious febrile illness

Tumours:

- cerebral tumours
- cancer treatment

Other:

- hypoxia
- liver failure
- metabolic/electrolyte imbalance
- dehydration

**Pitfalls (often missed)**

- Major depression
- Extreme fatigue
- Vitamin deficiency (esp. B group)
- Seizure disorders (esp. complex partial)
- Rarities:*
  - narcolepsy

- post-concussion
- bereavement
- multiple sclerosis

### Seven masquerades checklist

Depression

Diabetes

Drugs (iatrogenic/social, illicit)

Thyroid/other endocrine (hypothyroid)

Urinary tract infection (esp. elderly)

### Is the patient trying to tell me something?

Consider conversion disorder (hysteria); fabrication.

The differential diagnoses of patients presenting with psychoses is presented in [TABLE 69.8](#) .

### Table 69.8 Causes of psychoses<sup>8</sup>

Functional psychoses:

- schizophrenia
- schizopreniform disorder (shorter duration of symptoms)
- schizoaffective disorder (core symptoms of schizophrenia + mood symptoms)
- bipolar mood disease (depressed or manic phase)

Drug-induced psychoses

Organic-based psychoses

Other:

- delusional disorder (paranoid psychoses)
- brief psychotic disorder (rapid resolution)
- folie à deux (psychosis occurring simultaneously in two close associates)

## Early diagnosis

Early recognition of a psychosis, particularly schizophrenia, is extremely important, as early intervention leads to improved outcomes. Early or prodromal symptoms include the following:

- social withdrawal
- reduced attention and concentration

- reduced drive and motivation
- depressed mood
- anxiety
- irritability/agitation
- suspiciousness
- sleep disturbance
- deterioration in role functioning

It is appropriate to ask the correct questions in order to elicit psychotic symptoms. These are presented in [TABLE 69.9](#).

**Table 69.9** Questions for eliciting psychotic symptoms

<b>Anxiety</b>	Have you been feeling especially nervous or fearful? Have you felt tense and shaky, or experienced palpitations?
<b>Depressed mood</b>	Have you been feeling sad or 'down in the dumps' recently, not enjoying activities as much as before?
<b>Elevated mood</b>	Have you been feeling especially good in yourself, more cheerful than usual and full of life?
<b>Auditory hallucinations</b>	Do you hear voices of people talking to you even when there is no one nearby?
<b>Thought insertion</b>	Have you felt that thoughts are being put into your mind?  Do you experience telepathy?
<b>Thought withdrawal</b>	Have you experienced thoughts being taken out of your mind?
<b>Thought broadcasting</b>	Have you felt that other people are aware of your thoughts?
<b>Thought echo</b>	Have you experienced voices or people echoing your thoughts?
<b>Delusion of control</b>	Have you felt under the control or influence of an outside force?
<b>Delusions of reference</b>	Do programs on the television or radio hold special meaning for you?
<b>Delusions of persecution</b>	Do you feel that you are being singled out for special treatment? Is there a conspiracy against you?

<b>Delusions of grandeur</b>	Do you feel special, with unusual abilities or power?
<b>Delusions of guilt</b>	Do you believe that you have sinned or have done something deserving punishment?

Source: Reproduced with permission from Keks N , Blashki G. The acutely psychotic patient: assessment and initial management. Aust Fam Physician, 2006; 35 (3): 90–4.

## DSM-5 key diagnostic criteria<sup>5</sup>—schizophrenia

- A** Two or more of the following, each present for a significant portion of time during a 1-month period
- 
- B** Social, learning or occupational dysfunction
- C** Continuous signs of disturbance for at least 6 months
- D** No evidence of other psychoses, e.g. bipolar
- E** Not attributable to effects of substance abuse or other medical condition

## Schizophrenia and associated disorders

The term ‘schizophrenia’ (Bleuler, 1911) refers to a group of severe psychiatric illnesses characterised by severe disturbances of emotion, language, perception, thought processes, volition and motor activity. The causes of schizophrenia disorders are unknown, but genetic factors and drug abuse are implicated.

### Signs and symptoms of schizophrenia

- Positive
  - delusions
  - hallucinations
  - thought disorder
  - disorganized speech and behaviour
- Negative

flat affect

poverty of thought

lack of motivation

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social withdrawal

reduced speech output

- Cognitive

- distractibility

- impaired working memory

- impaired executive function (e.g. planning)

- impaired insight

- Mood

- mania (elevation)

- depression

Other features include:

- bizarre behaviour
- subject to tension, anxiety or depression
- deterioration in work and study performance
- peak incidence 15–25 years<sup>9</sup>—smaller peak at 40 years
- lifetime prevalence 1 in 100
- equal sex incidence
- high risk of suicide

## Differential diagnosis

Organic factors need to be excluded, especially drugs:

- amphetamines
- hallucinogens (e.g. LSD)

- marijuana

Also consider complex partial seizures and a personality disorder. Other psychoses are presented in [TABLE 69.8](#).

A comparison of delirium, dementia and functional psychosis is presented in [TABLE 69.10](#).

**Table 69.10** Comparison of the clinical features of delirium, dementia and acute functional psychoses<sup>9</sup>

Feature	Delirium	Dementia	Acute psychosis
<b>Onset</b>	Rapid	Slow—insidious	Rapid
<b>Duration</b>	Hours to weeks	Months to years	Depends on response to treatment
<b>Course over 24 hours</b>	Fluctuates—worse at night	Minimal variation	Minimal variation
<b>Consciousness</b>	Reduced	Alert	Alert
<b>Perception</b>	Misperceptions common, especially visual	Misperceptions rare	May be misperceptions
<b>Hallucinations</b>	Common, visual (usually) or auditory	Uncommon	Common, mainly auditory
<b>Attention</b>	Distractable	Normal to impaired	Variable—may be impaired
<b>Speech</b>	Variable, may be incoherent	Difficulty finding correct words	Variable: normal, rapid or slow
<b>Organic illness or drug toxicity</b>	One or both present	Often absent	Usually absent

## Management

Drug treatment is only a part of total management. Explanation and appropriate reassurance to the family with patient and family supportive care is obviously essential. Supportive psychotherapy is important in all phases. A team approach is necessary to cope with the disorder, which usually has a devastating effect on the family. Referral for specialist care is appropriate.

## Acute phase

- Hospitalisation usually necessary
- Drug treatment for the psychosis<sup>1</sup>

Drug treatment may include the first-generation (typical or conventional) antipsychotics such as haloperidol and chlorpromazine, which are effective for managing the ‘positive’ symptoms, or the second generation (atypical) antipsychotics such as risperidone, olanzapine, quetiapine, clozapine, amisulpride and aripiprazole, which in addition are more effective at treating the ‘negative’ and other symptoms of schizophrenia.<sup>10</sup>

The usual practice rule is to start with a second-generation antipsychotic at a low dose and titrate upwards at a rate and to a level that is optimal for the patient. Patients with a first psychotic episode may respond to lower than usual doses.<sup>5</sup>

- l. When oral medication is possible, first-line treatment (for the first episode) is one of (with starting doses):<sup>1,11,12</sup>

amisulpride 100 mg nocte

asenapine 5 mg sublingual bd

aripiprazole 10 mg once daily

olanzapine 5 mg nocte

paliperidone CR 3 mg nocte

quetiapine 50 mg bd → 200 mg bd (by day 5)

sertindole 4 mg daily

risperidone 0.5–1 mg nocte → 2 mg nocte

ziprasidone 40 mg bd → 80 mg bd (risk of ↑ QT)

zuclopentixol 20 mg nocte

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### Practice tip

Typical initial oral therapy:

olanzapine 5 mg

or

risperidone 0.5 mg

If response is inadequate in 3 weeks, increase the dose according to prescribing guidelines.

If no response after 4–6 weeks consider a change to:<sup>13</sup>

- an alternative second-generation agent (above)

*or*

- a first-generation antipsychotic such as:

chlorpromazine 200 mg once daily → 500 mg

haloperidol 1.5 mg once daily → 7.5 mg

trifluoperazine 2 mg bd

! Parenteral medication should be avoided if possible in acute care, but if required:<sup>1,12</sup>

haloperidol 2.5–10 mg IM initially, up to 20 mg in 24 hours, depending on the response

*or*

olanzapine 5–10 mg IM initially (do not use with benzodiazepines concurrently)

*add*

benztropine 1–2 mg (o) bd (to avoid dystonic reaction)

*or*

zuclopentixol acetate 50–150 mg IM as a single dose

If dystonic reaction:

benztropine 1–2 mg IV or IM

If very agitated use:

diazepam 5–10 mg (o) up to 40 mg/day or 5–10 mg IV

## Chronic phase

Long-term antipsychotic medication is recommended to prevent relapse.<sup>1</sup>

- Examples of oral medication regimens:<sup>12,13</sup>

olanzapine 5–10 mg (o) nocte

*or*

risperidone 0.5–1 mg (o) bd, up to 2–4 mg

*or*

quetiapine 150 mg (o) bd

- Aim for lowest possible dose to maintain control.
- Chlorpromazine is not recommended for long-term use because of photosensitivity reactions.
- Use depot preparations if compliance is a problem (usually test dose first).<sup>1,11</sup> Examples include:

haloperidol decanoate 50 mg IM initially, then 50–200 mg every 4 weeks

*or*

flupenthixol decanoate 10 mg IM initially, then 20–40 mg every 2–4 weeks

*or*

risperidone 25–25 mg IM initially, then every 2 weeks, titrated to clinical response

*or*

zuclopentixol 100 mg IM initially, then titrated to 200–400 mg every 2–4 weeks

Tips with depot preparations:

- Start with IM test doses and then titrate to recommended controlling levels (half or full starting dose).
- May take 2–4 months to produce a stable response, so oral supplements may be necessary.
- Not as effective as oral therapy.
- Give as deep IM injection with 21 gauge needle in buttock.
- Use lowest possible dose to avoid tardive dyskinesia.
- Reassess at least every 3 months.
- Closely monitor patient for movement disorders.

**Drug-resistant schizophrenia<sup>14,15</sup>**

Consider other causes (e.g. substances abuse). ECT may help the agitated patient, especially if catatonic. Consider a trial of clozapine (12.5 mg (o) bd initially, increasing to 200–600 mg daily), with strict monitoring for blood dyscrasias and cardiotoxicity, or olanzapine (5–20 mg daily). A trial of adjunctive ECT should be considered in patients who fail to respond to clozapine.

## Movement disorders from antipsychotic medication<sup>1</sup>

### Acute dystonias

- Usually bizarre muscle spasms affect face, neck, tongue and trunk
- Oculogyric crises, opisthotonus and laryngeal spasm

Treatment:

benztropine 1–2 mg IV or IM

### Akathisia

- Subjective or objective motor restlessness of feet and legs
- Generally later onset in course of treatment

Treatment:

- reduce dosage until akathisia less troublesome or substitute thioridazine
- can use oral propranolol, diazepam or benztropine as a short-term measure

### Parkinsonian

- Seen relatively early in treatment
- The akinesia can be confused with drug-induced depression

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Treatment:

- use lower dose or substitute a phenothiazine in low dosage
- alternatively, use benztropine or benhexol

### Tardive dyskinesia<sup>1</sup>

Tardive dyskinesia is a syndrome of abnormal involuntary movements of the face, mouth, tongue, trunk and limbs. This is a major problem with the use of long-term antipsychotic drugs and may occur months or years (usually) after starting treatment and with drug withdrawal.

Avoid prolonged use of metoclopramide.

Differential diagnosis:

- spontaneous orofacial dyskinesia
- senile dyskinesia
- ill-fitting dentures
- neurological disorders causing tremor and chorea

If drug withdrawal is ineffective, use tetrabenazine 12.5 mg (o) daily, increasing as necessary.<sup>13</sup>  
The risks and benefits of continuing therapy have to be weighed.

*Note:* Because of the difficulty with managing tardive dyskinesia, prevention in the form of using the lowest possible dosage of antipsychotic medication is essential. This involves regular review and adjustment if necessary.

### **Neuroleptic (antipsychotic) malignant syndrome**

This is a potentially fatal adverse effect that can develop at any time. It develops in hours to days.

Syndrome: high temperature, muscle rigidity, altered consciousness. Milder variants can occur (refer to [CHAPTER 42](#) ).

Treatment:

- discontinue medication
- ensure adequate hydration with IV fluids
- if life-threatening:
  - bromocriptine 2.5 mg (o) bd, gradually increasing to 5 mg (o) tds

*and*

dantrolene 50 mg IV every 12 hours for up to 7 doses

- consultant referral

### **Cardiac dysfunction**

Various psychotropic agents, particularly the phenothiazines, are prone to cause the adverse effect of prolongation of the QT interval with potential severe outcomes. Patients should be monitored.

## Bipolar disorder

The mood disorders are divided into depressive disorders and bipolar disorders (see [CHAPTER 68](#)). Bipolar is a broad term to describe a recurrent illness with episodes of either mania or depression with return to normal function in between. The swing in moods in bipolar disorders (manic depressive disorders) is illustrated in [FIGURE 68.1](#). It affects 1% of the population.

### DSM-5 criteria for a manic episode<sup>5</sup>

- A** Distinct period for at least 1 week of abnormal and persistent elevated, expansive or irritable mood
- B** Three or more of these unusual features:
  1. inflated self-esteem or grandiosity
  2. decreased need for sleep
  3. talkative/accelerated speech
  4. racing thoughts or flights of ideas
  5. distractability as reported or observed
  6. increased goal-directed activity or psychomotor agitation
  7. excessive activity with 'painful' consequences
- C** Marked impaired social or occupational functioning or need for hospitalisation or psychotic features
- D** Episode not due to substance abuse or other medical condition

**A–D** = a manic episode: at least one in a lifetime for diagnosis

Bipolar I disorder has one fully fledged manic or mixed episode and usually depressive episodes.

Bipolar II disorder is defined as a major depressive episode with at least one hypomanic episode lasting a minimum of 4 days but no classic manic episodes.

The symptoms of mania may appear abruptly, usually in the teens or young adulthood.

Typical inherent features, in addition to the above, include:

- reckless behaviour, overspending

- hasty decisions (e.g. job resignation, hasty marriages)
- impaired judgment
- increased sexual drive, energy and activity
- poor insight into the problem
- variable psychotic symptoms—paranoia, delusions, auditory hallucinations

*Note:* The peak onset is in early adult life. The exact cause is unknown, but there is a strong hereditary basis. Episodes may be precipitated by stress.

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‘Hypomania’ is the term used to describe the symptoms of mania that are similar to but less severe (without criterion C) and of shorter duration.

The subsequent major depressive phase is associated with a high risk of suicide.

Good questions to ask the patient with suspected ‘bipolar’ disorder:

- How have you been feeling in yourself?
- Have you felt especially good about yourself?
- Do you feel that you are special or have special powers?
- Have you been spending more than usual?
- Have you been needing less sleep than usual?

## Management of acute mania<sup>1,16</sup>

This is a medical emergency requiring hospitalisation for protection of both family and patient. Involuntary admission is usually necessary. It may be a first episode or a relapse due to poor treatment compliance or substance abuse. A recent meta-analysis indicates that antipsychotics are the most efficacious drugs.

### Treatment

First line:<sup>1</sup>

olanzapine 5 mg (o) nocte initially, then increasing to 10 mg

or

risperidone 1 mg (o) nocte initially, then increasing to 2 mg

Second line:

haloperidol or a second-generation antipsychotic, e.g. aripiprazole<sup>1,17</sup> quetiapine, asenapine

*or*

lithium carbonate 750–1000 mg (o) daily in 2 or 3 divided doses increasing according to serum levels (warn patients and family about toxicity)

*or (a mood stabilising agent)*

sodium valproate 200–400 mg (o) bd initially

*or*

carbamazepine 100–200 mg (o) bd initially

If a parenteral antipsychotic drug is required:

haloperidol 5–10 mg IM or IV

Repeat in 15–30 minutes if necessary (risk of tardive dyskinesia). Change to oral medication as soon as possible.

Failure to respond to treatment:<sup>18,19,20</sup>

- ensure maximum concentration of first drug
- switch to a different drug, e.g. olanzapine to lithium
- combine drugs, e.g. second-degree antipsychotic + lithium
- ECT is of proven benefit for recalcitrant problems

Remember to provide supportive psychotherapy with appropriate psychosocial interventions.

### **Prophylaxis for recurrent bipolar disorder**

Over 90% will have a recurrence at some time: consider medication if two or more episodes of either mania or depression in the previous 4 years.

### **Recommended prophylactic agents<sup>5</sup>**

lithium carbonate 125–500 mg (o) bd then adjusted

*or*

second-generation antipsychotic agent

*or (if depression prominent)*

lamotrigine or carbamazepine or sodium valproate

Use long-term lithium (e.g. 3–5 years). Target plasma level for maintenance is usually 0.6–0.8 mmol/L. A US study recommended lithium as the prime mood stabiliser.<sup>17</sup>

- If poor response, use another agent.
- Unwanted side effects of lithium include:
  - a fine tremor
  - muscle weakness
  - weight gain
  - gastrointestinal symptoms
  - hypothyroidism
  - nephrotoxicity

- With anti-epileptics, adjust dosage according to clinical response and toxicity.

### Management of bipolar depression<sup>11,18</sup>

This is a difficult component to treat and antidepressants should not be used alone.<sup>12</sup> Many mood-stabilising agents appear to have a bimodal (antidepressant and antimania) effect and can be useful in the absence of classical antidepressants.<sup>16</sup>

A recommended regimen is:

an antidepressant (e.g. SSRI, SNRI or MAOI)

*plus*

lithium, valproate, carbamazepine, quetiapine, lamotrigine or olanzapine (one of these used for prophylaxis). Avoid using antidepressants alone.

Antidepressants are usually withdrawn within 1–2 months because of a propensity to precipitate mania.

ECT is an effective treatment for bipolar depression while psychological therapies such as CBT and psychoeducation have proven efficacy.

Bipolar I patients usually recover but proceed to have further episodes of depression or mania.<sup>12</sup>

### Liaison with family and carers

Promote a caring support and psychoeducation program for patient and family. Educate about the patient's 'relapse signature' for a manic or depressive episode.

## **Body dysmorphic disorder<sup>5</sup>**

Body dysmorphic disorder, which is a type of somatoform anxiety disorder, is characterised by a preoccupation with the belief that some aspect of physical appearance is abnormal, unattractive or diseased. The person's concern and distress is out of proportion to any imagined or actual defect and usually not amenable to reassurance. This preoccupation causes significant functional impairment. The condition rarely presents directly and may be over-represented in the area of dermatology or plastic surgery. It begins in late childhood or early adolescence. The person's focus is on the face, head or secondary sexual characteristics.

Patients may be helped by counselling and psychotherapy including CBT. There is clinical evidence that SSRIs help if the symptoms suggest an obsessive-compulsive disorder. An antipsychotic agent may help where beliefs are delusional or in the context of a psychotic disorder.

## **Depression**

Depression is very common and presents in a great range of severity. In the context of 'the disturbed patient' depression can be confused with dementia or a psychosis, particularly if the following are present:

- psychomotor agitation
- psychomotor retardation
- delusions
- hallucinations

## **Assessment<sup>1</sup>**

The following questions need to be addressed:

- Is the depression primary (i.e. not secondary to another psychiatric condition such as schizophrenia or anxiety disorder)?
- Is it part of a bipolar disorder? Has there been a previous manic or hypomanic episode? If so, a different approach to treatment is required.
- Is the depression caused by another illness or physical factor (e.g. hypothyroidism, cerebrovascular disease or medication)?
- Is the patient psychotic?

- Is the patient a suicide risk?

The treatment of depression is presented in [CHAPTER 10](#) .

## Psychoactive substance use disorders

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It is important for the GP to be aware of the effects of self-administration of psychoactive substances, especially their toxic or withdrawal effects. They form a significant consideration for the differential diagnosis of disturbed patient behaviour. The following substances can cause these effects.

### Alcohol

Toxic and withdrawal effects, including delirium tremens, are outlined in [CHAPTER 12](#) . Abrupt withdrawal can cause symptoms ranging from tremors, agitation and dysphoria (feeling thoroughly miserable) to fully developed delirium tremens. Epileptic seizures may also occur.

### Barbiturate dependence

Tolerance and symptoms on withdrawal are the main features. Barbiturate withdrawal is a very serious, life-threatening problem and may be encountered in elderly people undergoing longstanding hypnotic withdrawal. Symptoms include anxiety, tremor, extreme irritability, twitching, seizures and delirium.

### Management<sup>1</sup>

Undertake withdrawal with medical supervision as an inpatient.

Transfer the patient to phenobarbitone or diazepam.

phenobarbitone 120 mg (o) hourly until sedation

*or*

phenobarbitone 30 mg for each 100 mg of shorter-acting barbiturate

reduce the dose gradually over 10–14 days

*or*

diazepam 20–40 mg orally, daily

reduce the dose gradually over 10–14 days

### Benzodiazepine dependence

Withdrawal symptoms in the dependent patient include anxiety, restlessness, irritability, palpitation and muscle aches and pains, but delirium and seizures are uncommon except with very high doses. Refer to [CHAPTER 70](#) for the adverse effects of benzodiazepines. The shorter the half-life, the greater the dependence.

Withdrawal is best achieved by supervising a gradual reduction in dosage aided by relaxation techniques and behavioural strategies to help patients cope with insomnia and anxiety.

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Refer to [CHAPTER 12](#) for the effects of opioid dependence, stimulant substance abuse, hallucinogen abuse and cannabis use and dependence.

## Psychiatric disorders of childhood and adolescence<sup>1</sup>

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The following disturbance problems do occur and must be taken seriously, especially the potential for suicide in the second decade. Many of these disorders are presented in more detail in [CHAPTER 87](#).

### Attention deficit hyperactivity disorder

Clinical features:

- short attention span
- distractibility
- overactivity
- impulsiveness
- antisocial behaviour

### Depression

Major depression follows the same criteria as for adults. Suicidal ideation has to be considered and taken very seriously if present. Imipramine is probably the drug of choice.

### Bipolar disorders

Mania is seldom diagnosed before puberty. Adolescents may present (uncommonly) with symptoms of mania or hypomania.

### Schizophrenia and related disorders

Schizophrenia is rare before puberty. The criteria for diagnosis are similar to adults:

- delusion
- thought disorder
- hallucinations
- 6 months or more of deterioration in functioning

## Autism

Aggression and irritability can be a feature, especially during adolescence.

## Tourette syndrome

Behavioural problems can be part of this syndrome, which requires the attention of an experienced consultant.

## Obsessive-compulsive disorders

In about one-third of cases the onset is between 5 and 15 years of age.

## When to refer<sup>21</sup>

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Indications for referral to a psychiatrist:

- severe depression
- high suicide risk
- actual suicide attempt: recent or in the past
- suspected psychiatric disorders in the elderly: ?depression or schizophrenia; ?depression or dementia
- failure to improve with treatment
- poor family and social supports

## Patient education resources

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Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Anger management

- Anxiety disorder
- Bipolar disorder
- Dementia
- Depression
- Schizophrenia

## Resources

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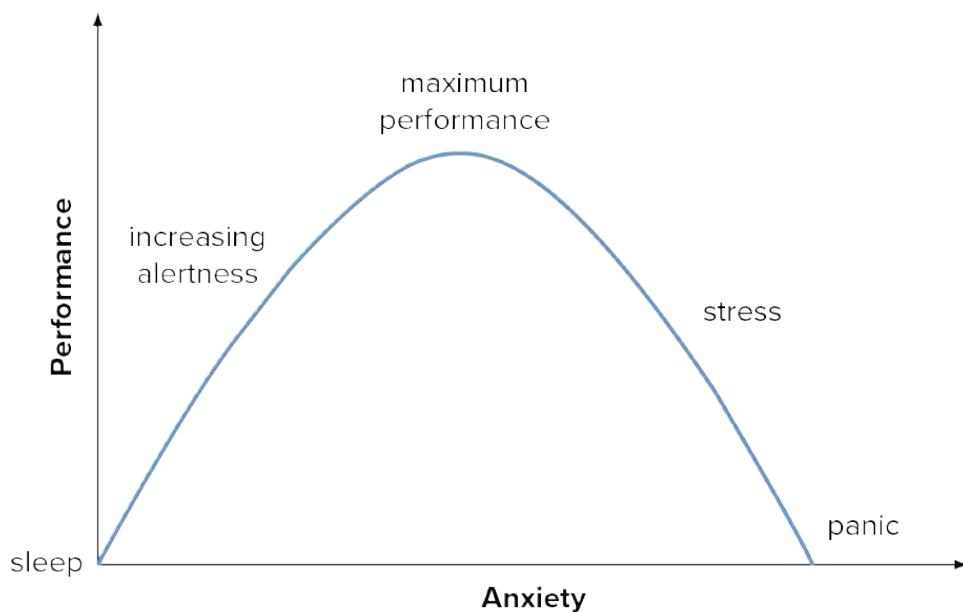
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## 70 Anxiety disorders

*Anxiety is a thin stream of fear trickling through the mind. If encouraged, it cuts a channel into which all other thoughts are drained.*

ARTHUR SOMERS ROCHE (1883–1935)

Anxiety is a state of nervousness or uneasiness in response to an actual or perceived stressor. It has three essential components: psychological (feelings, e.g. frightened),<sup>1</sup> somatic (physical, e.g. palpitations) and cognitive (thoughts, e.g. loss of control). Anxiety is a normal human physiological response that helps us respond to potential threats or dangers. We cannot get by without it. With an increase in anxiety, our performance should increase accordingly (e.g. preparing for exams). When we have too much anxiety, however, a further increase in anxiety will lead to our performance dropping off, described by the Yerkes–Dodson curve (see FIG. 70.1 ). The optimal place to be on the curve is to the left of the peak, so we have a positive response in our performance to an increase in stress. Being at the peak or to the right will make us vulnerable to stress.



## **FIGURE 70.1** Yerkes–Dodson curve

Anxiety becomes a problem when the stressor overwhelms us, resulting in poor performance, or if the unwanted consequences of the anxiety response give us undesirable physiological consequences. The stressor causes limbic activation, which in turn leads to autonomic and neuro-endocrine activity (down-regulation) that causes various physiological responses, including activation of the hypothalamic–pituitary axis. Blood flows from the gut to the skeletal muscles, smooth muscle contracts in the bowels and increases nausea, muscle tension increases, pupils dilate, the heart rate increases and blood pressure rises. These changes account for various somatic symptoms commonly seen in anxiety disorders (e.g. tension headaches, palpitations, eye strain and irritable bowel-like symptoms). In high stress situations, a flight–fright–freeze response can ensue.

The physiological role of anxiety and the way in which excess anxiety can cause these physical symptoms can be explained to patients suffering from anxiety disorders—and the GP is very well placed to conduct such psycho-education. Patients suffering from somatic symptoms are often unaware of the physiological consequences of anxiety and usually find this clarification comforting. It can help motivate patients to pursue strategies to reduce them.

## **Prevalence and classification of anxiety**

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Anxiety disorders affect 14% of the population,<sup>2</sup> with many people fulfilling the criteria for multiple anxiety disorders and/or a common co-diagnosis of depression.<sup>3</sup> The criteria for defining anxiety, as for many mental health disorders, have broadened over time, so it is difficult to compare historic prevalence figures. Specific phobia is the most commonly reported anxiety-related diagnosis (1 in 5 women and 1 in 10 men), and PTSD is the most common disorder (over 6%).<sup>2</sup> Anxiety disorders listed in the DSM-5 are:<sup>4</sup>

- separation anxiety disorder
- selective mutism
- specific phobia
- social anxiety disorder (social phobia)
- panic disorder
- panic attack (specifier)
- agoraphobia
- generalised anxiety disorder
- substance/medication-induced anxiety disorder

- illness anxiety disorder
- anxiety due to another medical condition
- other specified anxiety disorder
- unspecified anxiety disorder

Other conditions discussed in this chapter include:

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- obsessive-compulsive disorder
- body dysmorphic disorder (see [CHAPTER 69](#) )
- post-traumatic stress disorder
- acute stress disorder
- adjustment disorder with anxious mood
- somatic symptom disorder

## Generalised anxiety disorder

Generalised anxiety comprises excessive anxiety and worry about various life circumstances and is not related to a specific activity, time or event such as trauma, obsessions or phobias. It affects up to 5% of the population. There is an overlap between generalised anxiety disorder (GAD) and other anxiety disorders.

General features:

- persistent unrealistic and excessive anxiety
- worry about a number of life circumstances for 6 months or longer

### Diagnostic criteria for generalised anxiety disorder

Three or more of:

- restless, 'keyed up' or 'on edge'
- easily fatigued
- difficulty concentrating or 'mind going blank'
- irritability

- muscle tension
- sleep disturbance

## Clinical features

### Psychological

- Apprehension/fearful anticipation (even of dying)
- Irritability
- Exaggerated startle response
- Sleep disturbance and nightmares
- Impatience
- Panic
- Sensitivity to noise
- Difficulty concentrating or ‘mind going blank’

### Physical

- Motor tension:

muscle tension/aching

tension headache

trembling/shaky/twitching

restlessness

tiredness/fatigue

- Autonomic overactivity:

dry mouth

palpitations/tachycardia

sweating/cold, clammy hands

flushes/chills

difficulty swallowing or ‘lump in throat’

diarrhoea/abdominal distress

frequency of micturition

difficulty breathing/smothering feeling

dizziness or lightheadedness

## Symptoms and signs according to systems

- *Neurological*: dizziness, headache, trembling, twitching, shaking, paraesthesia
- *Cardiovascular*: palpitations, tachycardia, flushing, chest discomfort
- *Gastrointestinal*: nausea, indigestion, diarrhoea, abdominal distress
- *Respiratory*: hyperventilation, breathing difficulty, air hunger
- *Cognitive*: fear of dying, difficulty concentrating, ‘mind going blank’, hypervigilance

## Diagnosis of generalised anxiety disorder

The diagnosis is based on:

- history—it is vital to listen carefully to what the patient is saying
- exclusion of organic disorders simulating anxiety by history, examination and appropriate investigation, e.g. TFTs, ECG, CXR, drug screen
- exclusion of other psychiatric disorders, especially depression and adjustment disorder with anxious mood (comorbidity is a feature of anxiety)

*Note:* Anxiety and major depression often coexist.

Main differential diagnoses (note that this conforms to the seven masquerades list):

- depression
- substance abuse—alcohol or drug (including benzodiazepine) dependence/withdrawal
- hyperthyroidism
- angina and cardiac arrhythmias
- iatrogenic drugs
- caffeine intoxication

These organic (medical) causes need to be ruled out.

## Important checkpoints

These self-posed questions should be considered by the family doctor before treating an anxious patient:

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- Is the anxiety primary or secondary?
- Is this hyperthyroidism?
- Is this depression?
- Is this normal anxiety?
- Is this mild anxiety or simple phobia?
- Is this moderate or severe anxiety?
- Is this an adjustment disorder?

## Management

The management applies mainly to generalised anxiety, as specific psychotherapy is required in other types of anxiety. Much of the management can be carried out successfully by the family doctor using brief counselling and support. Cognitive behaviour therapy (CBT), in which maladaptive thinking, feelings, perceptions and related behaviours are identified, assessed, challenged and modified, can be of considerable benefit.<sup>5</sup> Exercise, both low or high intensity, has been shown to decrease anxiety symptoms. Hence psychological therapy and non-drug strategies are first-line therapy for most anxiety disorders.<sup>6</sup>

**Table 70.1** Significant differential diagnoses of anxiety

### Psychiatric disorders

- Depression
- Drug and alcohol dependence/withdrawal
- Benzodiazepine dependence/withdrawal
- Personality disorder, e.g. borderline
- Schizophrenia

Acute or chronic organic brain disorder

Early dementia

### **Organic disorders**

Drug-related:

- amphetamines
- bronchodilators
- caffeine excess
- ephedrine/pseudoephedrine
- levodopa
- thyroxine

Cardiovascular:

- angina
- cardiac arrhythmias
- mitral valve prolapse

Endocrine:

- hyperthyroidism
- phaeochromocytoma
- carcinoid syndrome
- hypoglycaemia
- insulinoma

Neurological:

- epilepsy, especially complex partial seizures
- acute brain syndrome

Respiratory:

- asthma
- acute respiratory distress
- pulmonary embolism

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### **Principles of management<sup>6,7</sup>**

- Psychological interventions (e.g. ‘life coaching’ and CBT) are first line.

- Give careful explanation and reassurance:

explain the reasons for the symptoms

be aware that patients often ‘worry about worrying’ (e.g. that anxiety is dangerous, that they are going crazy or ‘losing it’)<sup>7</sup>

reassure the patient about the absence of organic disease (can only be based on a thorough examination and appropriate investigations)

direct the patient to appropriate resources to give insight and support

- Provide practical advice on ways of dealing with the problems.
- Advise on the avoidance of aggravating substances such as caffeine, nicotine and other drugs.
- Advise on general measures such as stress management techniques, relaxation programs, mindfulness and regular exercise and if possible organise these for the patient (don’t leave it to the patient).
- Advise on coping skills, including personal and interpersonal strategies, to manage difficult circumstances and people (in relation to that patient).
- Provide ongoing supportive psychotherapy.

TABLES 70.2 and 70.3 list a number of good sources of reliable information about anxiety.<sup>8</sup>

**Table 70.2** Australian websites providing information about anxiety

Provider and hosting organisation	Website
Beyond Blue, The National Depression Initiative	<a href="http://www.beyondblue.org.au/home">www.beyondblue.org.au/home</a> <a href="http://www.youthbeyondblue.com">www.youthbeyondblue.com</a> (for young people aged 12–25 years)
Black Dog Institute, Prince of Wales Hospital, University of New South Wales	<a href="http://www.blackdoginstitute.org.au">www.blackdoginstitute.org.au</a> <a href="http://www.biteback.org.au">www.biteback.org.au</a> (for young people aged 12–18 years)
Centre for Clinical Interventions, Government of Western Australia, Department of Health	<a href="http://www.cci.health.wa.gov.au">www.cci.health.wa.gov.au</a>
Clinical Research Unit for Anxiety and Depression (CRUfAD), St Vincent’s Hospital, Sydney	<a href="http://www.crfad.org">www.crfad.org</a>
ReachOut Australia	<a href="http://au.reachout.com">http://au.reachout.com</a> (for young people aged 14–25)

years)

**Table 70.3** Openly accessible Australian-based interactive internet programs for anxiety

Organisation and program name	Website
CRUfAD clinical programs	<a href="http://www.crfad.org">www.crfad.org*</a>
e-hub Australian National University, MoodGYM	<a href="http://www.moodgym.anu.edu.au">www.moodgym.anu.edu.au</a>

\*in-house clinical support is supplied, but costs apply

## Pharmacological treatment<sup>9,10</sup>

The key principles of using medication for anxiety disorders are:

- Non-pharmacological management is first line for a reason. Do not rush into using medication and repeatedly reassess whether to continue prescribing.
- Of the medications, SSRIs are regarded as first line and other antidepressants such as SNRIs, e.g. duloxetine and venlafaxine, and the tetracyclic mirtazapine have shown some benefit in anxiety disorders (see TABLES 70.4 and 70.5), but their benefits are not as long-lasting as psychological and behavioural approaches.<sup>11,12</sup>
- Assess efficacy of antidepressants after at least 12 weeks (in contrast to 6–8 weeks when treating major depression) and, if of clear benefit, treat for at least 6 months. Page 844
- The cessation of an SSRI commonly causes withdrawal symptoms. A common trap (for doctor and/or patient) is to interpret these symptoms as a recurrence of the underlying anxiety and as evidence that the drug continues to be required.
- Propranolol is of benefit in social anxiety disorder, particularly with anticipated stressful events (e.g. public speaking, presenting at work events).
- Benzodiazepines have a limited role in anxiety disorders and should not be recommended. If used, they should be reserved for people who have not responded to at least 2 therapies (e.g. psychological therapy and antidepressant) and used only in the short term (stop within 6 weeks), e.g. diazepam 2–5 mg (o), as a single dose. They can also be used for specific phobias (e.g. fear of flying, agoraphobia and MRI machines).
- Consider buspirone, which has negligible potential for tolerance or dependence.

**Table 70.4** Dosage recommendations for SSRIs in anxiety disorders<sup>11</sup>

Drug	Initial dose	Maximum dose
citalopram	10 mg	40 mg
escitalopram	5 mg	20 mg
fluoxetine	10 mg	80 mg
fluvoxamine	50 mg	300 mg (split dose to bd over 150 mg)
paroxetine	10 mg	60 mg
sertraline	25 mg	200 mg

**Table 70.5** Initial medication options for anxiety disorders<sup>11</sup>

Condition	Medication options
Generalised anxiety disorder	SSRI, duloxetine (30–120 mg), venlafaxine controlled release (75–225 mg)
Panic attack	none, psychological intervention
Panic disorder	SSRI, venlafaxine controlled release (75–225 mg)
Obsessive-compulsive disorder	SSRI
Agoraphobia (without panic)	none
Social anxiety disorder (generalised)	SSRI, venlafaxine controlled release (75–225 mg)
Social anxiety disorder (non-generalised)	propranolol 10–40 mg orally, 30–60 minutes before the social event or performance
Specific phobias	not recommended
Post-traumatic stress disorder	SSRI, mirtazapine

Note: Not every SSRI has Australian TGA approval for use for each anxiety disorder; the TGA website can provide up-to-date information on this

changing landscape. There is insufficient evidence to differentiate between efficacy of the individual SSRIs.

## Panic attack<sup>9</sup>

A panic attack is defined<sup>4</sup> as a discrete period of intense fear or discomfort in which four (or more) of the following symptoms develop abruptly and reach a peak within 10 minutes:

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- shortness of breath or smothering sensations
- dizziness, unsteady feelings, lightheadedness or faintness
- palpitations or accelerated heart rate
- trembling or shaking
- sweating
- feeling of choking
- nausea or abdominal distress
- depersonalisation or derealisation
- numbness or tingling sensations (paraesthesia)
- flushes (hot flashes) or chills
- chest pain or discomfort
- fear of dying
- fear of going crazy or of doing something uncontrolled
- the 3 Cs—chest pain, chills, choking

Organic disorders that simulate a panic attack are hyperthyroidism, phaeochromocytoma and hypoglycaemia.

*Note:* A single panic attack is not synonymous with panic disorder, which is characterised by recurrent panic attacks. Some 40% of young people have had at least one spontaneous panic attack. Panic disorder, which affects 2-3% of the population, is when there are recurrent attacks that are followed by at least a month of worrying about future attacks and/or the consequences of them. Panic disorder can occur with or without associated agoraphobia, though >90% of people with agoraphobia develop it as a result of recurrent panic attacks.<sup>11</sup>

## Management

Reassurance, explanation and support (as for generalised anxiety). This is the mainstay of

treatment. A patient who is experiencing a panic attack should be taught breathing techniques to help control hyperventilation (e.g. timing breaths, breathing through nose, slow inspiration, measured medium-sized breaths). Relaxation techniques can also be employed, such as progressive muscle relaxation, and patients can teach themselves these techniques via online resources (see TABLES 70.2 and 70.3). Rebreathing into a paper bag is rarely indicated in a general practice setting<sup>10,11</sup> as the hyperventilation has usually settled by the time the patient presents. The above breathing techniques can be used by the patient anywhere and are more socially acceptable than breathing noisily into a paper bag when an attack is feared.

The danger in a panic attack is the danger *to the self by the self*<sup>9</sup> (e.g. fleeing into danger, non-intentional overdose).

## Cognitive behaviour therapy (CBT)

(See CHAPTER 4 .)

CBT aims to reduce anxiety by teaching patients how to identify, evaluate, control and modify their negative, fearful thoughts and behaviour. If simple psychotherapy and stress management fail, then patients should be referred for CBT, usually to a psychologist (or occasionally psychiatrist), although some GPs have a particular interest in providing CBT.

Patients' fears, especially if irrational, need to be clearly explained by the therapist, examined rationally and challenged, then replaced by positive calming thoughts.

## Pharmacological treatment

Pharmacological treatment is rarely of benefit in the acute attack, as the attacks occur too quickly for their effect to be of use. For ongoing treatment of panic disorder with or without agoraphobia, there is little good quality evidence comparing medication to CBT.<sup>10</sup>

Continual use of benzodiazepines (BDZs, e.g. alprazolam or clonazepam) has previously been utilised in panic and other anxiety disorders but is now no longer recommended.<sup>11</sup> Problems associated with benzodiazepine use include:

- impaired alertness, oversedation
- dependence
- increased risk of accidents
- adverse effects on mood and behaviour
- interaction with alcohol and other drugs
- potential for abuse and overdose
- risks during pregnancy and lactation

- muscle weakness
- sexual dysfunction
- diminished motivation
- lowered sense of competency
- lowered self-esteem

Some principles of using BDZs in anxiety disorders<sup>11</sup> are:

- always check for a history of problem alcohol or drug use
- be wary of prescribing to unfamiliar patients, especially if asking for a particular drug by name (may indicate drug-seeking behaviour)
- carefully discuss the potential for addiction with the patient
- avoid using short-acting drugs as they are the most highly addictive
- prescribe only small quantities of medication at a time
- use only as short-term treatment
- ensure regular review of the patient and continuity of care

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If already being used, BDZs should be tapered very slowly (this may take 6–12 months or longer). A benzodiazepine withdrawal syndrome, which can include rebound anxiety, depression, confusion, insomnia and seizures, may occur (see [CHAPTER 69](#) ). However, the doctor's fear of being responsible for a withdrawal syndrome has also been used as leverage by those seeking drugs. If in doubt, seek specialist drug and alcohol advice.

## Phobic disorders

In phobic states, the anxiety is related to specific situations or objects out of proportion to the apparent stimulus. Phobic disorders include agoraphobia, social anxiety disorder (otherwise known as social phobia) and specific phobias. Patients avoid these objects or situations, become anxious when they anticipate having to meet them and/or endure them with intense distress.

Common phobias are spiders, people and social situations, flying, open spaces, confined spaces, heights, cancer, thunderstorms, death and heart disease. The problem is seldom encountered in practice and there is usually no call for drug therapy.

### Agoraphobia

Avoidance includes staying away from many situations where there are issues of distance from home, crowding or confinement. Typical examples are travel on public transport, crowded shops

and confined places. Patients fear they may lose control, faint and suffer embarrassment.

## Social anxiety disorder

Social anxiety disorder (social phobia) is experienced in anxiety-provoking social situations in which the person feels subject to critical public scrutiny (e.g. canteens, restaurants, staff meetings, speaking engagements). It can either be generalised (fear of numerous social situations, including both performance and interactional situations) or non-generalised (fear of one or just a few situations or performance type). The treatments for the two subtypes are quite different (see TABLE 70.5 ). The sufferer may be a shy, self-conscious, premorbid personality. Social phobias, including performance anxiety and symptoms, are often related to sympathetic overactivity.

### Management

The basis of treatment for all phobias is psychotherapy that involves behaviour therapy (e.g. graduated exposure therapy) and cognitive therapy.

## Illness anxiety disorder

Also referred to as hypochondriasis or health disorder, it implies worrying excessively about being or becoming seriously ill despite no actual physical symptoms. Treatment is counselling through stress management, including relaxation techniques.

## Obsessive-compulsive disorder (OCD)

Anxiety is associated with obsessive thoughts and compulsive rituals.

The obsessions are recurrent and persistent intrusive ideas, thoughts, impulses or images that are usually resisted by the patient. Compulsions are repetitive, purposeful and intentional behaviours conducted in response to an obsession to prevent a bad outcome for the person (e.g. excessive washing of the genitals).

Mild obsessional or compulsive behaviour can be regarded as normal in response to stress.

### Management

Optimal management is a combination of psychotherapeutic—particularly CBT—and pharmacological treatment, namely:

- cognitive behaviour therapy for obsessions
- exposure and response prevention for compulsions
- an SSRI is first line if pharmacological required

## Body dysmorphic disorder

The person with this disorder has an exaggerated preoccupation with an imagined defect in appearance (see [CHAPTER 69](#) ).

Patients may be helped by counselling and psychotherapy.

## Post-traumatic stress disorder (PTSD)

PTSD is defined somewhat differently, in terms of time lapses from the traumatic event. It refers to a similar constellation of symptoms that persist for more than 1 month after exposure:

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- acute PTSD: duration of symptoms <3 months
- chronic PTSD: duration of symptoms  $\geq 3$  months
- delayed onset PTSD: onset of symptoms at least 6 months after the stressor

Typical distressing recurrent symptoms:

- intrusive features regarding experiences—recollections, nightmares, flashbacks
- avoidance of events that symbolise or resemble the trauma
- persistent negative alterations in cognitions and mood
- hyperarousal phenomena: exaggerated startle response, irritability, anger, difficulty with sleeping and concentrating, hypervigilance, reckless or self-destructive behaviour

## Treatment

This is difficult and involves counselling, the basis of which is facilitating abreaction of the experience by individual or group therapy. The aim is to allow the patient to face up openly to his or her memories. Persistent symptoms are an indication for referral for focused psychological intervention therapy.

### Pharmacological treatment

SSRIs have limited evidence of some benefit, but response is slower than for their use in depression (trial for 8–12 weeks) and, if effective, they should be used for at least 12 months.<sup>11,12</sup>

## Hyperventilation

Hyperventilation syndrome can be a manifestation of anxiety. The main symptoms are: