Examination of gait and posture[1]

Disorders of gait and posture go hand in hand because of a common physiological process.

The source of the abnormality is indicated.

1. Ask the patient to stand.

   Note any difficulty in reaching a standing position. Difficulty = proximal muscle weakness.

2. Ask the patient to stand with feet together and eyes open at first, then eyes closed for up to 60 seconds (Romberg test).

   If positive (sways or falls) = loss of proprioception (e.g. peripheral neuropathy).

3. Ask the patient to walk normally for a few metres (ensure sufficient testing length).

Gait initiation:	hesitancy = basal ganglia or frontal cortex
Stride length:	very short = basal ganglia or frontal cortex irregular = cerebellar
Narrow or broad base:	narrow = UMN, muscle weakness, basal ganglia broad = cerebellum, proprioception, vestibular
Stiff or ‘sloppy’:	stiff = UMN, basal ganglia sloppy = LMN, muscle weakness
Heel strike:	loss of normal strike = UMN, LMN, myopathy
High stepping:	positive = LMN (distal), proprioception, muscle weakness
Arm swing:	decreased = basal ganglia, UMN (frontal lobes)
Pelvis control:	Trendelenburg gait = proximal muscle weakness

4. Ask the patient to walk using provocation tests.

‘Tightrope’ walk:	tests proprioception
Stand on tiptoes and back on the heels:	tests distal muscle weakness

Note: Peripheral neuropathy is a common cause of LMN lesion.

Neurological disorders of gait

Cerebellar (ataxic) gait

The patient has difficulty standing, has a broad-based stance and an unsteady gait, with swaying from side to side. It has been described as the ‘dirty diaper’ gait. The person may stagger or bump into walls and may be accused of being drunk. When asked to walk in a straight line they tend to veer towards the side of the lesion. Ask them to walk ‘heel to toe’ in a straight line and perform a heel–knee–shin test to demonstrate cerebellar ataxia. Significant causes are multiple sclerosis, alcoholic cerebellar degeneration and space-occupying lesions. Cannot climb stairs.

Basal ganglia gait (Parkinson type)

Identifying this disorder in its early stages can be difficult as the first sign may be a limp with one leg being described as weak or stiff or slow.[2] However, the typical gait is a shuffling of the feet with small steps in a forward flexed posture, rather like a person walking with a belt around the ankles. This leads to a hurrying (festinant) gait as though there is an impending feeling of falling forward (propulsion). Another feature is freezing. There is hesitation in starting.

Spastic gait

Spastic gait may be regarded as the typical stiff bilateral or paraplegic gait, or as a hemiplegic gait. With the former the gait affects both legs—they are stiff or weak, leading to slow, jerking walking, dragging of the feet and scraping of the toes. This scuffing can be heard as the front of the shoe drags along the ground. Every step can be a struggle and the person may appear as though walking through glue on the floor.

A scissor-type gait will develop with bilateral hip adduction. Spasticity is caused by a UMN lesion, including multiple sclerosis and spinal cord compression. The typical UMN posture is of a flexed upper limb and an extended lower limb.

With hemiplegia, the person drags the affected leg stiffly with the hips adducted, the knee extended and the foot plantar flexed, leading to scraping of the toes. Mounting stairs can be very difficult, especially if clonus is induced with dorsiflexion of the foot.

Foot drop gait

The person cannot dorsiflex the foot, leading to a high-stepping gait with extra flexion of the hip and knee to lift the foot off the ground. The foot then slaps down on the ground.

Vestibular gait

If unilateral, the person tends to veer off to the side of the lesion.

Apraxia

With apraxia of gait (due to a prefrontal lobe lesion) there is a failure of control of the legs. The

person may stand up and try to walk but looks with bewilderment at the legs (the ‘glued to the floor’ effect) and moves them in an inappropriate manner with a broad-based, small-stepping unsteady gait. Turning is difficult. It is common in the elderly, usually secondary to cerebrovascular disease, and can be described as lower body Parkinsonism. Apraxia is caused by bilateral cortical involvement, such as normal pressure hydrocephalus, multi-infarct states and tumours of the corpus callosum.[2]

Neurogenic claudication

The person with intermittent claudication of the cauda equina, due to spinal canal stenosis, develops pain in the leg after walking a certain distance. However, weakness and numbness are usually more prominent than pain.

Drop attacks

In a drop attack the person suddenly falls to the ground, without other symptoms, and gets up almost immediately. There is no loss of consciousness. Drop attacks can be caused by disorders such as epilepsy, Parkinson disease and vertebrobasilar insufficiency. However, in most cases, particularly middle-aged and elderly women, there is no obvious cause. (See CHAPTER 43 .)

Waddling (myopathic) gait

A waddling gait is usually caused by muscular dysfunction affecting the pelvic girdle muscles and trunk. There is a wide-based gait with a marked ‘rocking and rolling’ body swing from side to side and related compensatory movements of the pelvis; that is, a bilateral Trendelenburg gait. Cannot climb stairs.

Proximal muscle weakness

The person may complain when getting out of a low chair or going up or downstairs. The weakness can be demonstrated by asking them to squat down and, after a second, rise from the squatting position.[2] Waddling of gait reflects extreme cases. Causes include myopathies, motor neurone disease and Guillain–Barré syndrome.

Distal muscle weakness

This causes a high-stepping gait as the foot is floppy and tends to flap, with walking similar to foot drop gait. Causes include peripheral neuropathy, myotonic dystrophy and peroneal muscular atrophy.

Limp

Limp is a symptom commonly associated with painful disorders of the lower limb, especially of

the hip and knee joints. A limp implies an asymmetrical gait pattern caused by one of four general factors:

1. unequal leg length
2. antalgic (painful) gait (e.g. hip disorder)
3. restricted joint movement (e.g. ankylosed knee)
4. neuromuscular weakness (e.g. poliomyelitis)

Limp has an inseparable relationship with painful hip and buttock conditions, especially those of the hip. Painful hip and pelvic conditions that cause limp are presented in CHAPTER 54 .

Limp in adults

In adults the cause of limp is usually more obvious than in children and is commonly due to degenerative osteoarthritis of the hip or knee, to spinal disorders, especially sciatica caused by a disc prolapse, or to overuse disorders of the knee, ankle or foot. Other causes to consider are trauma to the back and legs, vestibular ataxia, Parkinson disease and intermittent claudication.

Limp in children

The child who limps presents an interesting diagnostic dilemma. The limp must be considered to be due to a definite organic cause, although conversion reactions can be a factor.[3] It is appropriate to focus initially on the hip. The diagnostic strategy is presented in TABLE 58.1 .

Table 58.1 Limp in children: diagnostic strategy model (modified)

Probability diagnosis	
Post trauma/intense exercise causing strain syndromes	
Ill-fitting shoes	
Hip disorders, esp. transient synovitis	
Heel disorders (12–14 years)	
Serious disorders not to be missed	
Toddlers	DDH
	Child abuse
	Septic arthritis
	Foreign body (e.g. needle in foot)
4–8 years	Perthes disorder

	Transient synovitis
Adolescents	SCFE Avulsion injuries (e.g. ischial tuberosity) Osteochondritis dissecans of knee Duchenne muscular dystrophy
All groups	Septic infections: • septic arthritis • osteomyelitis • tuberculosis Tumour (e.g. osteosarcoma) Juvenile chronic arthritis Spinal disorders: • discitis • fracture

Pitfalls (often missed)

Foreign body (e.g. glass in foot)

Osteochondritis (aseptic necrosis):

- femoral head—Perthes disease
- knee—Osgood–Schlatter disease
- calcaneum—Sever disease
- navicular—Köhler disease

Myalgia = growing pains

Overuse syndrome (esp. adolescent):

- patellar tendinopathy (jumper's knee)

Stress fractures (e.g. tibia, femoral neck, navicular)

Limp can be considered as acute, subacute or chronic. An acute limp may be due to injury, infection (osteomyelitis, septic arthritis), spinal injuries, a fracture or an irritable hip (transient synovitis). Subacute causes include juvenile rheumatoid arthritis and tumour or leukaemia. Chronic causes include cerebral palsy, DDH, Perthes disease and chronic SCFE.

Key facts and checkpoints

- Trauma, sepsis and DDH are perhaps the most common reasons for an infant to limp and refuse to walk. However, a painless waddling gait suggests DDH or Perthes disease, which usually begins with a painless limp.

- Multiple fractures and epiphyseal separations in toddlers are highly suggestive of physical child abuse; a skeletal survey should be ordered if this is suspected.
- Perthes disease can present from ages 4–12 years but is usually found from 4–8 years with a peak age of 5–7 years.
- Infections of and around the hip joints are most common in infancy. Classically, the hip is held immobile in about 30° of flexion with slight abduction and external rotation. The commonest organism is *Staphylococcus aureus*, followed by *Haemophilus influenzae*.
- Tuberculosis may also occur in children (usually under 10 years) with a presentation similar to Perthes disorder.
- SCFE typically presents in the obese adolescent (10–15 years) with knee pain and a slight limp.
- Growing pains are a controversial issue but do appear to exist as an aching myalgia usually manifest in the leg muscles (anterior thigh, calf, posterior knee). The pain is bilateral, non-articular and usually unrelated to activity.

A diagnostic approach to limp

History

The age of the patient gives a diagnostic pointer. A careful history, especially of trauma, may lead to the diagnosis. A history of injury is usually but not always available. The relationship of the limp to exercise and footwear is significant. The location of any associated pain is relevant: low back pathology can refer to the buttocks and hip pathology can cause knee pain.

Examination

The hip and knee joints should be carefully examined if the source of limp has no specific localisation. Get the child to walk and run on the toes and heels (if appropriate). Note the gait and check whether it is antalgic (painful), hemiplegic (the arm is held out in a balancing action) or Trendelenburg (classic for DDH). Look for evidence of muscular dystrophy. Never forget to examine the soles of the feet and between the toes.

Investigations

The following should be considered:

- FBE and ESR

- blood culture
- needle aspiration of joint
- radiological: plain X-ray, ultrasound, bone scan, CT or MRI scan

Management

Management is based on the cause. Surgical drainage supplemented with antibiotics is essential for septic arthritis. If the child initially had a limp and now cannot walk, consider hospital admission.

Specific conditions

Osteomyelitis

Acute haematogenous osteomyelitis is mainly a disease of childhood, occurring in the proximal ends of long bones, particularly the femur and tibia. It should be suspected in a child with an acute febrile illness, unwillingness to move the limb and ‘fingertip’ tenderness near one of the large points (the metaphysis). Main organisms—*S. aureus, S. pneumonia, Kingella kingae, Propionibacterium acnes.* Sources of infection—boils, abscesses, septic toes, surgical procedures. Consider impaired immunity and diabetes.

Blood should be collected for FBE, ESR and culture (positive in 60%). A plain X-ray and nuclear scan are valuable but may not provide immediate confirmation of the diagnosis (may be normal for the first 10 days). The child should be admitted to hospital, an IV line should be inserted and IV antibiotics commenced to cover the organisms involved—empirical therapy:

> flucloxacillin:[4] 50 mg/kg up to 2 g IV 6 hourly; adjust according to the site of infection, patient’s age and particularly to culture and sensitivity results[4]

Septic arthritis

Septic arthritis should be suspected in a child with pyrexia and an acute arthritis with limited motion. Manage as for osteomyelitis as causative organisms are similar.

Bone tumour

Chronic limp is a common presentation of malignant bone tumours. Radiological investigation is mandatory.

Irritable hip syndrome (transient synovitis)

Typical age is 3–8 years and the child presents with an acute limp with restricted hip motion.

Plain X-ray is normal. Orthopaedic assessment is recommended.

Perthes disease, SCFE and DDH

Refer to CHAPTER 54 .

Paget disease

Paget disease of bone (osteitis deformans) is a chronic focal disorder of the adult skeleton in which new soft bone replaces localised areas of normal bone. The cause is unknown but a viral aetiology is suspected. There is a great increase in bone turnover with osteoclastic resorption followed by increased but disorganised osteoblastic activity. The disorder is quite common in Caucasians:

- 1 in 200 of the population at 40 years
- 1 in 10 of population at 90 years

Paget disease is usually asymptomatic but some patients may present with deep aching pain in the lower back and lower limbs. They may also present with a disturbance of gait due to unequal leg length, osteoarthritis of associated joints such as the knee or hip, or a change in the distribution of mechanical forces in the lower extremities (see FIG. 58.1).

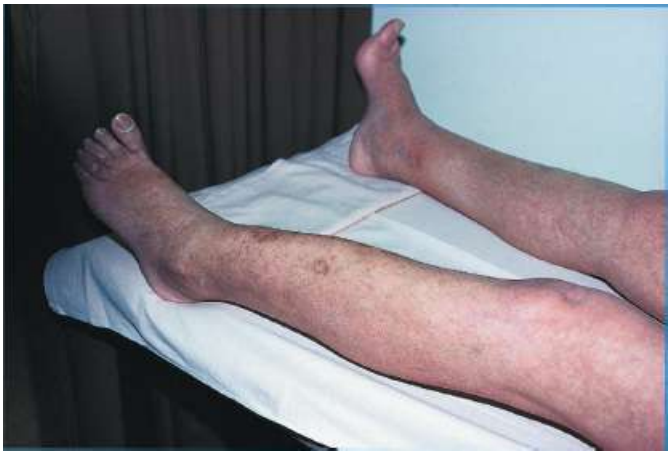

FIGURE 58.1 Paget disease of the left leg showing deformity of the tibia with a ‘sabre’ tibia due to its enlargement of length and bulk

Clinical features[5]

- Male:female ratio = 2:1.
- 95% asymptomatic (discovered by X-ray or raised serum alkaline phosphatase [ALP] level).

- Symptoms may include joint pain and stiffness (e.g. hips, knees), bone pain (usually spine), deformity, headache and deafness.
- Bone pain is typically deep and aching; it occurs at rest, particularly at night.
- Signs may include deformity, enlarged skull (‘hats don’t fit any more’), bowing of tibia, waddling gait, hyperdynamic circulation (see FIG. 58.2).
- Bones most commonly affected, in decreasing order, are the pelvis, femur, skull, tibia, vertebrae, clavicle and humerus.
- Sensory disability—taste, smell, vision, hearing, loss or reduction of feeling in areas of the face if cranial nerves involved.

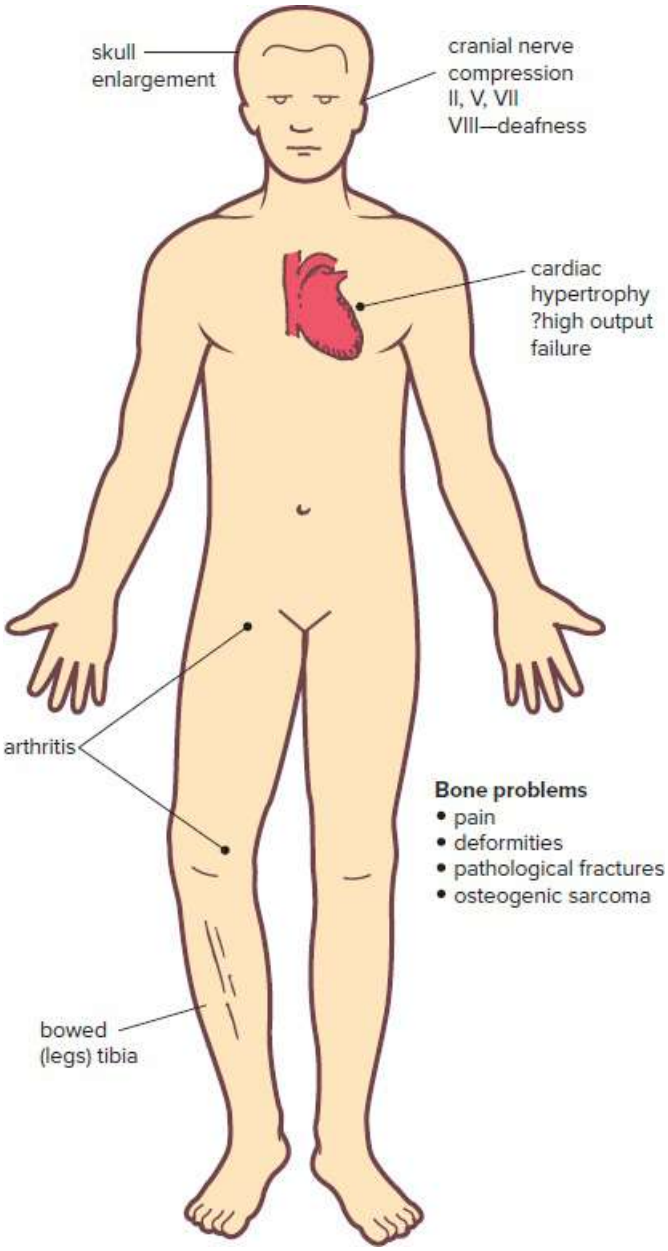

FIGURE 58.2 Paget disease: possible clinical features

Diagnosis

- Raised serum ALP level (often very high >1000 U/L). A result >125 U/L suggests active disease.[6]

  Note: Calcium and phosphate normal.

- Plain X-ray: dense expanded bone—best seen in skull and pelvis.

Note: Can mimic prostatic secondaries, so every male Pagetic patient should have a DRE and serum PSA test.

- Bone isotopic scans: useful in locating specific areas.
- Watch for the uncommon complication of osteogenic sarcoma.

Note: Screen siblings and children every 5 years after the age of 40.[6]

Treatment[5,6]

The major goals are relief of pain, normal biochemical indices of bone turnover and prevention of long-term complications (e.g. deafness, deformities). Treatment is required for symptomatic features such as bone pain and neurological complications, and for selected asymptomatic patients such as those with active disease and <50 years.

Localised disease and most patients with asymptomatic disease require no treatment; hence, population screening is not recommended.

Bisphosphonates, particularly IV zoledronate, are first-line therapy:[6]

zoledronic acid 5 mg IV over at least 15 minutes, once yearly (certain biomedical parameters must be met, e.g. eGFR >35)

If zoledronic acid is inappropriate, use:

pamidronate disodium 30–60 mg IV infused over 2–4 hours

or

risedronate 30 mg (o) daily for 2 months (on empty stomach)

Repeated doses may rarely be required in severe cases as judged by symptoms and disease activity (e.g. monitoring with serum ALP usually 6 months post-therapy and then 2 yearly; X-rays also help monitor response).

Leg swelling

Diagnostic features and pitfalls

- Not all swollen legs require investigation and treatment.
- The significance of leg swelling varies according to the age group, whether it is bilateral or unilateral, and whether the onset is sudden or gradual (see TABLE 58.2).
- If the onset of oedema is acute (often <72 hours) suspect DVT.[7]

- DVT must be considered in all unilateral cases and ultrasound examination performed if appropriate.
- If a DVT is present, consider occult malignancy (e.g. pancreatic cancer).
- Consider pelvic cancer causing lymphatic obstruction in a woman >40 years presenting with painless unilateral leg oedema.
- A drug history is essential as several drugs can cause oedema.
- Pitting oedema is a feature of venous thrombosis or insufficiency, not lymphatic obstruction.

Table 58.2 Leg and ankle swelling: diagnostic strategy model

Probability diagnosis

Physiological:
- dependency/gravitational
- prolonged sitting, standing, walking
- hot weather
- pregnancy
- mechanical (e.g. constricting clothing)

Chronic venous insufficiency (varicose veins)
Congestive cardiac failure
Drugs (e.g. calcium antagonists, NSAIDs, steroids, glitazones, beta blockers)
Local trauma
Obesity

Serious disorders not to be missed

Vascular:
- deep venous thrombosis (DVT)
- inferior vena cava thrombosis
- thrombophlebitis

Infection:
- cellulitis
- tropical infections (e.g. filariasis, hookworm)

Cancer:
- obstruction from pelvic cancer
- localised malignancy

Other:
- kidney disease (e.g. nephrotic syndrome)

- liver disease (e.g. cirrhosis)
- skin allergy (e.g. angioneurotic oedema)

Pitfalls (often missed)

Idiopathic (periodic or cyclic) oedema
Protein-losing enteropathy (e.g. Crohn)
Lipoedema (fat and fluids) of legs
Factitious oedema
Rarities:

- malnutrition
- lymphoedema: primary or secondary

Seven masquerades checklist

Diabetes
Drugs (multiple)
Thyroid/endocrine (hypothyroidism, Cushing syndrome)
Anaemia

Investigations

Select from these first-line tests:

- urinalysis (?albumin)
- FBE and ESR
- serum UEC, LFTs, glucose
- TSH level
- ultrasound (DVT screen)
- other radiographs (e.g. CT scan, venogram)

Calf swelling of sudden onset

Causes to consider:

- acute arterial occlusion
- ruptured Baker cyst
- ruptured medial head of gastrocnemius

- DVT (usually gradual)
- cellulitis/erysipelas
- compartment syndrome

Pain accompanies most of these conditions but the absence of pain does not exclude DVT or thrombophlebitis. Refer to CHAPTER 55 .

Lipoedema

Lipoedema (also spelled lipedema) is the development of painful bilateral leg swelling that does not involve the feet (whereas in lymphoedema the swelling starts with the most distal part of the foot). Lipoedema is abnormal fatty tissue.

Clinical features

- Exclusive to obese women; hereditary
- Typically affects the thighs, buttocks and lower legs (sometimes the arms)
- Spares the feet
- Bilateral and symmetrical distribution of fat
- The legs are often painful and bruise easily
- The Stemmer sign (the ability to pick up a fold of skin at the base of the large toe) is usually negative[8]

It is difficult to treat. The focus is on changes to diet, exercises, massage and bandage compression.

Patient education resources

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

- Deep venous thrombosis
- Paget disease of bone
- Parkinson disease

Resource

A physician's guide to the management of Paget disease of bone. Brooklyn NY: The Paget

Foundation. Available from: https://www.paget.org/index-php/healthcare-professionals/pagets-disease-of-bone/126-a-physicians-guide-to-the-management-of-pagets-disease-of-bone.html.

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59 Palpitations

The most important requirement of the art of healing is that no mistakes or neglect occur. There should be no doubt or confusion as to the application of the meaning of complexion and pulse. These are the maxims of the art of healing.

HUANG TI (THE YELLOW EMPEROR) (2697–2597 BCE)

Palpitations are an unpleasant awareness of the beating of the heart. By definition it does not always imply 'racing' of the heart but any sensation in the chest, such as 'pounding', 'flopping', 'skipping', 'jumping', 'thumping' or 'fluttering' of the heart. The problem requires careful attention and reassurance (if appropriate) because heartbeat is regarded as synonymous with life. To the practitioner it may simply represent anxiety or it could be a prelude to a cardiac arrest. In many circumstances prompt referral to a cardiologist is imperative.

Key facts and checkpoints

- The symptom of palpitations is suggestive of cardiac arrhythmia but may have a non-cardiac cause.
- Palpitations not related to emotion, fever or exercise suggest an arrhythmia.
- Symptomatic premature ventricular beats/complexes (ventricular ectopics) are common, and atrial fibrillation is the most common sustained cardiac arrhythmia (1–2% of population), while cardiologists claim that the commonest is the symptomatic ventricular ectopic beat.[1]
- The commonest cause of an apparent pause on the ECG is a blocked premature atrial beat/complex (atrial ectopic).
- A 12-lead electrocardiographic diagnosis is mandatory. If the cause is not documented, an ambulatory electrographic monitor (e.g. Holter) may be used.
- Consider myocardial ischaemia as a cause of the arrhythmia.
- Consider drugs as a cause, including prescribed drugs and non-prescribed drugs such as alcohol, caffeine and cigarettes.

- Common triggers of paroxysmal supraventricular tachycardia (PSVT) include anxiety and cigarette smoking.
- The commonest mechanism of any arrhythmia is re-entry.
- Ask patients to tap out the rate and rhythm of their abnormal beat.

A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 59.1 , which includes significant causes of palpitations.

Table 59.1 Palpitations: diagnostic strategy model

Probability diagnosis
Anxiety
Premature beats (ectopics)
Sinus tachycardia
Drugs (e.g. stimulants)
Supraventricular tachycardia
Serious disorders not to be missed
Myocardial infarction/angina
Arrhythmias:
• atrial fibrillation/flutter
• ventricular tachycardia
• bradycardia
• sick sinus syndrome
• torsade de pointes
Long QT syndrome
Wolff–Parkinson–White (WPW) syndrome
Electrolyte disturbances:
• hypokalaemia
• hypomagnesaemia
• hypoglycaemia (type 1 diabetes)
Pitfalls (often missed)
Fever/infection
Pregnancy

Menopause (sudden vasodilatation)
Drugs (e.g. caffeine, cocaine)
Mitral valve disease
Aortic incompetence
Hypoxia/hypercapnia
Rarities:

- tick bites (T1–5)
- phaeochromocytoma

Seven masquerades checklist

Depression
Diabetes (indirect)
Drugs
Anaemia
Hyperthyroidism
Spinal dysfunction
UTI (possible)

Is the patient trying to tell me something?

Quite likely. Consider cardiac neurosis, anxiety.

Probability diagnosis

If the palpitations are not caused by anxiety or fever, the common causes are sinus tachycardia and premature complexes/ectopics (atrial or ventricular). Sinus tachycardia, which by definition is a rate of 100–160/min (bpm), may be precipitated by emotion, stress, fever or exercise. PSVT and atrial fibrillation are also quite common arrhythmias.

Sinus tachycardia can be differentiated clinically from PSVT in that it starts and stops more gradually than PSVT (abrupt) and has a lower rate of 100–150 compared with 160–220.

Important causes of tachyarrhythmias are:

- ischaemic heart disease, especially acute coronary syndromes
- hypertension
- heart failure
- mitral disease
- thyrotoxicosis

- atrial septal deficit

Serious disorders not to be missed

It is vital not to overlook acute coronary syndromes as a cause of the arrhythmia manifesting as palpitations. About 25% of infarcts are either silent or unrecognised. Sinister life-threatening arrhythmias are:

- ventricular tachycardia
- atypical ventricular tachycardia (torsade de pointes)
- sick sinus syndrome (SSS)
- complete heart block

It is also important not to miss:

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- hypokalaemia
- hypomagnesaemia

Pitfalls

There are many pitfalls in the diagnosis and management of arrhythmias, especially in the elderly, where symptoms of infection may be masked. Palpitations associated with the menopause can be overlooked. Valvular lesions, usually associated with rheumatic heart disease, such as mitral stenosis, and aortic incompetence may cause palpitations. The rare tumour, phaeochromocytoma, presents with palpitations and the interesting characteristic of postural tachycardia (a change of more than 20 beats/ min). The toxin from tick bites in dermatomes T1–5 can cause palpitations.

General pitfalls

- Misdiagnosing PSVT as an anxiety state
- Overlooking a cardiac arrhythmia as a cause of syncope or dizziness
- Overlooking atrial fibrillation (AF) in the presence of a slow heartbeat
- Overlooking mitral valve prolapse in someone, especially a middle-aged woman presenting with unusual chest pains and palpitations (auscultate in standing position to accentuate click(s) ± murmurs)

Red flags for palpitations

- Lightheadedness
- Chest pain
- New onset of irregular heart rhythm
- Heart rate >120 or <45/minute at rest
- Significant underlying heart disease
- Family history of sudden death

Seven masquerades checklist

Surprisingly, all the masquerades have to be considered, either as direct or indirect causes: depression, especially with anxiety and in the postpartum period; diabetes, perhaps as an arrhythmia associated with a silent myocardial infarction or with hypoglycaemia; drugs as a very common cause (see TABLE 59.2); anaemia, causing a haemodynamic effect; hyperthyroidism; spinal dysfunction of the upper thoracic vertebrae T1–5; and urinary tract infection, especially in the elderly.

Table 59.2 Some drugs that cause palpitations

Alcohol
Alendronate
Aminophylline/theophylline
Amphetamines
Antipsychotics (e.g. CPZ, haloperidol, olanzapine)
Antiarrhythmic drugs (esp. Class 1a and 1c)
Antidepressants:
- tricyclics
- MAO inhibitors

Appetite suppressants
Atropine, hyoscine, hyoscyamine
Caffeine, including energy drinks
Cocaine
Digitalis
Diuretics → K ↓, Mg ↓
Glyceryl trinitrate
Nicotine in cigarettes

Sympathomimetics:

- in decongestants (e.g. pseudoephedrine, ephedrine)
- β-agonists (e.g. salbutamol, terbutaline)

Thyroxine

PSVT has been described as resulting from injury or dysfunction of the upper thoracic (especially T4 and T5) and cervical spine in the absence of organic heart disease.[2] The author has personally encountered several cases of PSVT alleviated by normalising function of the spine.

Psychogenic considerations

Emotional factors can precipitate a tachycardia, which in turn can exaggerate the problem in an anxious person. Some people have a cardiac neurosis, often related to identification with a relative or friend. A family history of cardiac disease can engender this particular anxiety. Evidence of anxiety and depression should be sought in those presenting with palpitations without clinical evidence of cardiovascular disease.

The clinical approach

Careful attention to basic detail in the history and examination can point the way clearly to the clinical diagnosis.

History

Ask the patient to describe the onset and offset of the palpitations, the duration of each episode and any associated features. Then ask them to tap out on the desk the rhythm and rate of the heartbeat experienced during the ‘attack’. If they are unable to do this, tap out the cadence of the various arrhythmias to find a matching beat.

An irregular tapping ‘all over the place’ suggests atrial fibrillation, while an isolated thump or jump followed by a definite pause on a background of a regular pattern indicates premature beats (ectopics/ extrasystoles) usually of ventricular origin. The thump is not the abnormal beat but the huge stroke volume of the beat following the compensatory pause.

Key questions

- Do the palpitations start suddenly? How long do they last?
- What do you think brings them on?
- Are they related to stress or worry or excitement?
- What symptoms do you notice during an attack?

- Do you have pain in the chest or breathlessness during the attack?
- Do you feel dizzy or faint during the attack?
- What medications do you take?
- How much coffee, tea, Coke do you drink?
- Have you been using nasal decongestants?
- Did you eat Chinese takeaway food before the attack?
- Do you smoke cigarettes, and how many?
- Do you take any of the social drugs, such as cocaine or marijuana?
- Have you ever had rheumatic fever?
- Have you lost weight recently or do you sweat a lot?

Chest pain may indicate myocardial ischaemia or aortic stenosis; breathlessness indicates anxiety with hyperventilation, mitral stenosis or cardiac failure; dizziness or syncope suggests severe arrhythmias such as SSS and complete heart block, aortic stenosis and associated cerebrovascular disease.

Examination

The ideal time to examine the patient is while the palpitations are being experienced. Often this is not possible and the physical examination is normal. Measurement of the heart rate may provide a clue to the problem.

As a working guide, a rate estimated to be about 150 beats per minute (bpm) suggests PSVT, atrial flutter/fibrillation or ventricular tachycardia (see FIG. 59.1). A rate less than 150 bpm is more likely to be sinus tachycardia, which may be associated with exercise, fever, drugs or thyrotoxicosis.[3]

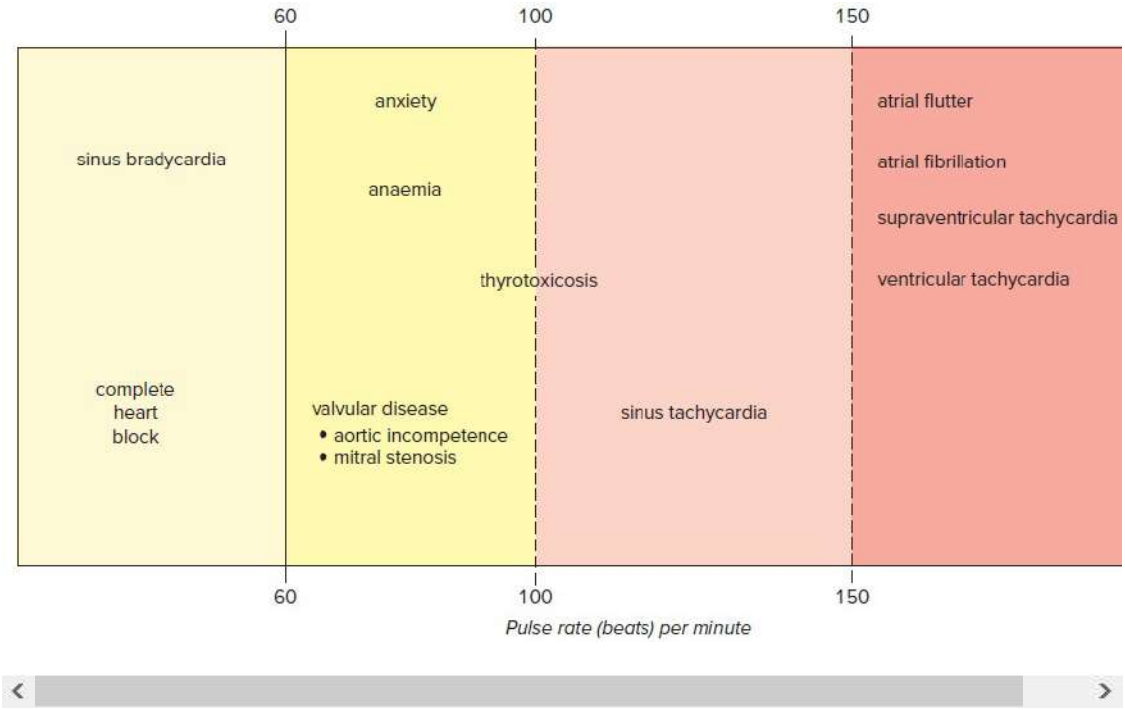


FIGURE 59.1 Heart rate guide to causes of various arrhythmias

Assess whether the pulse is regular or irregular. If irregular, the possible causes are ectopic beats, atrial fibrillation and atrial flutter with varying degrees of block.

The nature of the pulse, especially the pulse pressure and rhythm, should be carefully evaluated (see FIG. 59.2). Look for evidence of fever and infection and features of an anxiety state or depressive illness.

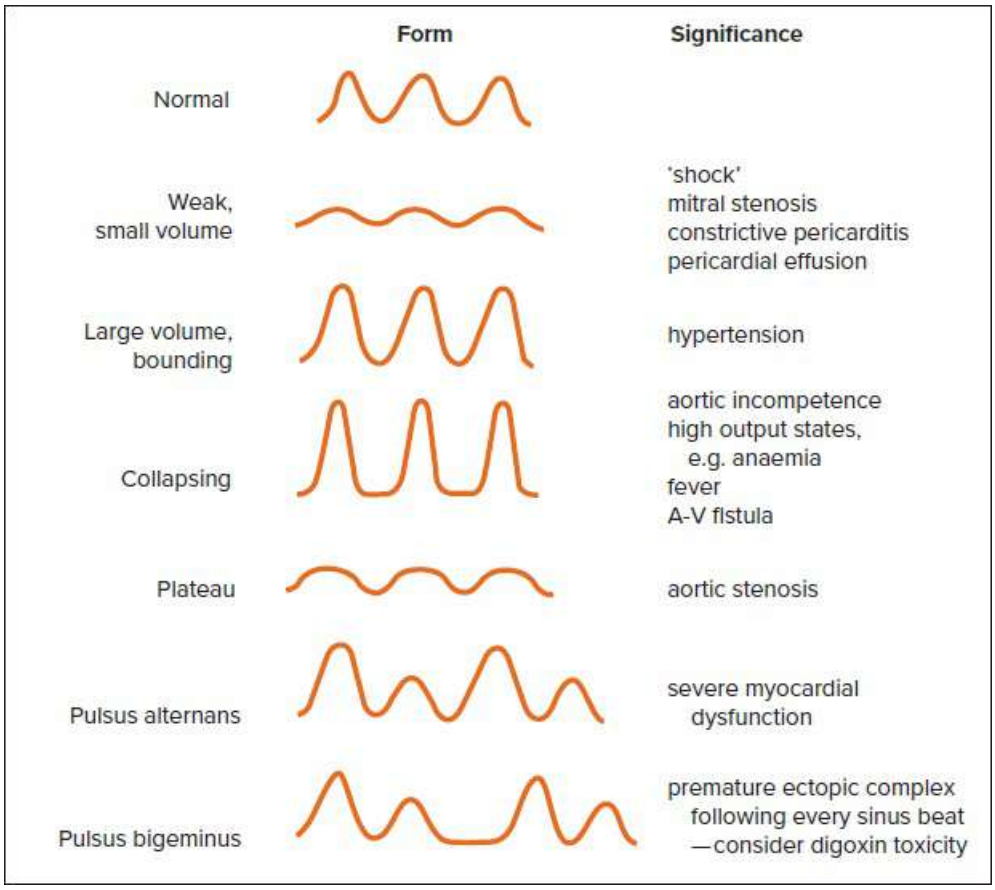


FIGURE 59.2 Various pulse forms

Have the patient hyperventilate for 2–3 minutes to determine whether the arrhythmia is induced. Seek evidence of underlying disease such as anaemia, thyroid disorder, alcohol abuse or cardiac disease including the JVP and pulmonary congestion. Also look for evidence of a mitral valve prolapse (mid-systolic click; late systolic murmur). Possible signs in the person presenting with palpitations are shown in FIGURE 59.3 .[4]

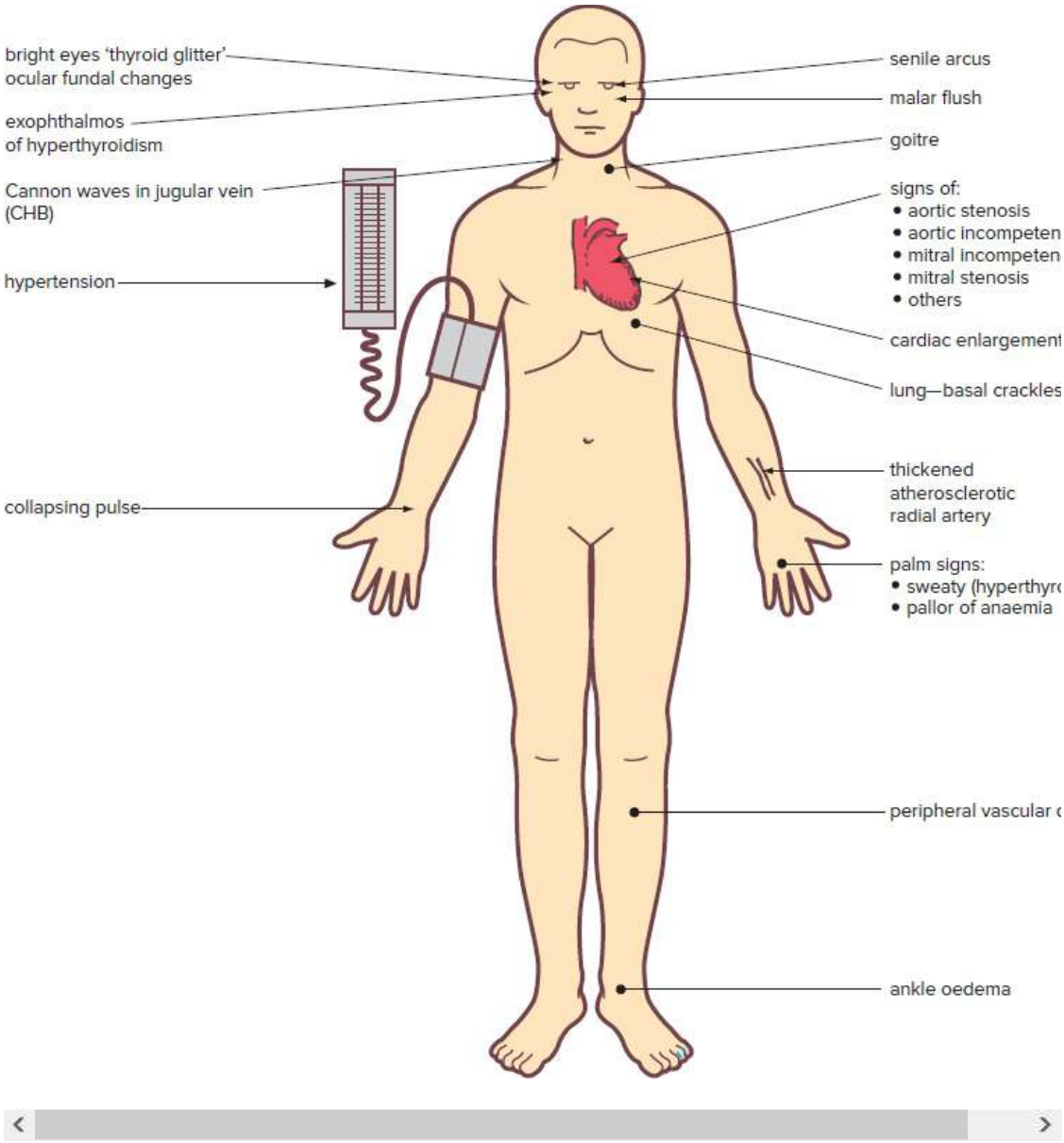


FIGURE 59.3 Signs to consider in a person with palpitations

Investigations

The number and complexity of investigations should be selected according to the problem and test availability. A checklist would include:

- blood tests (for underlying disease):

  haemoglobin and film

  thyroid function tests

serum electrolytes and magnesium

serum digoxin (?digitalis toxicity)

virus antibodies (?myocarditis)

- chest X-ray
- cardiac (ischaemia and function):

ECG (12 lead)

ambulatory 24-hour ECG (Holter) monitoring

echocardiography (to look for valvular heart disease and assess left ventricular function)

electrophysiology studies

exercise stress test (?underlying CAD)

event monitor (can record up to 2 weeks)

implantable monitor (may last 1 year)

Palpitations in children

Children may complain of palpitations which may be associated with exercise, fever or anxiety. Various arrhythmias can occur with three requiring special consideration—PSVT, heart block and ventricular arrhythmias.[5]

PSVT is characterised by 200–300 bpm, the fastest rates occurring in infants. The cause Page 726 is often not found but some children have ECG abnormalities compatible with the Wolff–Parkinson–White (WPW) syndrome. The recommended first-line treatment of PSVT is vagal stimulation via the application of ice packs to the upper face (forehead, eyes and nose) of the affected infant. Intravenous adenosine will usually terminate the episode.

A particular concern is those children who have the familial long QT syndrome. They are prone to develop ventricular tachyarrhythmias, which may lead to sudden death. Consider it in children developing syncope on exertion.

Palpitations in the elderly

The older the person, the more likely is the onset of palpitations due to cardiac disease such as myocardial infarction/ischaemia, hypertension, arrhythmias and drugs, especially digoxin. Occasional atrial and ventricular arrhythmias, especially premature complexes (ectopics), occur in 40% of older people[6] and treatment is rarely required. Atrial fibrillation occurs in 5–10% of

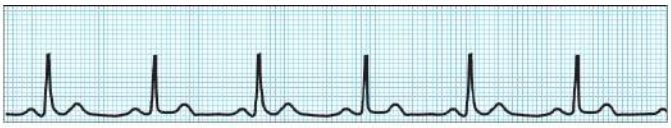
people over 65 years of age, 30% of whom have no clinical evidence of cardiovascular disease, and in around 15% of those over 80 years of age.[7] A rapid ventricular rate with symptoms is the only indication for digoxin in the elderly, but beware of SSS, especially if dizziness or syncope accompanies the fibrillation.

In the elderly, thyrotoxicosis may present as sinus tachycardia or atrial fibrillation with only minimal signs—the so-called 'masked thyrotoxicosis'—so it is easy to overlook it. The only clue may be bright eyes ('thyroid glitter') due to conjunctival oedema (see CHAPTER 14).

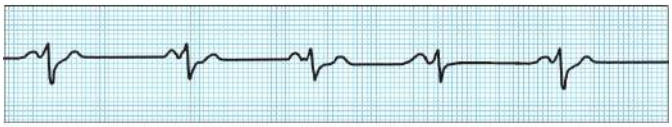
Arrhythmias

Facts and figures

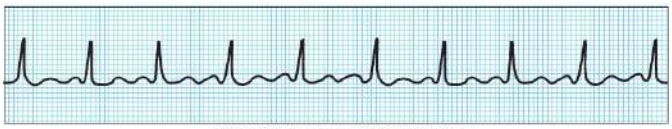
- See FIGURE 59.4 for ECG tracings of important arrhythmias.
- Cardiac arrhythmias account for about 25% of management decisions in cardiology (see TABLE 59.3)
- Commonest are premature (ectopic) ventricular beats and atrial fibrillation.
- PSVT is next most common—6 per 1000 of population.
- The commonest mechanism of paroxysmal tachycardias is re-entry (see FIG. 59.5).
- Electrophysiological studies are the gold standard investigation for tachycardias but are rarely needed for diagnosing most arrhythmias.
- Almost all antiarrhythmic drugs have a proarrhythmic potential (i.e. they may worsen existing arrhythmias or provoke new arrhythmias in some patients) (see TABLE 59.4). Always consider the no-treatment option.
- Sinus tachycardia is usually physiological. If no obvious cause, but the symptoms are troublesome, treat with a beta blocker or verapamil.[8]
- Avoid digoxin in cases with an accessory pathway.
- If 'quinidine syncope' occurs, consider torsade de pointes as the cause.
- Any patient commencing antiarrhythmic therapy should have a 12-lead ECG 1–2 weeks later to check the QT interval. If prolonged, treatment should usually be ceased.
- The two main indications for permanent pacemaking are SSS (only if symptomatic) and complete heart block.



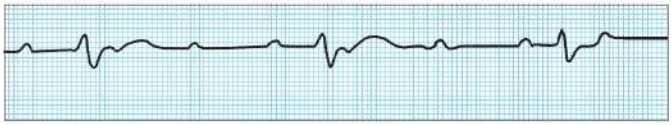
normal sinus rhythm



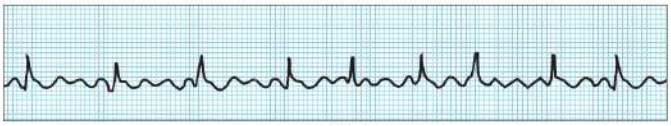
sinus bradycardia and sinus arrhythmia—rate approx. 55 per minute



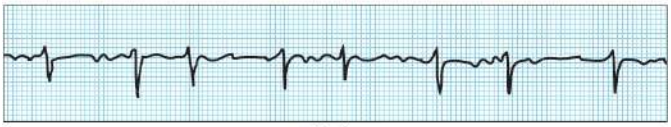
sinus tachycardia—rate approx. 100 per minute



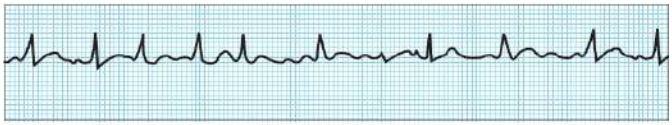
complete heart block



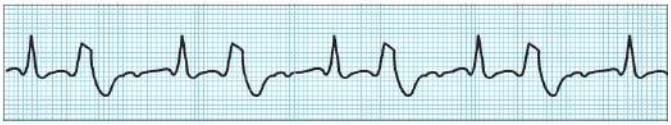
atrial flutter



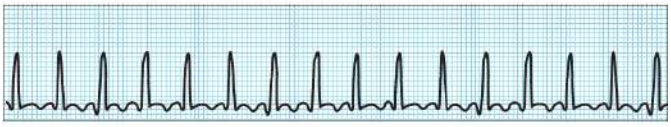
atrial fibrillation



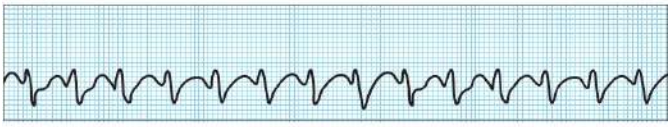
atrial premature complexes



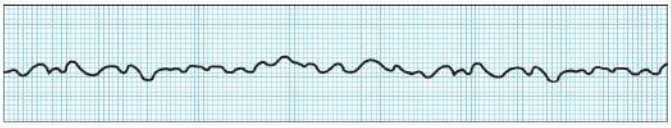
ventricular premature complexes



supraventricular tachycardia



ventricular tachycardia



ventricular fibrillation

FIGURE 59.4 Tracings of important arrhythmias

Table 59.3 Types of arrhythmias

Non-pathological sinus rhythms
Sinus arrhythmia Sinus bradycardia Sinus tachycardia
Pathological bradyarrhythmias
Sinus node disease (sick sinus syndrome) Atrioventricular (AV) block: • first-degree AV block • second-degree AV block • third-degree (complete) AV block
Pathological tachyarrhythmias
1. Atrial: • atrial premature (ectopic) complexes • PSVT • atrial flutter • atrial fibrillation (AF) **2.** Ventricular: • ventricular premature (ectopic) complexes • ventricular tachycardia: non-sustained/sustained • ventricular fibrillation • accelerated idioventricular rhythm • torsade de pointes (twisting of points)[1]

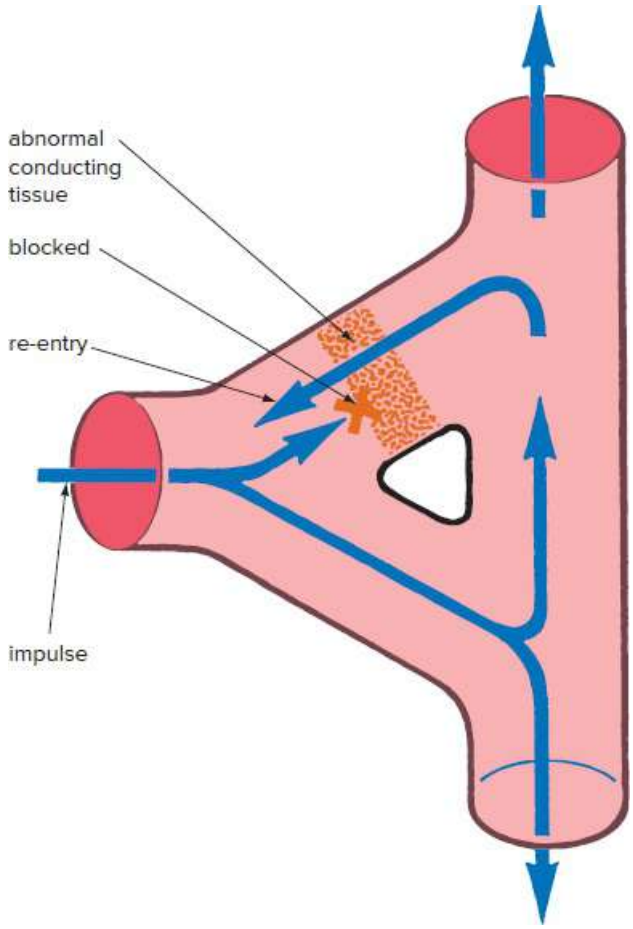


FIGURE 59.5 Diagrammatic mechanism of re-entry tachycardia

Table 59.4 Electrophysiological classification of common antiarrhythmic drugs (Vaughan Williams)

Class	Drug	Usual dosage	Common side effects
I	Membrane depressant drugs (sodium-channel blockers)		
Ia	Disopyramide	100–200 mg qid	Blurred vision, dry mouth, urinary problems in males (avoid in men >50)
	Procainamide	1 g qid IV use	Anorexia, nausea, urticaria
	Quinidine	2–3 SR tabs (0.25 g) bd	Diarrhoea, headache, tinnitus

Ib	Lignocaine	IV use	Nausea, dizziness, tremor
	Mexiletine	200 mg tid	Nausea, vomiting, tremor, dizziness
Ic	Flecainide	100 mg bd	Nausea, dizziness, rash
II	Beta blockers (slows AV conduction)	Various	Fatigue, insomnia, nightmares, hypotension, bronchospasm Avoid in asthmatics
III	Potassium channel blockers (prolongs action potential)	SVT: 200 mg daily VT: 400 mg daily	Rash, pulmonary fibrosis, thyroid, hepatic and CNS effects
	Amiodarone	80–160 mg bd	As for beta blockers
	Sotalol		
IV	Calcium-channel blockers	(SR) 160–480 mg daily	Constipation, dizziness, hypotension
	Verapamil	(CR) 180–360 mg daily	Hypotension, headache
	Diltiazem		

Note: Sotalol is a beta blocker and thus is a class II and III agent. All drugs are taken orally unless IV indicated. Adenosine and digoxin are not classified.

Management strategies

- Treat the underlying cause.
- Give appropriate reassurance.
- Provide clear patient education.
- Explain about the problems of fatigue, stress and emotion.
- Advise moderation in consumption of tea, coffee, caffeine-containing soft drinks and alcohol. Page 727 Page 728
- Advise about cessation of smoking and other drugs.

Sinus bradycardia

Sinus bradycardia can be a normal occurrence, but look for causation (e.g. hypothyroidism, myocardial ischaemia and drugs). Correct the cause. Treatment is required only if symptomatic, which is uncommon at rates >40–45 bpm. Use IV atropine or isoprenaline if acute treatment is required. However, mild or transient bradyarrhythmias may be asymptomatic or even

physiological, such as in a healthy athlete. Palpitations are not a feature but they can cause dizziness, fatigue or syncope (e.g. Stokes–Adams attack—transient bradycardia due to complete heart block).

Stokes–Adams attack

- Sudden onset without warning
- Patient falls to ground
- Collapse with loss of consciousness
- Pallor and still as if dead with slow or absent pulse

- Recovery—in seconds back to normal
- Patient flushes as pulse increases
- Refer for management as attacks may be recurrent

Premature (ectopic) complexes

Premature (ectopic) atrial complexes

- These are usually asymptomatic.
- Management is based on reassurance.
- Check lifestyle factors such as excess alcohol, caffeine, stress and smoking; avoid precipitating factors.
- Drug treatment is rarely required and should be avoided if possible.
- At present there is no ideal anti-ectopic agent.
- They may be a forerunner of other arrhythmias (e.g. PSVT, atrial fibrillation).
- For intolerable symptoms give:[6]

  atenolol or metoprolol 25–100 mg (o) daily

  or

  verapamil SR 160–480 mg (o) daily

Premature (ectopic) ventricular complexes

- These are also usually asymptomatic (90%).
- They occur in 20% of people with ‘normal’ hearts.
- Symptoms are usually noticed at rest in bed at night.
- Check lifestyle factors as for atrial premature beats.
- Drugs that can cause both types of premature beats include digoxin and sympathomimetics.
- Look for evidence of ischaemic heart disease, mitral valve prolapse (especially women), thyrotoxicosis and left ventricular failure.
- Ventricular premature complexes may be a forerunner of other arrhythmias (e.g. ventricular tachycardia).
- If symptomatic but otherwise well with a normal chest X-ray and ECG, reassure the patient.
- Drug therapy: never commence drug therapy without performing an echocardiograph. This will help to guide the choice of agent. Class 1 agents can make the arrhythmia worse or even life-threatening if there is reduced ventricular function. If considering therapy, refer to a cardiologist.
- For troublesome symptoms, the beta blockers atenolol or metoprolol can be used, e.g. atenolol 25–100 mg (o) daily.

Supraventricular tachycardia[9,10]

- SVT can be paroxysmal or sustained.
- Rate is 160–220/min.
- There are at least eight different types of SVT with differing risks and responses to treatment.
- PSVT commonly presents with a sudden onset in otherwise healthy young people.
- Passing copious urine after an attack is characteristic of PSVT.
- Look for predisposing factors such as an accessory pathway and thyrotoxicosis.
- Approximately 60% are due to atrioventricular (AV) node re-entry and 35% due to accessory pathway tachycardia (e.g. WPW).[10]
- Look for evidence of accessory pathways after reversion because accessory pathways can lead to sudden death (avoid digoxin in WPW).
- Consider SSS in a patient with SVT and dizziness.

Wolff–Parkinson–White syndrome

The structural basis for the arrhythmia of SVT in WPW syndrome is an accessory pathway that bypasses the AV node. A typical ECG shows a short PR interval and slurred upstroke of the QRS complex (delta wave). Patients are prone to sudden attacks of SVT. Up to 30% of patients will develop atrial fibrillation or flutter. Even one episode of PSVT requires consideration for radiofrequency ablation.[11]

Management of PSVT

1. Vagal stimulation can be attempted. Carotid sinus massage was the treatment of choice, but it has fallen out of favour because of the potential for stroke. A popular method is to blow hard into the end of an empty 20 mL plastic syringe to move the plunger. Other methods of vagal stimulation include:

- Valsalva manoeuvre (easiest for patient)
- self-induced vomiting
- ocular pressure (avoid)
- cold (ice) water to face or swallowing ice
- immersion of the face in water

2. If vagal stimulation fails, give adenosine IV (try 6 mg first over 5–10 seconds, then 12 mg in 2 minutes if unsuccessful, then 18 mg in 2 minutes if necessary and well tolerated). Second-line treatment is verapamil or metoprolol. Verapamil IV 5–10 mg over 2 minutes. If ineffective but well tolerated, repeat dose after 30 minutes. Metoprolol IV 2.5–5 mg over 2 minutes. If ineffective but well tolerated, repeat dose after 10 minutes.[10]

   Precautions:

- Adenosine causes less hypotension than verapamil but may cause bronchospasm in asthmatics, as may metoprolol
- Use only if narrow QRS and BP >80 mm Hg
- Carefully monitor blood pressure
- AVOID verapamil if taking beta blockers

   and

   persistent tachycardia with QRS complexes >0.14 s (suggests ventricular tachycardia)

3. In the rare event of failure of medical treatment, refer for consideration of DC cardioversion or

overdrive pacing.

Prevention and maintenance

To prevent recurrences (frequent episodes) use first-line atenolol, metoprolol, sotalol or verapamil. Second-line: add flecainide (only if echocardiography shows no structural heart damage). If these agents fail, consider amiodarone. Radiofrequency catheter ablation, which is usually curative, is indicated for frequent attacks not responding to medical therapy.

Carotid sinus massage[1]

Carotid sinus massage causes vagal stimulation and its effect on SVT is all or nothing. It slows the sinus rate and breaks the SVT by blocking AV nodal conduction.

In general, right carotid pressure tends to slow the sinus rate and left carotid pressure tends to impair AV nodal conduction. Never massage both sides simultaneously. As a rule, avoid this method and use one of the simpler alternatives.

Precautions

Avoid in the elderly (risk of embolism or bradycardia).

Ventricular tachycardia

Management requires special supervision.[8,11] It may be non-sustained, where drug treatment such as beta blockers and amiodarone may be used for symptomatic cases, or sustained, which may result in cardiac arrest. An implantable cardioverter defibrillator or radiofrequency catheter ablation may be used.

Torsades de pointes

This uncommon condition is potentially lethal as it may occasionally be prolonged or jump into ventricular fibrillation with sudden death. It is associated with disorders that prolong the QT interval. Treatment involves cessation of drugs that may prolong the QT interval, correcting electrolyte imbalance (especially potassium), cardiac pacing and IV magnesium sulphate or IV isoprenaline.[8]

Atrial fibrillation

Facts and figures

- A common problem (9% incidence in the over-65 age group).[7]
- It usually presents with an irregular ventricular rate of about 160–180 bpm in untreated patients with a normal AV node.

- Apart from acute AF (new onset <48 hrs), it tends to fall into one of the ‘three Ps’ patterns:

  paroxysmal AF: abrupt onset, spontaneous return to normal rhythm (usually lasts <48 hrs)

  persistent AF: abrupt onset, lasts >7 days

  permanent (chronic) AF: cannot be converted to normal rhythm

All types appear to have a similar risk for thromboembolism.

- Remember to look for the underlying cause: myocardial ischaemia (15% of cases), mitral valve disease, thyrotoxicosis, hypertension (60–80% association), pericarditis, diabetes mellitus, cardiomyopathy including chronic alcohol dependence, alcohol binge.
- No cause is found in 12%—isolated or ‘lone’ atrial fibrillation.[8,11]
- All patients should have thyroid function tests and an echocardiograph to help find a cause.
- With sustained atrial fibrillation there is a 5% chance per annum of embolic episodes. There is a fivefold risk of CVA overall.
- The risk of CVA is greater in those with previous CVA, valvular heart disease, prosthetic mitral valve and cardiac failure.
- For reversion anticoagulate with warfarin for 4 weeks beforehand and maintain for 4 weeks afterwards.
- Digoxin controls the ventricular rate but does not terminate or prevent attacks.
- Sotalol, flecainide and amiodarone are used for conversion of atrial fibrillation and maintenance of sinus rhythm. Flecainide should never be prescribed in patients with reduced LV function.
- Evidence basis: RCTs showed that digoxin was beneficial for lowering the ventricular rate in the short term but no better than placebo in restoring rhythm. Beta blockers and calcium-channel antagonists benefitted rate control but verapamil was much less effective than amiodarone at restoring cardiac rhythm.[12]

Atrial flutter

The ECG of atrial flutter has a regular saw-tooth baseline ventricular rate of 150 with narrow QRS complexes. This is a 2:1 AV block. It is often misdiagnosed as SVT. Rarely, conduction occurs 1:1, giving a ventricular rate of 300/min. Treatment is via DC shock or overdrive pacing if indicated (seek specialist advice). Otherwise, drug therapies are identical to those used in atrial fibrillation.

Treatment for atrial fibrillation/flutter[10,11]

As soon as AF is diagnosed, commence anticoagulation therapy unless contraindicated (high bleeding risk) or very low risk of CVA (low CHA_2DS_2-VASc score). Anticoagulation can commence before specialist review, and affects mortality more than decisions around rate/rhythm control. Paroxysmal or persistent atrial fibrillation confers a risk of thromboembolism similar to permanent atrial fibrillation.

Decisions around rate and rhythm control are best made in consultation with a specialist. The AFFIRM study[13] confirmed that there was little overall difference in outcomes between groups whose rate vs rhythm was controlled. However, in general control rate in those who are asymptomatic with good left ventricular function, and control rhythm in the highly symptomatic and with poor LV function.

Anticoagulation[10]

(Refer to CHAPTER 122 .)

Those with ‘valvular’ AF (moderate mitral stenosis or mechanical heart valve) require warfarin (and not a DOAC).

For everyone else with AF, the decision to anticoagulate generally follows the CHA_2DS_2-VASc score. Males who score 0 and women who score 0 or 1 do not require anticoagulation. Scores 1–2 points above this threshold usually benefit from cardiology review, whereas higher scores (male ≥2, female ≥3) should commence anticoagulation unless contraindicated (high bleeding risk).

Use:

Either a direct-acting oral anticoagulant (DOAC) (if Cr clearance >30 ml/min)

- dabigatran, apixaban or rivaroxaban

or

- warfarin—start with a low dose (e.g. 2–4 mg) and maintain a relatively low INR of 2–3 with regular checks. (It is the appropriate agent for valvular AF.)
- Do not use aspirin, clopidogrel or ticagrelor for this purpose.

Rate control

Aim for a ventricular rate >50 bpm and <110 bpm at rest (ideally <90 bpm).[10]

Rapid, urgent control of ventricular rate:

Metoprolol 5 mg (1 mg/min) IV to max 20 mg (provided no evidence of heart failure and well-monitored BP)

or

verapamil 2.5–10 mg/min IV over 2–3 minutes, up to maximum 15 mg then orally

or

esmolol

Routine control and maintenance[10]

Atenolol 25 mg (o), increasing to 100 mg daily prn

or

metoprolol 25 mg (o) bd, increasing up to 100 mg bd

or (if blockers contraindicated)

verapamil SR 180–480 mg (o) daily

or

diltiazem CR 180–360 mg (o) daily

Digoxin still has a place in the elderly, especially with cardiac failure:

digoxin 0.0625–0.25 mg (o) daily according to age, plasma creatinine and digoxin level

Amiodarone has a place in management.

Rhythm control

This should be considered if the patient is symptomatic and the arrhythmia is of recent onset—less than 6 months. Ensure anticoagulation first (as per above, or heparin if urgent cardioversion).

Medical cardioversion

Amiodarone or flecainide

If the rate cannot be well controlled despite maximal medical therapy, consider AV node ablation and a permanent pacemaker. Atrial fibrillation with a rapid ventricular response over a long period gradually causes left ventricular dysfunction.

Synchronised electrical DC cardioversion

This can be given for first-line treatment or for failed medical conversion.

Other treatment for arrhythmias

A number of non-pharmacological treatments are useful for particular arrhythmias.

Rate-responsive pacemakers

These respond to signals other than purely the atrial rate, taking into account real-time physiological needs (such as exercise) in those with bradycardia.

Radiofrequency ablation

Specific abnormal foci in the conducting pathways can be ablated using direct current electrical surgery or radiofrequency burns via a catheter electrode. Radiofrequency or cryotherapy catheter ablation is indicated for recurrent episodes of supraventricular tachycardia, accessory pathways and nodal re-entry tachycardia with success rates of up to 95%.

Automatic implantable cardioverter defibrillator (AICD)

This implant is the most effective therapy yet devised for the prevention of sudden cardiac death in patients with documented sustained ventricular tachycardia or fibrillation. Operative mortality should be less than 10%, after which survival at 1 year is over 90%. These new defibrillators incorporate an antitachycardia pacemaker. Patients can either be paced out of arrhythmia or, if they develop ventricular fibrillation, they can be defibrillated using higher energy.

Surgery

Less common as catheter ablation methods improve. Usually done concurrently with other heart surgery. Guided by electrophysiological monitoring, a small section of the AV ring is dissected to sever all aberrant connections between the atria and the underlying ventricular muscle.

TABLE 59.5 presents a summary of the treatment of arrhythmias.

Table 59.5 Summary of treatment of arrhythmias[7,8]

Arrhythmia	First line	Second line	Third line
Sinus tachycardia	Treat cause Reduce caffeine intake	Metoprolol or atenolol *or* Verapamil (rarely indicated)	

Bradycardia			
Sick sinus syndrome	Permanent pacing if symptomatic or persistent		
AV block			
First degree	No treatment		
Second degree: • Mobitz I • Mobitz II	No treatment Consider pacing	Pacing if problematic	
Third degree: • acute (e.g. MI) • chronic	Temporary pacing Permanent pacing	Adrenaline IV	Atropine Isoprenaline
Atrial tachyarrhythmias			
PSVT	Valsalva manoeuvre	Adenosine IV *or* Verapamil IV	DC cardioversion Class III drug? Ablation
	Rate control with beta blocker *or* verapamil (with care)	Add digoxin (if necessary)	AV node ablation + permanent pacemaker
	Rhythm control with cardioversion, electrical or drugs	Cardioversion → *or* Maintenance of sinus rhythm → Sotalol, flecainide, amiodarone + anticoagulation	Electrical or chemical
Atrial premature complexes	Treat cause Check lifestyle	Metoprolol or atenolol *or* verapamil	
Ventricular tachyarrhythmias			
Ventricular	Treat cause	Beta blocker	Class I or III

premature beats	Check lifestyle	(especially mitral valve prolapse)	drugs (rarely needed)
Ventricular tachycardia: • non-sustained • sustained	Beta blocker or amiodarone or sotalol if stable—if not: DC shock	Lignocaine IV	DC cardioversion
Ventricular fibrillation	DC cardioversion	IV adrenaline if fine VF then DC cardioversion	Amiodarone (maintenance) Class III (if recurrent)
Torsades de pointes	Correct cause, e.g. potassium Cease drugs if ↑ QT	Cardiac pacing, IV magnesium sulphate	IV isoprenaline

When to refer[14]

Patients should be referred to a cardiologist[13] when:

- a sustained supraventricular tachycardia is suspected
- a sustained ventricular tachycardia is suspected
- an ECG shows sustained delta waves of WPW syndrome, even if asymptomatic

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- syncope or dizziness suggests a cardiovascular cause
- a paroxysmal arrhythmia may be the cause of unexplained cardiovascular symptoms
- the need for anticoagulation, rate or rhythm control is uncertain

Practice tips[15]

- Atrial fibrillation and dizziness (even syncope) are suggestive of SSS (bradycardia–tachycardia syndrome), which is made worse by digoxin.
- Consider thyrotoxicosis as a cause of atrial fibrillation or sinus tachycardia, even if clinical manifestations are not apparent.
- Check for a history of palpitations in anyone complaining of dizziness or syncope (and vice versa). Consider an arrhythmia, especially in the elderly.

- PSVT is rarely caused by organic heart disease in young patients.
- Arrhythmia of sudden onset suggests PSVT, atrial flutter/fibrillation or ventricular tachycardia.
- A normal ECG in sinus rhythm does not exclude an accessory pathway.
- Consider conduction disorders such as the WPW syndrome in PSVT. Avoid digoxin in WPW syndrome.
- Common triggers of premature beats and PSVT are smoking, anxiety and caffeine (especially 8 or more cups a day).

Patient education resource

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

- Atrial fibrillation

References

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60 Sleep disorders

Sleep . . . the first great natural resource to be exhausted by modern man. The erosion of the nerves, not to be halted by any reclamation project, public or private.

IRWIN SHAW, 'THE CLIMATE OF INSOMNIA', *THE NEW YORKER*, 1949

Sleep is a fundamental need in humans. Disorder of this basic function is one of the most common health-related problems presenting to the GP. It may represent the clue to some very important disorders, such as depression, anxiety, adverse drug reactions, drug abuse and obstructive sleep apnoea (OSA), which is the most common form of sleep disordered breathing. About half of the population report having some sleep-related problem in a year, and 25% of the Australian population report trouble getting enough sleep when asked.[1] Normal sleep requirement varies considerably.

EEG studies indicate that sleep is divided into rapid eye movement (REM—also called dream sleep) and non-rapid REM sleep (NREM), which is subdivided into stages 1, 2, 3 and 4. Most stage 4 sleep (deepest) occurs in the first hours. REM sleep is accompanied by dreaming and physiological arousals; some dreaming occurs in NREM.

Disorders of the sleep–wake cycle, which are invariably caused by a disruption of the body's endogenous time clock, can result in insomnia or hypersomnolence (excessive sleepiness) or a combination of both. This is familiar to shift workers and people with jet lag.

Key facts and checkpoints

- Normal sleep: in a fit young person the ideal is 7.5–8 hours; latency <30 minutes; wakefulness within sleep usually <5% of time.
- Humans can stay awake without a problem for 16–18 hours. Sleepiness is wake-state instability.
- The evaluation of sleep disorders involving the sleep–wake cycle is enhanced by the patient keeping a sleep chart.
- It is important to take a drug history from those complaining of insomnia or hypersomnolence.

- Drugs that can disturb sleep include alcohol, nicotine, antihistamines, SSRIs, caffeine, hypnotics, venlafaxine, selected β-blockers (e.g. propranolol), β_2-agonists, theophylline, corticosteroids, sympathomimetic agents.
- Sleep disorders in children, including snoring, should be taken seriously and investigated. They have many potential consequences, such as learning and behavioural difficulties, hyperactivity, failure to thrive and short stature.
- A claimed or actual presentation of insomnia accompanied by a medication request, particularly by a younger person, can reflect benzodiazepine dependency.
- People with OSA usually present with the TATT syndrome—'tired all the time'—or excessive daytime sleepiness. These patients are often unaware of waking or becoming aroused during the night.
- A patient who snores, has witnessed apnoeas and sleepiness is likely to have OSA.
- The majority of cases of excessive somnolence are caused by OSA and narcolepsy.[2]
- Non-pharmacological therapies, which include basic education and practice of sleep hygiene and behavioural therapy, should be used in management wherever possible.
- Referral to a specialist sleep disorder centre provides enhanced objective evaluation, diagnosis and treatment of the more complex disorders.
- It is illegal for a driver with a commercial driver's licence to continue to drive while suffering from untreated OSA.

Sleep-related disorders

Disorders of sleep are a common and significant contribution to community illness and death. Those with severe OSA have more than twice the risk of death compared to those without OSA (HR=2.1), mainly from cardiovascular and motor vehicle accident related deaths. CPAP treatment reverses much of this excess mortality.[3] A classification of sleep disorders is presented in TABLE 60.1 .[4]

Table 60.1 Classification of sleep and wake disorders (DSM-5)[4]

Insomnia disorder

Hypersomnolence disorder
Narcolepsy
Breathing-related sleep disorders
- obstructive sleep apnoea/hypopnoea
- central sleep apnoea
- central alveolar hypoventilation syndrome

Circadian rhythm sleep disorders
Non-REM* sleep arousal disorder
Nightmare disorder
REM* sleep behaviour disorder
Restless legs syndrome
Substance- or medication-induced sleep disorder

*REM = rapid eye movement

Many conditions may disturb breathing during the night (see FIG. 60.1). Nocturnal dyspnoea may result from cardiac causes (mitral stenosis, ischaemic cardiomyopathy, cardiac arrhythmias, fluid overload or retention), which usually present with orthopnoea, pulmonary crepitations and peripheral oedema. Asthma is another common cause of nocturnal dyspnoea, cough (with or without wheeze) occurring classically between 2 am and 5 am. Gastro-oesophageal reflux with or without aspiration may disturb respiration at night, but it usually presents with daytime or postural reflux. All these conditions can usually be differentiated from sleep apnoea clinically or with further investigation.

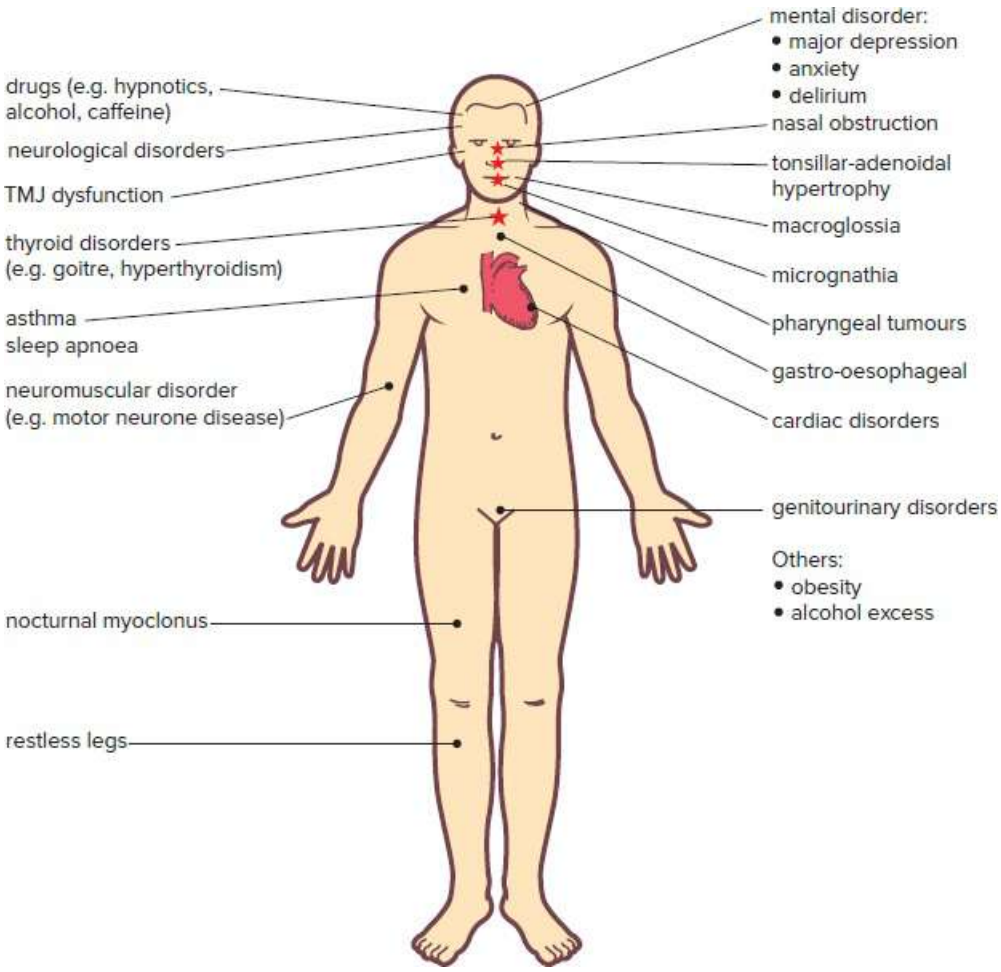

FIGURE 60.1 Significant causes of sleep disturbance

The sleep apnoea syndromes are a common group of disorders that result in periodic hypoventilation during sleep. They occur in about 2% of the general population in all age groups, and in about 10% of middle-aged men.

Insomnia

Insomnia is defined as the inability to initiate or maintain sleep. The person may complain of difficulty getting to sleep or staying asleep, of frequent intermittent nocturnal arousals, early morning awakening or combinations of these.

A careful history is required because some individuals have unrealistic expectations about the required amount of sleep they need or have misperceptions of how long they have slept.

When taking the history, explore lifestyle factors, especially psychological reasons, painful conditions, drug use and abuse, appetite, energy, sexual issues and physical factors. Ask about

sleep hygiene: the habits and physical conditions surrounding getting to bed. Check thyroid status, especially hyperthyroidism.

Basic principles for managing insomnia[4,5]

- Address anxiety about sleep.
- Address maladaptive behaviours around sleep.
- Address fear of further sleep disruption.
- Consider predisposing dispositions.
- Consider comorbidities.
- Assess pharmacological options, but consider hypnotics only as a short-term measure.
- Refer to a specialist if there are ongoing symptoms despite the above considerations.

Management[5]

1. Discuss and agree on the therapeutic objective (e.g. to reinstate sleep without medication). If using hypnotics, consider formalising this agreement in a ‘contract’.
2. Sleep–wake history: take a sleep–wake history (preferably with a sleep diary) and evaluate daytime functioning.
3. Exclude and treat any underlying problem (see TABLE 60.2).
4. Explanation and reassurance, including patient education handout.
5. Sleep hygiene advice:
    - Try to recognise what helps the person to settle best (e.g. warm bath, music).
    - Establish a routine before going to bed.
    - Regular daytime exercise.
    - Regular time of arising.
    - Avoid daytime naps.
    - Avoid strenuous exercise close to bedtime.
    - Avoid alcohol and drinks containing caffeine in the evening, especially close to retiring.
    - Avoid caffeine after midday.

- Avoid a heavy evening meal within 3 hours of bedtime.
- Avoid smoking, especially in the evening.
- Remove pets from the bedroom.
- Use a suitable mattress and pillow for comfort and support.
- Ensure a dark, quiet room for sleeping.
- Avoid lights, including poorly screened windows and highly illuminated clocks in the room.

Sleep-promoting adjuvants:

- Try a drink of warm milk before retiring.
- Organise a comfortable, quiet sleep setting with the right temperature.
- Try a warm bath before bed.

5. Non-pharmacological treatment: psychological and behavioural interventions are effective. Various techniques should be adopted according to patient personality and preference, the clinician's expertise and the available resources. These include relaxation therapy, meditation and stress management, which are all highly recommended. Other measures include sleep restriction programs, cognitive behaviour therapy (first-line treatment), structured problem solving and electromyographic feedback. Hypnosis is also worth considering.

7. Pharmacological treatment:[6] it is advisable to avoid hypnotic agents as first-line treatment. If any form of continuous agent is necessary, it is best to limit it to 2 weeks.

Options:

temazepam 10 mg tablets (o) before bedtime

or

consider other non-benzodiazepine options, e.g. GABA agents such as zopliclone, sedating TIAs, melatonin PR, antihistamines (e.g. promethazine, doxylamine).

Note:

- The cyclopyrrolone derivative zopiclone and the imidazopyridine derivative, zolpidem, are non-benzodiazepine hypnotics with a similar action to the benzodiazepines represented by temazepam. However, warnings have been issued about adverse neurological and psychiatric reactions.[6]
- Tricyclic antidepressants with sedative effects (e.g. amitriptyline) are often used as hypnotics but should generally be avoided in the absence of depressive disorders, especially in older people.

Table 60.2 Common causes of insomnia

Drugs (prescribed, illicit, stimulants, addictive, alcohol)
Anxiety, stress (esp. PTSD)
Depression
Inappropriate sleep hygiene or lifestyle
Delirium and dementia
Biorhythm disruption, e.g. shift work, travel
Restless legs syndrome
Pain, e.g. carpal tunnel syndrome
Sleep apnoea
Parasomnias—nightmares, sleepwalking
Physical disorders (e.g. CCF, arthritis, asthma)
Bed-wetting
Reflux disease
Thyroid disorders (e.g. thyrotoxicosis)
Menopausal symptoms
Snoring partner
Lower urinary tract symptoms with nocturia

Delayed sleep phase syndrome

This circadian rhythm disorder is a differential diagnosis of common insomnia. Sufferers struggle to fall asleep, which may be delayed by 2 or more hours beyond the socially acceptable or conventional bedtime. Diagnosis is based on the history and sleep studies. People should follow their biological cues and factor in extra time to sleep. Hypnotics are discouraged.

Sleep apnoea[7]

The term ‘sleep apnoea’ is used to describe cyclical brief interruptions of ventilation, each cycle lasting 15–90 seconds and resulting in hypoxaemia, hypercapnia and respiratory acidosis, terminating in an arousal from sleep (often not recognised by the person). The interruption is then followed by the resumption of normal ventilation, a return to sleep and further interruption of ventilation.

Sleep apnoea is broadly classified into obstructive and central types.

Obstructive sleep apnoea (OSA) refers to the presence of apnoeas (no breathing) and hypopnoeas (abnormally low or shallow breathing) during sleep together with daytime

dysfunction, predominantly excessive daytime sleepiness. The effects include snoring (see FIG. 60.2).

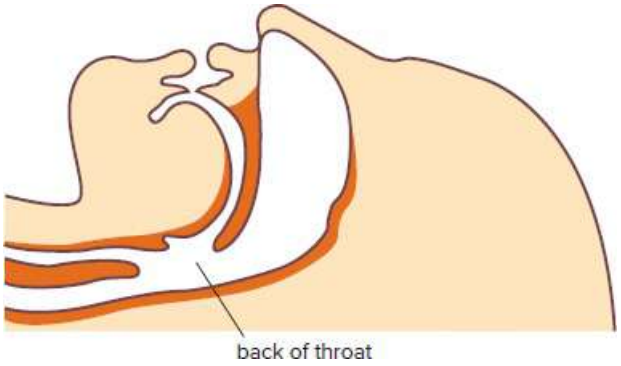

FIGURE 60.2 Normal airway when sleeping

Predisposing causes include:

- diminished airway size (e.g. macroglossia, obesity, tonsillar-adenoidal hypertrophy)
- upper airway muscle hypotonia (e.g. alcohol, hypnotics, neurological disorders; see FIG. 60.3)
- nasal obstruction

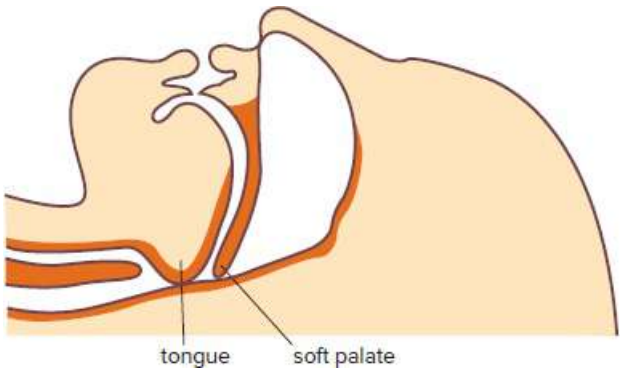

FIGURE 60.3 Sleep apnoea: obstructed airway when sleeping

Central sleep apnoea (CSA), which occurs when there is either brief or prolonged loss of breathing during sleep, is less common (accounts for <10% of sleep-disordered breathing). It is due mainly to neurological conditions such as brain-stem disorders leading to reduced ventilatory drive, and neuromuscular disorders such as motor neurone disease. Cardiorespiratory disease and regular opioid use are also risk factors. It requires specialist referral. Treatment of CSA is based on optimal therapy for these underlying conditions and attending to lifestyle modification as outlined below.

Clinical effects of sleep apnoea syndromes[4,7]

Important clinical presentations include:

- excessive daytime sleepiness and tiredness
- nocturnal problems (e.g. loud snoring, heavy sweating, thrashing, ‘seizures’, choking, pain reactions)
- morning headache
- subtle neuropsychiatric disturbance—learning difficulties, loss of concentration, irritability, personality change, depression
- sexual dysfunction, e.g. erectile dysfunction
- occupational and driving problems

Causes of excessive daytime sleepiness are presented in TABLE 60.3 . In OSA, sleepiness results from repeated arousals during sleep and the effects of hypoxaemia and hypercapnia on the brain. Physical examination may reveal few or no signs.

Table 60.3 Causes of excessive somnolence

Inadequate sleep duration
Sleep apnoea syndromes
Narcolepsy
Endocrine (e.g. hypothyroidism, hyperparathyroidism, hypercalcaemia)
Drug induced
Purposeful sleep deprivation
Nocturnal myoclonus
Bereavement
Idiopathic

Management of sleep apnoea[7]

Referral to a comprehensive sleep disorder centre especially for overnight in-laboratory sleep polysomnography (the gold standard diagnostic test) is advisable if this disorder is diagnosed or suspected. Diagnosis requires recurrent apnoeas (i.e. absent airflow >10 seconds) or hypnoeas (reduction in airflow >10 seconds) sufficient to cause fall in PaO2 or arousal failure occurring at least 5 times an hour, association with snoring and symptoms of daytime sleepiness. An

apnoea/hypnoea index ≥5 is significant. Consider also the validated Berlin questionnaire[8] or the Epworth sleepiness scale.[9]

The general principles are as follows:

1. Lifestyle modification

   - Weight loss (e.g. loss of 10–15%, e.g. 7–10 kg can significantly reduce severity).
   - Achieve physical fitness with regular exercise.
   - Good sleep hygiene and adequate sleep hours (increase time in bed).
   - Reduce or cease sedatives/hypnotics.
   - Avoid alcohol for up to 3 hours before going to sleep and drugs in general.
   - Cease cigarette smoking (increases nasal resistance).
   - Medical management of nasal obstruction (e.g. short-course nasal decongestants) or 6-week trial intranasal topical corticosteroids.
   - Obtain positional therapy, avoid supine sleep—sleeping on side is best. Consider neck support.

2. Continuous positive airway pressure (CPAP)

   - CPAP is currently the most effective treatment for OSA (consider it for CSA)—the gold standard treatment.
   - Delivered by facial (or nasal) mask.
   - Provides an air splint to the upper airway and prevents pharyngeal collapse.
   - Sleepiness and neurocognitive function improved.
   - Not tolerated by everyone.

3. Surgery

   In children, OSA is usually due to tonsillar and/or adenoid hypertrophy and is relieved by surgery (see FIG. 60.4). In adults, depending on the cause, the options are:

   - correction of the specific upper airway anatomical problem—up to 2% of problems
   - correction of nasal obstruction (improves snoring and OSA)
   - palatal surgery: uvulopalatopharyngoplasty for carefully selected patients—conventional or laser assisted

- nasal polypectomy
- tonsillectomy
- base of tongue surgery
- radiofrequency treatment to soft palate and base of tongue (‘somnoplasty’)
- bariatric surgery if indicated

4. Oral appliances
   - The mandibular advancement splint—a custom-made dental device that supports the mandible and tongue during sleep to increase pharyngeal dimensions. An effective alternative to CPAP.
5. Medication

   There are no reliable drug treatment options for OSA. Consider:

   - amitriptyline 25–100 mg (o) nocte, in severe cases during REM sleep and intolerance of CPAP
   - trial of corticosteroid sprays in children with mild OSA

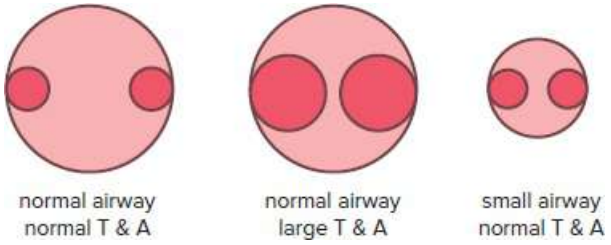

FIGURE 60.4 Influence of tonsils and adenoids on airways

Obesity hypoventilation syndrome (OHS)—Pickwickian syndrome

OHS may occur alone or secondary to OSA. The main features of the OHS patient are:

- marked/morbid obesity
- cyanosis or plethora
- excessive daytime sleepiness
- right heart failure

There is impaired breathing leading to hypercapnia (↑ $PaCO_2$) and hypoxia. Diagnosis is by sleep studies. Treatment is weight loss plus CPAP. It is a complex problem with a risk of premature death.

Narcolepsy

Narcolepsy is a specific, permanent neurological disorder that is characterised by brief spells of irresistible sleep during daytime hours in inappropriate circumstances, even during activity and usually at times when the average person simply feels sleepy. It is uncommon with an incidence of two to five per 100 000.

Features

Usual onset between adolescence and 30 years of age—in teens and 20s (but has been reported in children as young as 2 years).

Tetrad of symptoms

- Daytime hypersomnolence: sudden brief sleep attacks (15–20 minutes).
- Cataplexy: a sudden decrease or loss of muscle tone in the lower limbs that may cause the person to slump to the floor, unable to move. These attacks are usually triggered by sudden surprise or emotional upset.
- Sleep paralysis: a frightening feeling of inability to move while drowsy (between sleep and waking).
- Hypnagogic (terrifying) hallucinations on falling asleep or waking up (hypnopompic hallucination).

Several attacks per day are possible.

Diagnosis

The diagnosis is clinical through the taking of an appropriate history. If doubtful, include:

- EEG monitoring
- sleep laboratory studies (sleep latency test—rapid eye movement is a hallmark)

Treatment[5]

Treatment is mainly symptomatic and initiated by a consultant. Central nervous system psychostimulants (dexamphetamine, methylphenidate) are of proven effectiveness in increasing alertness. Drug holidays from these drugs may be necessary.

- Tricyclic antidepressants (e.g. clomipramine) are used to treat cataplexy, sleep paralysis and

hypnagogic hallucinations.

- Modafinil is used successfully in some countries.
- Reflect on driving licence issues as appropriate.

Idiopathic hypersomnia[10]

This type of excessive daytime sleepiness (EDS) can present similarly to narcolepsy without cataplexy. The condition, which accounts for 5–10% of patients in sleep clinics with EDS, is diagnosed despite adequate sleep and exclusion of other causes. They usually have non-refreshing deep nocturnal sleep but, unlike narcolepsy, naps are not refreshing. The onset is usually insidious before 30 years and persists for life. Treatment is usually based on psychostimulant therapy to improve EDS.

Snoring

Definition

Snoring is a sonorous sound with breathing during sleep, caused by soft tissue vibrations in the upper airways from the nose to the back of the throat. It is caused by partially obstructed breathing during sleep (see FIG. 60.5).

FIGURE 60.5 Snoring: vibrations of tongue and soft palate

Features

- Sometimes indicates OSA, especially in perimenopausal women[7]
- Three times more common in obese persons
- Generally harmless, but if very severe, unusual or associated with periods of no breathing (>10 s) assessment is advisable

Aggravating factors[9,11]

- Obesity
- Old age
- Sleeping on the back
- Sleep deprivation
- Excess alcohol
- Neck problems, especially a ‘thick’, inflexible neck
- Various drugs, especially sedatives and sleeping pills
- Hay fever and other causes of nasal congestion
- Problems in the upper airways, such as nasal polyps, enlarged tonsils or a foreign body (e.g. a piece of plastic or metal) Page 741
- Endocrine abnormalities (e.g. acromegaly, hypothyroidism)

Management

If an examination rules out a physical problem causing obstruction in the back of the nose and OSA, then the following simple advice can be given.

- Obtain and keep to ideal weight.
- Avoid drugs (including sedatives and sleeping tablets), alcohol in excess and smoking.
- Treat nasal congestion (including hay fever) but avoid the overuse of nasal decongestants (rebound effect).
- For neck problems, keep the neck extended at night by wearing a soft collar.
- Consider a trial of an intranasal device such as the Breathing Wonder, which is a hollow intranasal plastic insert and may work for a subset of snorers, or the Clippie soft silicone ring device. Pharmacists can advise about the range of such devices.
- Try to sleep on the side. To avoid the tendency to roll on to the back at night, a maverick method is to consider sewing ping pong balls or tennis balls on the back of nightwear, or wear a bra or shirt with a pocket (with tennis ball) back to front.

Periodic limb movements (nocturnal myoclonus)

Periodic limb movements (PLMs) and restless legs syndrome are important causes of insomnia and excessive daytime sleepiness. They may coexist in the same person. Periodic limb movements, which are also referred to as nocturnal myoclonus or ‘leg jerks’, tend to occur

usually in the anterior tibialis muscles of the leg but can occur in the upper limbs. The prevalence increases with age. Most people with PLMs are completely asymptomatic. The diagnosis is often made during sleep studies.[12] If troublesome, refer to a sleep clinic or neurologist.[12]

Medication

Medication that may help includes:

levodopa plus carbidopa (e.g. Sinemet 100/25, tablets before bedtime)

or

clonazepam 1 mg (o) nocte increasing to 3 mg (o) nocte

or

sodium valproate 100 mg (o) nocte

Sleep paralysis

This is the temporary inability to move or speak when falling asleep or upon awakening, with intact consciousness. It is REM atonia. It can occur in the general population (8%) and about 30% of those with narcolepsy. It can be terrifying but is not dangerous.

Treatment is educational and preventive with optimal sleep hygiene.

Restless legs syndrome (RLS)

Restless legs syndrome is a rather common movement disorder of the nervous system where the legs feel as though they want to exercise or move when the body is trying to rest. Sensations that may be experienced include ‘twitching’, ‘prickling’ and ‘creeping’.[13] The major complaint of sufferers is of disruption both to sleep and of relaxing activities, such as watching television or reading a book. Prolonged car or airplane travel can be difficult.

RLS is frequently an undiagnosed disorder because people often don’t complain about it to their doctor. A US and European survey of adult GP patients suggested a 12% prevalence, with 2.5% reporting an impact on quality of life.[14]

The diagnosis is made from the history—there are no special diagnostic tests.

Its prevalence increases with age so it mainly affects elderly people. Women are more prone to get RLS and it is aggravated by pregnancy. The exact cause of primary RLS is not clear. It is not related to exercise and does not appear to follow strenuous exercise.

Symptoms

There is an urge to move legs upon resting, particularly after retiring to bed. This urge is a

response to unpleasant sensations in the legs, especially in the calves. The sensations are commonly and variously described as crawling, creeping, prickly, tingling, itching, contractions, burning, pulling or tugging, electric shock-like. However, some people are unable to describe the sensation or refer to it as simply a compulsion to move the legs.

In some, the arms are affected in a similar way. The symptoms seem to be aggravated by warmth or heat. Many people with RLS also experience nocturnal myoclonus.

Secondary (medical) causes include:

- anaemia (common)
- iron deficiency (common)
- uraemia
- hypothyroidism
- pregnancy (usually ceases within weeks of delivery)

- drugs (e.g. antihistamines, anti-emetics, selective antidepressants, lithium, selective major tranquillisers and antihypertensives)

Management

Iron studies should be performed and, if low, treat with iron (good evidence)[15] and vitamin C tablets (limited evidence outside those on haemodialysis). Advise that although RLS can come and go for years, it usually responds well to treatment.

Self-help advice

- Perform activities that can reduce symptoms, for example, a modest amount of walking before bedtime, massage or prescribed exercises (see FIG. 60.6).

  Note: Getting out of bed and going for a walk or run does not seem to help RLS.

- Good sleep hygiene, namely regular sleeping hours, gradual relaxation at bedtime, avoidance of non-sleep activities in bed (e.g. reading, eating).
- Diet: follow a very healthy diet. Avoid caffeine drinks, smoking and alcohol.
- Try keeping the legs cooler than the body for sleeping.
- Exercises: a popular treatment is gentle stretching of the legs, particularly of the hamstring and calf muscles, for at least 5 minutes before retiring. This can be done by using a wide crepe bandage, scarf or other length of material around the foot to stretch and then relax the legs.

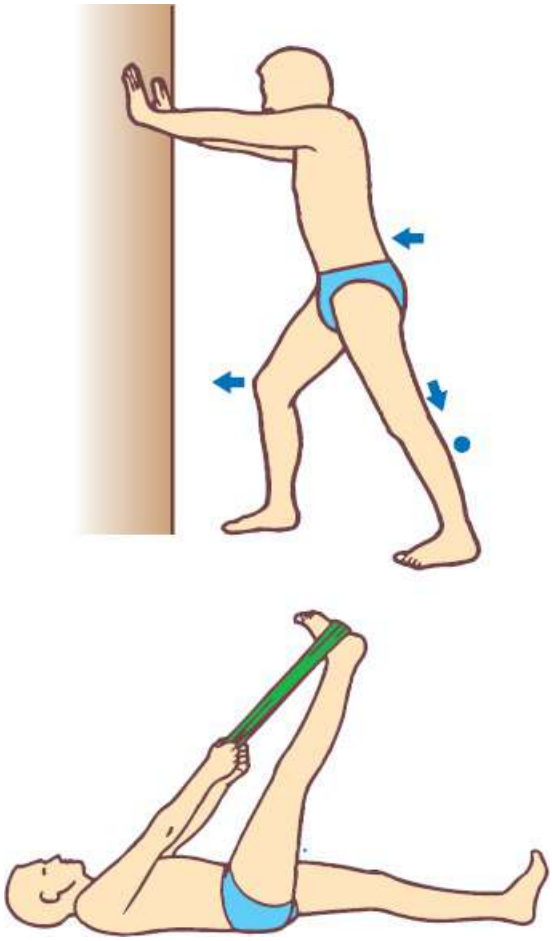

FIGURE 60.6 Stretching exercises for restless legs

Pharmacological treatment[5]

If simple measures fail and iron supplementation is not indicated (i.e. ferritin >50 mcg/L), the following may be effective: (taken before bedtime)

levodopa (+ benserazide or carbidopa) 100–200 mg (o) (especially if limb movements at sleep onset are mild and infrequent [be aware of risk of augmentation, especially at higher dosage])

For more severe symptoms consider low-dose dopamine agonists (first line):

pramipexole 0.125 mg (o), increasing weekly as tolerated to 0.75 mg

or

ropinirole 0.25 mg (o) increasing if tolerated to 0.5 mg, then weekly increases of 0.5 mg up to 2 mg (usual dose) or 4 mg (maximum dose)

or use a gabapentinoid

gabapentin 100–300 mg, increasing every 3–7 days to 1200 mg or beyond

or

pregabalin 75 mg, increasing every 3–7 days to max. 450 mg

Avoid continuing these medications indefinitely just because they were prescribed in the past. Consider a trial of deprescribing (reducing with a view to ceasing the dose).

Benzodiazepines, particularly clonazepam, are often suggested, but have not been supported by any quality evidence. Cabergoline, codeine, baclofen and propranolol may possibly be helpful. Avoid carbamazepine, quinine, antipsychotics, antihistamines and antidepressants.

Bruxism (teeth grinding)

Bruxism is the habit of grinding, clenching or tapping teeth, which may occur while awake (especially in children) or more commonly while asleep. The usual symptom is annoying, teeth-grinding noises during sleep that disturb family members. It may result in headaches and TMJ dysfunction in the person during the day. The cause may be a habit or a response to subconsciously correct a faulty bite by making contact between the upper and lower teeth when the jaws are closed. It is aggravated by stress and is more common in heavy alcohol drinkers and SSRI users.

Management

- Educate the person to recognise, understand and try to overcome the habit.
- Practise keeping the jaws (and teeth) apart.
- Slowly munch an apple before retiring.
- Practise relaxation techniques, including meditation, before retiring.
- Consider other stress-management techniques (e.g. counselling, relaxation exercises, yoga and tai chi).
- Place a hot towel against the sides of the face before retiring to achieve relaxation.
- If this fails and bruxism is socially unacceptable, a plastic night-guard mouthpiece can be fashioned by a dentist to wear at night.

Parasomnias

Parasomnias are defined as dysfunctional or unusual behaviour episodes associated with sleep, sleep stages or partial arousal. They are more common in children. Diagnosis is clinical.

Nightmares (dream anxiety)

These dream-related anxiety episodes usually occur later in the REM sleep period and are accompanied by unconscious body movements, which usually cause the person to awaken.

Associations include traumatic stress disorders, fever, drug withdrawal (e.g. alcohol, barbiturates, drugs such as zolpidem, SSRIs, beta blockers, benzodiazepines, mirtazapine). Violent behaviour can occur during these dreams due to a REM behaviour disorder and this requires a sleep study and specialist evaluation.

Psychological evaluation with cognitive behaviour therapy (CBT) is appropriate. Medications that may help include phenytoin or clonazepam.

REM sleep behaviour disorder

A feature is complex and elaborate motor activity associated with dreams. The behaviour may be violent with profane verbalisation. It is more common in older males and among those with CNS degeneration disorders (e.g. dementia, Parkinson disease). Diagnosis is by sleep studies and treatment is low-dose clonazepam.

Somnambulism (sleepwalking)[2]

This is a complex motor activity in which the person performs some repetitive activity in bed or walks around freely while still asleep. There is amnesia for the event. No treatment is usually required but, if it is repetitive and potentially dangerous, then the sleeping environment should be rendered safe. Psychological assistance is required for recurring episodes. Benzodiazepines such as clonazepam 0.5–2 mg (o) nocte may temporarily help, but withdrawal usually leads to rebound problems.

Sleep-related leg cramps

These usually occur in the muscles of the calf and foot. Prevention is by stretching the affected muscles for several minutes before sleep. Magnesium is often promoted as a preventative, but clinical trials suggest its effect is either minimal or non-existent.[16] Refer to CHAPTER 55 .

Sleep terrors

These are part of the same non-REM sleep cycle disorder as somnambulism. Characteristics of night terrors are sharp screams, violent thrashing movements and autonomic overactivity, including sweating and tachycardia. The sufferers, usually preadolescent, may or may not wake and usually cannot recall the event. They also require psychological evaluation and therapy. Similar medication as used for nightmares may help (e.g. a 6-week trial of phenytoin or clonazepam).

Sleep disorders in children

Sleep disorders in children are very common in late infancy, toddlerhood and early preschool age groups. By 3 months 70% begin to sleep through the night. Over 50% of toddlers and preschool children resist going to bed.[12] At least 30% of infants and toddlers wake at least once during the night every night. Toddlers begin to have dreams coinciding with language development in the second year of life.[17]

The child who wakes during the night needs reassurance, protection and the parent's presence, but ideally given discreetly without too much 'fuss'. Although psychosocial stresses can trigger sleep problems, serious psychological problems in children with sleep disorders are uncommon.[18]

Management[17]

If both parents are caregivers, aim to see them together and get them to agree on the approach, including sharing the workload. Check that they both agree that the aim is to have the child sleep through the night with no or minimal parental attention. If that is the case (it usually is if they come to the GP), advise the following:

- Unless you are happy to have your child regularly sleeping with you, resist taking them into bed during the night.
- Beware of giving much attention to your child in the middle of the night—it encourages repeated desire for attention in future nights.
- Avoid extra feeding or other pacifiers during the night.

- Return the child to bed promptly and spend only a brief time to give reassurance.
- A regular series of rituals performed before bedtime helps the child to develop a routine. Settling to sleep may be assisted by soft music, a soft toy and a gentle night light.
- Take the child into the bedroom while still awake.
- A sleep diary can be a useful tool.

Sedative medication has a minimal place in the management of sleep disturbances,[5] and is not recommended for children <2 years although the judicious use of a sedative/hypnotic for a short term may break the sleepless cycle. Such drugs include promethazine 0.5 mg/kg (max. 10 mg) and trimeprazine (Vallergan) 1–2 mg/kg per dose (not for infants under 6 months).[17]

Parasomnias (sleep terrors, sleep walking and sleep talking)[2]

These are not true sleep disorders or night-time arousals. They occur in deep non-REM sleep. With sleep terrors, which usually develop within 2 hours of sleep and last 1–2 minutes, the child is usually inconsolable and has no memory of the event. These events cluster in age ranges:

- sleep terrors 4–8 years
- sleep walking 8–12 years
- sleep talking 6–10 years
- nightmares 3–6 years

They are self-limiting over a period of months. Usually, no active treatment is needed but for persistent, severe problems a 6-week trial of phenytoin, diazepam or imipramine is worthwhile.

Sleep problems in older people

The elderly constitute the bulk of long-term users of hypnotics and benzodiazepines. Two key issues to consider are the benefits of sleep and the risk of confusion. Problems associated with long-term benzodiazepine use are dependence, confusion, memory impairment and falls.

A study of older people with insomnia showed that:[19]

- 25% had insomnia either coexisting with or related to other sleep disorders, such as sleep apnoea or periodic limb movement disorder
- 10% had insomnia related to medical or psychiatric conditions
- 13% had insomnia associated with an inability to stop taking sedative-hypnotic agents

Principles of management in older people

- Exclude underlying causes of sleep disturbance.
- Educate older people and their carers about the changing needs with ageing and the rational use of medicines.
- Avoid hypnotics if possible.
- Avoid hypnotics combined with alcohol.
- Beware of risks of long-term use and drug accumulation.
- Consider non-drug measures where possible (e.g. CBT).
- Avoid the ‘why bother changing’ factor when carers and patients are comfortable with hypnotics.

- In nursing homes, practise ‘bothering’ and use team effort to encourage less prescribing.

Medication[5]

If medication is necessary, prescribe a short-acting benzodiazepine for as limited a time as possible. An alternative non-benzodiazepine hypnotic (e.g. zopiclone or zolpidem) taken just before retiring may be useful in the elderly (beware of adverse effects). Consider melatonin prolonged release medication. Tricyclic antidepressants with a sedative action are frequently used, especially if there is coexistent depression, but side effects limit their use. However, commonsense prescribing needs to be used and the best option may be to continue the long-term prescribing of hypnotics in those with chronic medical problems or long-term dependence on a low effective dose.

Patient education resources

Hand-out sheets from *Murtagh’s Patient Education* 7th edition:

- Sleep problems: insomnia
- Restless legs syndrome
- Sleep problems in children

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61 Sore mouth and tongue

The key to managing diseases of the oral mucosa is to evaluate lifestyle factors carefully, including causes of immune suppression.

DR JONATHAN TVERSKY 2002

Evaluating the sore mouth and tongue is basically an understanding of disorders of the oral mucosa. Disorders of the oral mucosa are a common problem in general practice, with recurrent aphthous ulceration being the most common oral mucosal disease in humans.

There are three types of stratified squamous epithelium in the oral mucosa:[1]

1. masticatory—surface layer, cornified (orthokeratinised), attached to underlying periosteum (e.g. hard palate and gingivae)
2. lining—(e.g. lip and buccal mucosa, alveolar mucosa, floor of mouth, soft palate and tongue—lateral and undersurface)
3. specialised—with taste buds and papillae, e.g. on dorsum of tongue

Red flag pointers for oral conditions

- Dehydration in children with herpetic gingivostomatitis
- Petechiae on soft palate with gingivostomatitis or pharyngotonsillitis
- Oral ulcers and skin disorders
- Oral ulcers (especially solitary) or soft tissue lesions persisting for >3 weeks
- Oral ulcers and bowel dysfunction
- Oral candidiasis (may indicate diabetes or other immunosuppression)
- Glossodynia may indicate psychological disorder (e.g. depression)

Oral ulceration

The histology of oral ulceration is usually non-specific, with fibrin slough covering granulation tissue, and the aetiology is varied. The ulceration is a breach in the epithelial compartment with inflammatory cell infiltrate in the submucosa. The most common form is recurrent aphthous ulceration. Always inquire about a history of skin problems, medication, bowel function and psychological stress.

Key facts and checkpoints

- Dental trauma or neglect is an important cause of many oral mucosal disorders, such as ulceration, bleeding gums and hyperplasia.
- Non-healing oral ulcers warrant biopsy to exclude squamous cell carcinoma (SCC).
- If oral mucosal cancer is suspected, palpate the lesions to check for induration or a firm, discrete edge and check regional nodes.
- Any oral ulcer or soft tissue lesion that persists 3 weeks after the apparent cause has been removed should be biopsied. The initial ‘cause’ (e.g. tooth biting lip) may be just the trigger for noticing the lesion.
- Consider Epstein–Barr virus (EBV) infection with unusual faucial ulceration and petechial haemorrhages of the soft palate.
- Aphthous ulcers are usually 3–5 mm in diameter—minor ones have an erythematous margin.
- Intraoral bony exostoses, other than palatal and mandibular tori, are often variations of normal, or less commonly part of a syndrome, e.g. Gardner syndrome. No treatment is usually required.[2]
- Shared care of more complex lesions of the mouth and tongue with an oral or dental surgeon is best practice.

A list of causes is presented in the diagnostic strategy model (see TABLE 61.1). Depending on the clinical picture, investigations may include FBE, swabs, autoantibody screen, syphilis serology, blood sugar, vitamin B12 and folate levels, and biopsy.

Table 61.1 Mouth ulcers: diagnostic strategy model

Probability diagnosis

Recurrent aphthous ulceration
Trauma, e.g. sharp broken tooth
Acute herpes simplex infection
Candidiasis

Serious disorders not to be missed

Cancer: SCC, leukaemia
Agranulocytosis
Severe herpetic gingivostomatitis
HIV
Syphilitic—chancre or gumma
Tuberculosis

Pitfalls (often missed)

Aspirin burn
Inflammatory bowel disease (e.g. Crohn)
Herpes zoster virus
Glandular fever (EBV)
Lichen planus
Coxsackie virus:

- herpangina
- hand, foot and mouth disease

Immunosuppression therapy
Lupus erythematosus
Coeliac disease
Rarities:

- Behçet syndrome
- pemphigoid and pemphigus vulgaris
- erythema multiforme
- radiation mucositis

Seven masquerades checklist

Diabetes (*Candida*)
Drugs
Anaemia (iron-deficiency)

Is the patient trying to tell me something?

Unlikely.

Recurrent aphthous ulceration

Aphthous ulcers are round/oval ulcers usually 3–5 mm in diameter with an erythematous margin and sloughing base.

Aphthous ulcers occur in all ages on un(para)-keratinised mucosa such as the buccal and labial mucosa and the floor of the mouth (not on orthokeratinised mucosa) (see FIG. 61.1). The lifetime frequency of aphthous ulcers in the population is perhaps 20%. The cause is unknown, although human herpes virus 6 has been implicated, as have nutritional and autoimmune factors.[3] People with recurrent aphthous ulcers have a genetic predisposition.

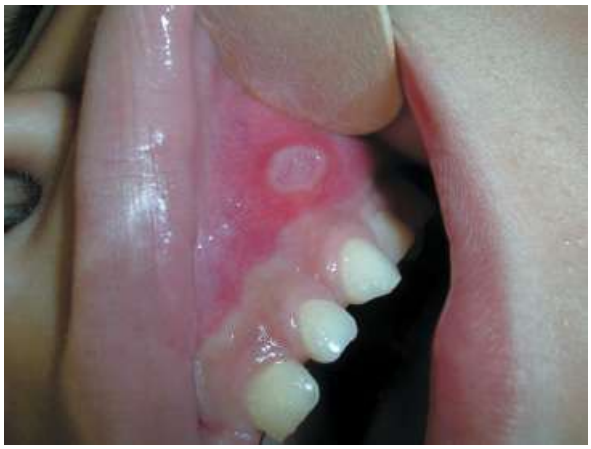

FIGURE 61.1 Apthous ulcer located on unkeratinised (movable) mucosa in a 5-year-old girl

Precipitating factors

- Trauma (e.g. cheek and tongue biting, toothbrush, dental pressure)
- Drug reaction (e.g. new medication)
- Stress
- Allergy
- Systemic factors (e.g. iron, folate, vitamin B12 deficiency, hormonal)

Note: Exclude blood dyscrasias, Crohn disease, Behçet syndrome, coeliac disease, drug therapy (e.g. phenytoin, cytotoxics, corticosteroids, immunosuppressants).

Rules

- Minor ulcer <5 mm in diameter: lasts 5–10 days and heals without scarring
- Major ulcer >8 mm: can persist for up to 6 weeks[3]
- Major ulcers usually occur on lips, soft palate and fauces and sometimes on the tongue
- Minor ulcers are usually found on the buccal and labial mucosa and the floor of the mouth
- Non-healing ulcers: consider SCC (biopsy required)
- Recurrent ulcers: consider Behçet syndrome. Check serum iron and folate

DxT recurrent oral and genital ulcers + uveitis + arthritis → Behçet syndrome

Treatment

There are multiple methods but none are specific.

Symptomatic relief

topical lignocaine (e.g. 2% jelly or 5% ointment) with cotton bud: after 2 minutes apply lignocaine gel or paint (e.g. SM-33 adult paint formula or SM-33 gel children) every 3 hours

or

eutectic EMLA cream 5%—apply on a cotton bud or gauze for 5 minutes

Healing: options[4]

- Triamcinolone 0.1% (Kenalog in Orabase) paste, apply three times daily after meals and nocte (preferred method but be careful of herpes simplex ulcers).
- Other topical steroids (e.g. betamethasone 0.05% ointment, hydrocortisone 1%). Apply topically to lesion bd after meals.
- Hydrocortisone lozenges (if available)—dissolve in contact with ulcer, qid.
- Beclomethasone dipropionate 50 mcg spray onto ulcer tds.

All of the above treatments have been shown to be effective in controlled trials.

Major ulceration

Consider:

injection of steroids into the base of the ulcer

and/or

oral prednisolone 25 mg daily, 5–7 days[4]

Referral: Patients with a non-healing ulcer within 3 weeks of presentation.

Complementary measures

1. *Teabag method.* Consider applying a wet, squeezed-out, black teabag directly to the ulcer regularly (the tannic acid promotes healing). Must be used when ulcer is worse.
2. *Melaleuca (tea-tree) oil.* 1% tea-tree oil used as a mouth rinse for 1 minute has been shown to prevent secondary infection.[5]

Traumatic ulceration

- This is commonly caused by sporting injuries, biting of the cheek or lip, and hot food.
- Factitious causes include scratching of an ‘itchy’ mouth or over-brushing of the teeth.
- Other relevant causes include dentures, sharp tooth surfaces, orthodontic bands and sharp objects such as pencils and hard food.
- Aspirin ‘burns’ are caused by people leaving salicylate-based tablets to dissolve against oral mucosa.
- Iatrogenic causes include surgical procedures such as intubation and endoscopy, and dental treatment such as retractors and removing dry cotton rolls.

Management[1]

- Explanation, including removal of the cause.
- Warm salt-water mouthwashes, and/or local anaesthetic mouthwashes:

  benzocaine compound (Cepacaine), swirl in mouth 10–15 mL for 10–15 seconds and expel, every 3 hours prn

  or

  benzydamine hydrochloride (Difflam), swirl in mouth 15 mL for 30 seconds and expel

These ulcers can take up to 10 days to resolve.

Lichenoid drug reaction

Several drugs can induce a lichenoid drug reaction of the oral mucosa; that is, cause shallow mucosal erosions similar to lichen planus. The drugs include gold, the NSAIDs, carbimazole, selected antihypertensives and cytotoxics.

Herpes infection

- Herpes simplex virus is a fairly common cause of oral lesions.
- Primary herpetic gingivostomatitis is usually obvious but herpes infection has an extraordinary ability to present in many ways. It can spread from the hands to the mouth.
- Application of a topical corticosteroid, such as Kenalog in Orabase, can aggravate and spread the herpetic lesion.
- Treatment: aciclovir or similar antiviral if seen early, e.g. 48 hours from onset; fluids + +; analgesic mouth rinses, e.g. Difflam; consider admission for IV aciclovir and hydration.
- Lesions of herpes zoster virus affecting the maxillary division of the trigeminal nerve, for example, can involve the buccal mucosa in a unilateral pattern.

Red patches

A reduction in the surface epithelial layer causes erythematous patches. Causes include trauma (e.g. cheek biting), infection (e.g. *Candida albicans*), geographic tongue, haematologic disorders, the dermatoses and neoplasia.

Neoplasia that can look red includes squamous cell carcinoma, Kaposi sarcoma and erythroplakia. Erythroplakia is similar in significance to leukoplakia except for the erythematous feature. It is an important condition to recognise since about 90% of cases are either dysplastic or cancer.[6]

White patches

White patches occur where the epithelial compartment thickens. Causes include inflammation due to trauma or infection, especially *Candida*, dermatoses and neoplasia.

An interesting condition is hyperkeratotic burns on the dependent floor of the mouth, which appear white. Causes include tea-tree oil mouthwash and the sucking of aspirin.

Leukoplakia is any white lesion that cannot be removed by rubbing the mucosal surface (unlike oral candidiasis). About 5% of cases represent either dysplasia or early SCC.[6] Any persistent white patch should be biopsied (see FIG. 61.2).

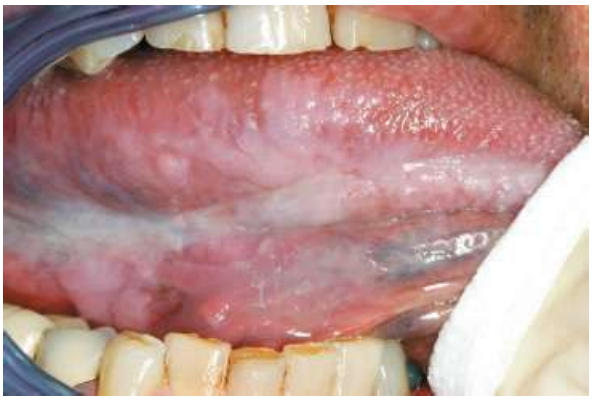

FIGURE 61.2 Leukoplakia showing white patch below tongue

Specific conditions causing red and/or white patches follow.

Oral candidiasis (thrush)

This is usually tender and looks like white or yellowish curd-like patches overlying erythematous mucosa. Unlike lichen planus or leukoplakia, they are usually readily rubbed off, after which only the underlying red patch may be seen.

Patients may also complain of a bad metallic taste or halitosis and dysphagia. They often complain of sensitivity to toothpaste or acidic substances in general.

Consider predisposing factors:

- immunodeficiency and cytotoxic therapy
- medication, especially broad-spectrum antibiotics and corticosteroids, including inhalers
- debility and anaemia (iron, folic acid, vitamin B6 deficiency)
- diabetes mellitus and HIV infection

The carriage rate of *Candida albicans* in the oral cavity is 60–75%. The diagnosis is made clinically but a wet preparation using potassium hydroxide will reveal spores and perhaps mycelia.

Treatment

Attend to underlying cause. Consider dental hygiene multivitamin preparations. Antifungal agents should not be used until the fungal infection is confirmed.[5]

Topical therapy

nystatin 100 000 U/mL suspension, rinse and swallow qid after food: place 1 mL under the tongue then swallow

or

miconazole oral 2% gel (as directed by manufacturer)

or

amphotericin 10 mg or nystatin 100 000 U lozenges dissolved slowly in oral cavity, 6 hourly, for 7–14 days. Avoid with a severe dry mouth.

Oral therapy

Use if unresponsive to topical therapy and the immunocompromised:[5]

fluconazole 50 mg (o) daily for 7–14 days

Denture therapy[1]

Dentures need to be decontaminated, especially if acrylic. Use:

- chlorhexidine denture scrub (care with bleaching), or
- dilute Milton's denture scrub (e.g. ¼ teaspoon White King in a cup of water)

Keep dentures in a dry place at night when not being worn.

If oral thrush, brush the dentures each night with a thin coat of nystatin cream or oral miconazole.

Angular cheilitis

Feature is redness, soreness and maceration of the corners of the mouth. Usually associated with oral candidiasis. Consider poor-fitting dentures, diet—vitamin B deficiency, iron deficiency and atopic or seborrhoeic dermatitis. Treat with topical nystatin or miconazole. 'Golden' crusting indicates *S. aureus.*

Bleeding or painful gums

Erythematous bleeding gums are a common worldwide problem, which is almost always a localised inflammation associated with poor dental hygiene.[7] Systemic problems usually as part of a bleeding diathesis need to be considered.

The causes are summarised in the diagnostic strategy model (see TABLE 61.2).

Table 61.2 Bleeding/painful gums: diagnostic strategy model (modified)

Probability diagnosis
Gingivitis/periodontal (gum) disease
Trauma: poor-fitting or partial dentures
Factitious: excessive brushing
Drugs: warfarin
Serious disorders not to be missed
Oral cancer/benign neoplasms (e.g. epulis)
Blood dyscrasias (e.g. AML)
Acute herpetic gingivostomatitis
Pitfalls (often missed) but uncommon
Acute ulcerative gingivitis (Vincent infection, trench mouth)
Autoimmune disease (e.g. lichen planus, SLE)
Hereditary haemorrhagic telangiectasia
Malabsorption
Scurvy

Acute necrotising ulcerative gingivitis (Vincent infection or trench mouth) caused by anaerobic organisms is rarely seen but is more common in undernourished or ill young adults under stress.

Gingivitis

Caused by plaque (bacterial biofilm) with calculus (tartar secondary to poor oral hygiene).

Features

- Red, swollen gingivae adjacent to teeth (see FIG. 61.3)
- Bleeds with gentle probing
- Halitosis
- Usually no pain

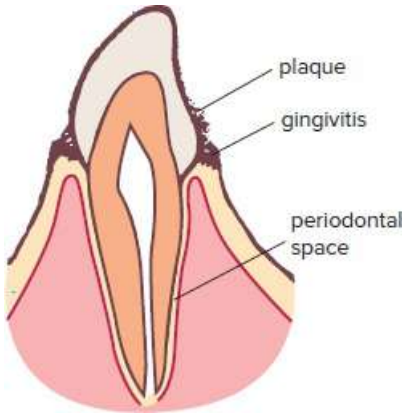

FIGURE 61.3 Gingivitis, showing plaque and gingivitis

Treatment[4,7]

- Dental care—remove plaque and clean
- Cease smoking
- Mouthwash: chlorhexidine 0.2 or 0.12% aqueous solution 10 ml for 1 minute 8–12 hourly for 10 days or until pain abates (beware of superficial discolouration of teeth with prolonged use)

Acute ulcerative gingivitis

This is a very painful form of gingivitis. Treatment is as for gingivitis but add antibiotics, e.g. metronidozole 400 mg (o) 12 hourly or tinidazole 2 g (o) single dose and drain pus from any associated abscesses.[4]

Periodontitis

This is inflammation of the periodontal space. It is a sequel to gingivitis and shows periodontal ligament breakdown with recession or periodontal pocketing and alveolar bone loss. There is possible loosening of teeth and periodontal abscess formation (see FIG. 61.4). An underlying medical condition must be suspected.

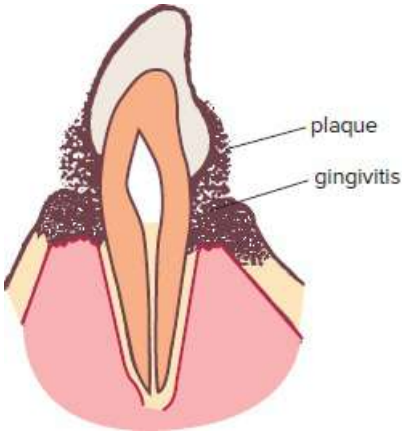

FIGURE 61.4 Periodontitis with more widespread gingival inflammation that invades the supporting alveolar bone

Risk factors are smoking and diabetes.

Treatment is meticulous dental treatment and mouthwashes. Antibiotics are rarely required.[4]

Prevention of gingival disease

- Use a fluoride, abrasive type of toothpaste.
- Brush with a medium-to-soft, nylon-tufted, small-headed toothbrush.
- Direct brush at gingival margin with a small horizontal motion.

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- Keep interdental spaces clean with dental flossing in a vertical direction or tooth picking.
- Regular dental review—eliminate plaque.

Oral dermatoses

The dermatoses include lichen planus, pemphigus vulgaris (uncommon), mucous membrane pemphigoid (uncommon) and lupus erythematosus. The clinical appearance of these conditions in the mouth is quite different to the skin condition because of the environment, especially due to the presence of saliva.

Diagnosis

Histopathological examination (after appropriate biopsy) with immunofluorescence is recommended, especially because of the similarity of the lesions of lupus erythematosus and lichen planus, which are both considered to be potentially premalignant in the mouth.[8]

Clinical features

Lichen planus:

- affects 2% of the population, usually over 45 years
- can vary from asymptomatic to severely painful
- usually white lace-like patterns on mucosa, cheeks and tongue
- may form superficial erosions

Lupus erythematosus:

- oral lesions may be first sign of SLE
- usually on lateral aspects of the hard palate
- can resemble lichen planus

Treatment

Consider specialist referral.

Oral hygiene and pain control:

chlorhexidine mouthwash

or

tetracycline/nystatin mouthwash

or

topical analgesics (e.g. lignocaine preparation)

Corticosteroids:

- topical (e.g. Kenalog in Orabase; betamethasone dipropionate 0.05%)
- intralesional (e.g. triamcinolone 10 mg/mL, especially for lichen planus)
- systemic—may be necessary in severe cases

The painful tongue[9]

Pain in the tongue is a reasonably common symptom in general practice. The cause is usually obvious upon examination, but there are some obscure causes. As for many other oral mucosal problems, shared care with a dental or oral medical specialist is important. The causes of a sore or painful tongue are similar to that of the sore throat or mouth. Xerostomia is common in the

elderly.

Investigations may include an FBE, serum vitamin B12, folate and ferritin levels, a swab or a biopsy of a suspicious lesion.

A diagnostic strategy with lists of causes is presented in TABLE 61.3 .

Table 61.3 Sore tongue: diagnostic strategy model

Probability diagnosis
Geographic tongue
Atrophic glossitis
Trauma (bites, teeth, hot food/drink)
Aphthous ulceration
Herpes simplex virus (children)
Fissured tongue
Disorders not to be missed
Cancer
Agranulocytosis (?drug induced)
HIV
Pitfalls (often missed)
Anaemia: iron, vitamins B6 and B12, folate deficiency
Citric acid-containing foods
Glossopharyngeal neuralgia
Lichen planus
Fissured tongue (rarely causes soreness)
Median rhomboid glossitis
Behçet syndrome
Crohn disease
Coeliac disease
Seven masquerades checklist
Depression
Diabetes (*Candida*)
Drugs (mouthwashes, aspirin)
Anaemia (various)
Is the patient trying to tell me something?

Possible with glossodynia.

Tongue tips

- Look for evidence of trauma, especially from a sharp tooth.
- A miserable child with a painful mouth and tongue is likely to have acute primary herpetic gingivostomatitis or hand, foot and mouth disease.
- When taking the history, take note of self-medications, especially sucking aspirin, a history of skin lesions (e.g. lichen planus) and consider underlying diabetes or immunosuppression.

- A long history of soreness with spicy or other foods indicates benign migratory glossitis (geographic tongue) or median rhomboid glossitis (see FIG. 61.5).
- Any non-healing or chronic ulcer requires urgent referral.
- Macroglossia (large tongue): consider acromegaly, myxoedema, amyloidosis, lymphangioma.
- Strawberry tongue: consider scarlet fever, Kawasaki disease.
- Glossodynia (painful tongue): characteristically presents as a burning pain on the tip of the tongue.[9] It can be a real ‘heartsink’ presentation. Consider depressive illness as an underlying cause.

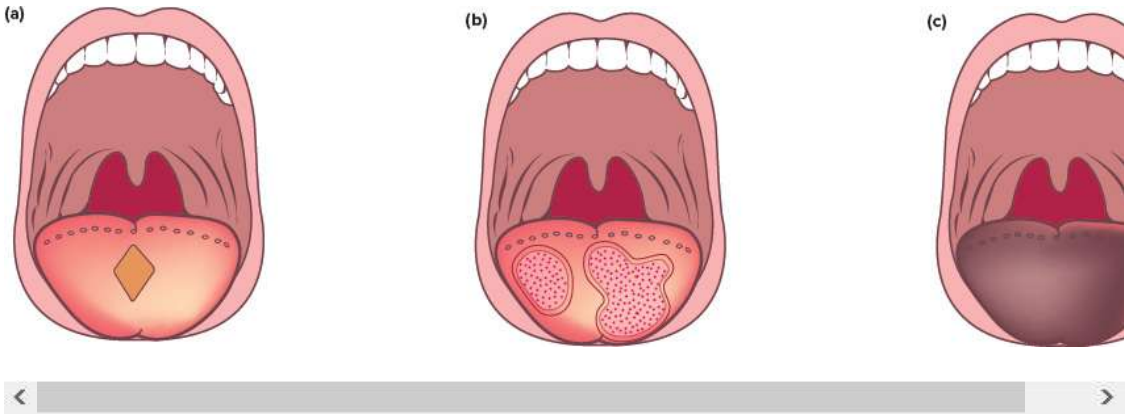

FIGURE 61.5 Disorders of the tongue: **(a)** median rhomboid glossitis, **(b)** geographic tongue, **(c)** black tongue

Erythema migrans (geographic tongue)

Also known as benign migratory glossitis, this benign condition shows changing patterns of desquamatous areas and erythema on the dorsum and edges of the tongue. With the smooth red

patches and raised whitish grey edges, the pattern resembles a relief map with mountain ridges. The border changes shape within weeks. It is irregular and slightly reddened. See FIG. 61.6 .

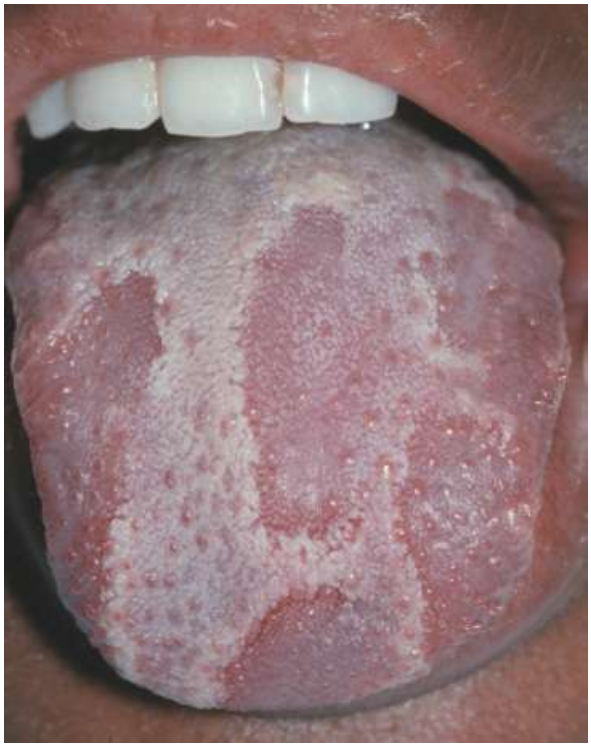

FIGURE 61.6 Geographic tongue (benign migratory glossitis). Note the pink continents among the white oceans.

(*Reproduced with permission from Gonsalves WC, Chi AC, Neville BW. Common oral lesions: Part I. Superficial mucosal lesions. Am Fam Physician, 2007; 75(4): 501–7.*)

It is considered to be a hypersensitivity reaction but the offending allergen has not been identified. Stress, tobacco, alcohol, marijuana and spicy foods can aggravate the condition in some individuals.

Management

- The condition is self-limiting and there is no specific therapy.
- Explanation and reassurance is important.
- No treatment is recommended if asymptomatic.
- If tender, benzocaine compound (Cepacaine) gargle 10 mL tds.
- If persistent and troublesome, low-dose inhaled glucocorticoid (e.g. beclomethasone 50 mcg tds—don’t rinse after use).

Black or hairy tongue

This is due to overgrowth of papillae or reduced wear of papillae, e.g. debility and lack of fibrous foods.

- Appearance: dark, elongated filiform papillae giving brownish appearance to dorsum (posterior) of tongue.
- Symptoms: bad tastes and malodorous oral cavity.

Causes

- Unknown
- Poor oral hygiene/debility
- Iatrogenic (e.g. antibiotics, major tranquillisers, corticosteroids)

Treatment

Brush or scrape tongue to remove stained papillae. Use a topical keratolytic agent such as salicylate, with pineapple being the most practical (95% cases are helped).

Method

- Cut a thin slice of pineapple into eight segments. Slowly suck a segment on the back of the tongue for 40 seconds and then slowly chew it. Repeat until all segments are completed. Do this bd for 7–10 days.[1] Repeat if recurs.

Note: The salicylate in pineapple can aggravate irritable bowel syndrome.

Consider sodium bicarbonate mouthwash.

Oral dysaesthesia ('burning mouth' syndrome)[5]

The classic chronic burning sensation of the oral cavity appears to have a neuropathic and/or psychological basis.[1] Symptoms include:

- altered sensitivity—burning pain or 'raw' sensation, mainly of the tongue and mucosa of lips
- altered taste—sweet, salty, bitter or metallic
- altered saliva (subjective)—quality and quantity
- altered tooth sensation (e.g. 'phantom tooth pain')

- dry mouth (xerostomia)

Consider the underlying cause:

- medications
- haematinic deficiency—iron, folate, vitamin B12
- autoimmune disorder (e.g. Sjögren syndrome)
- endocrine disorder (e.g. diabetes)
- psychological disorder

Management

- Detailed history with exclusion of organic causes
- Provision of education and understanding
- Promote lifestyle changes including stress management
- Consider specialist referral

Consider clonazepam 0.5–1 mg bd.

Dysguesia

This condition is a distortion of the sense of taste.

Causes include various drugs (notably the OTC andrographis-containing agents), various antibiotics, anticholinergics and antidepressives, common cold, COVID-19, smoking, postnasal drip and Sjögren syndrome.

Oral cancer

Cancer of the lip and oral cavity accounts for 2–3% of all newly diagnosed cancers in Australia.[10]

SCC is the most common malignancy of the oral cavity, accounting for 90% of cases. It has a 5-year survival rate of 65% without lymph node involvement and 50% with local node metastases.[11] Cancer of the lip is usually treated successfully by excisional biopsy but intraoral cancer has significant morbidity and mortality.[10]

Other malignancies include mucoepidermoid carcinoma, lymphoma, Kaposi sarcoma and malignant melanoma, which is usually found on the palate.

Predisposing or associated factors for SCC include tobacco and marijuana abuse, alcohol abuse, excessive sunlight and immune suppressive disorders such as HIV, lymphoma and various medications.

SCC is usually found as a chronic indurated ulcer on the ventral and lateral surfaces of the tongue followed by the floor of the mouth and buccal mucosa. It may present as a white patch or, more commonly, as a speckled white and red nodular patch or a red velvety patch.

The red patches of erythroplakia (in particular) and the white patches of leukoplakia may be premalignant or early invasive cancer and necessitate further investigation, particularly incisional biopsy.

Treatment for oral cancer is surgery ± radiotherapy and chemotherapy.

Benign intraoral swellings and tumours

Epulis

An epulis is a benign, localised gingival swelling. It is a very ancient term with no pathological significance, meaning a ‘tumour situated on the gum’. There are two distinct types —a fibrous epulis and giant cell epulis. An epulis emerges between two teeth from the periodontal membrane where there is usually dental decay or a site of irritation, such as a partial denture. It appears to be more common during pregnancy where the epulis has a more vascular appearance.

Treatment is usually by excision, with histological examination, curettage of the origin and extracting associated teeth. The ‘pregnancy’ epulis should be left for several weeks after childbirth before treatment.

Typical locations of intraoral tumours are shown in FIGURE 61.7 .

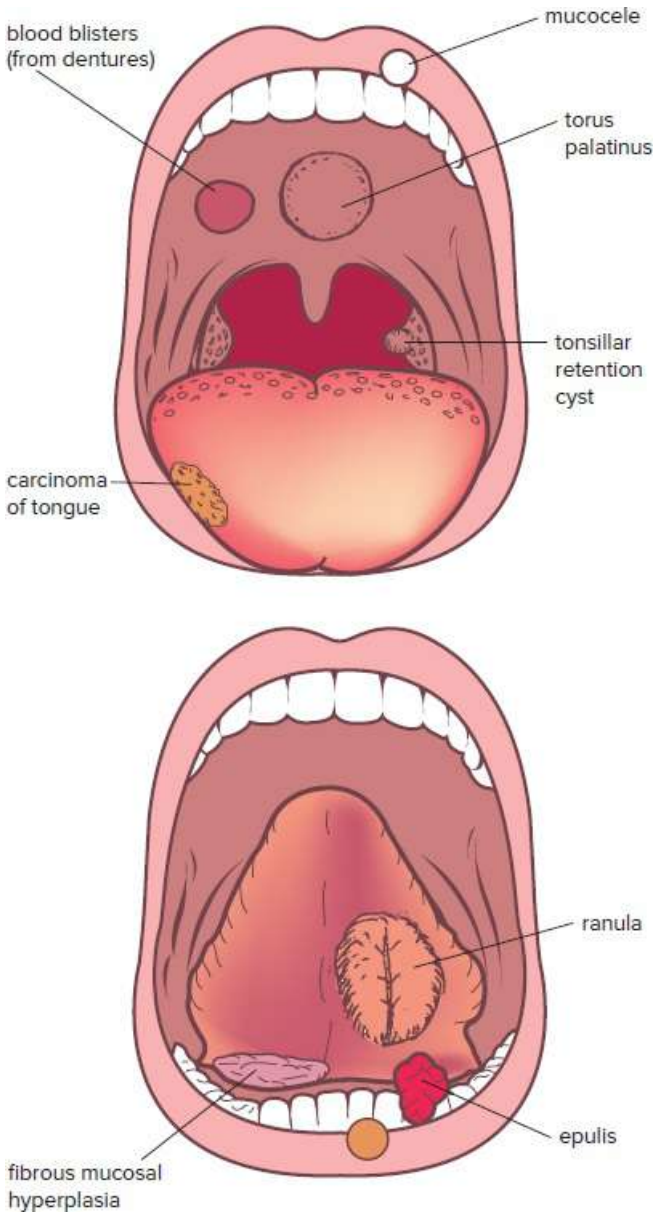

FIGURE 61.7 Typical location of swellings in the oral cavity

Pyogenic granuloma

These may occur on the gums or oral mucosa of the lips and look like pyogenic granulomas of the skin, which also are associated with minor trauma. They are best treated by excision.

Retention cysts (mucous cysts)

The oral mucosa contains numerous mucous cysts and accessory mucous and serous salivary glands.

Small retention cysts are probably caused by minor trauma to the duct. They may rupture spontaneously. They commonly occur on the mucosa of the lower lip. Treatment is by incision and enucleation under local anaesthesia. Larger ones require marsupialisation. Others occur on the tonsils where they usually appear as sessile yellow swellings. A special type of retention cyst is the ranula.

Ranula

A ranula is a large transparent mucocele occurring in the floor of the mouth. A blue colour and small tortuous veins stretched across the surface is typical.

It is usually unilateral and simple but may extend into the tissues of the floor of the mouth and neck (plunging ranula). Patients may give a history of a cyst that bursts and then returns.

Treatment is usually by marsupialisation.

Fibrous (fibroepithelial) hyperplasia

Hyperplasia of the oral mucosa, a very common condition, is usually seen on the floor of the mouth and is due to chronic irritation from ill-fitting dentures. Removal of the offending irritation is necessary. The hyperplasia may resolve, but if it doesn't, surgical removal of the residual mass is necessary.

Haemangioma

These appear as a dark blue/purple sessile or modular swelling anywhere in and around the mouth, especially on the vermilion border of the lips, floor of the mouth and tongue. They blanch on pressure. No treatment is needed except for pressing cosmetic reasons. Copious bleeding can occur with attempted removal.

Other soft tissue swellings

Swelling that may be encountered includes squamous papillomas (like viral warts), fibroepithelial polyps (inner side cheek), mixed salivary tumours, vascular haematomas and giant cell granulomas.

The most common benign intraoral salivary neoplasm is the pleomorphic adenoma, usually presenting as an asymptomatic swelling of the hard palate or cheeks.[2] Excision is recommended.

Bony exostoses

Bony outgrowths of the maxilla and mandible are reasonably common and the hard intraoral lump may cause concern. The most common is known as torus palatinus (see FIG 61.8), which is situated in the centre of the hard palate. A similar exostosis is torus mandibularis, which

occurs inside the mandible, opposite the premolar teeth and is usually bilateral. These lesions are hamartomas and do not require removal except if there is impending dental obstruction.

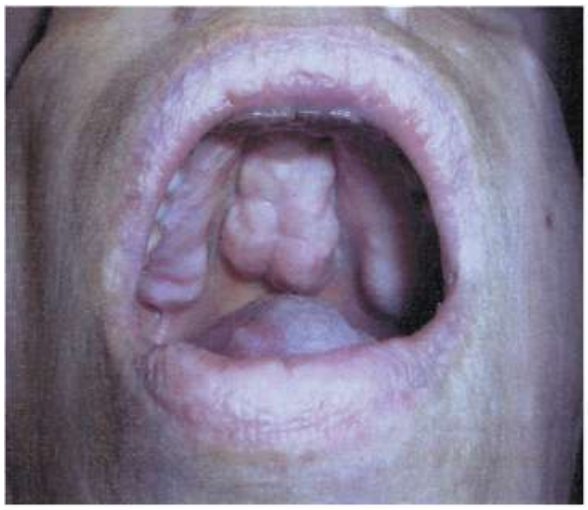

FIGURE 61.8 Torus palatinus in a woman aged 66 years. An incidental finding: it was asymptomatic.

Xerostomia (dry mouth)

This is a symptom rather than a disease entity. It occurs in about 10% of the population and approximately 70% of those have a systemic cause.[12]

The most common cause is a side effect of drug therapy and it is relative rather than absolute. Some patients who have a dry mouth on clinical examination do not complain of it while others who do complain of it may be found to have a normal salivary flow rate (can be a feature of depressive illness).

Other obvious causes are dehydration, mouth breathing and psychogenic.

Primary xerostomia

Causes

- Salivary gland atrophy due to ageing
- Salivary gland infections
- Autoimmune salivary gland disease (e.g. Sjögren syndrome)

DxT dry eyes + dry mouth + arthritis → Sjögren syndrome

Secondary xerostomia

Causes

- Mouth breathing
- Drugs: antidepressants (especially tricyclic agents), diuretics, anticholinergics, tranquillisers, antihistamines, anti-emetics, antihypertensives (some), antimigraine (some), antiparkinson, lithium and opioids
- Depression and anxiety (e.g. public speaking)
- Thirst/hunger
- Dehydration (e.g. diabetes, diarrhoea, kidney failure)
- Anaemias: iron, folate, vitamin B12 deficiency

Consequences[12]

Xerostomia interferes with speech, mastication and swallowing and causes difficulty in managing oral hygiene, especially dentures.

Symptoms include a burning sensation, a decrease in taste or a bad taste and fetid breath.

There is an increase in dental decay and perhaps a tendency to *Candida albicans* infection.

Treatment

This involves education, especially the need for meticulous oral hygiene, including topical fluoride preparations to the teeth and regular dental checks.

The cause must be diagnosed and treated if possible, especially a review of (and replacement of, if necessary) drug therapy.

Avoid decongestants and antihistamines.

Strategies to consider

- Suck ice or lozenges, sip sugarless fluids frequently and chew sugarless gum (avoid mouthwashes containing sugars and alcohol).
- Use a saliva substitute (e.g. Aquae, Saliva Orthana) or frequent mouthwashes (e.g. lemon and glycerin, 5–10 mL in 100 mL water as required—can be used in a plastic squeeze bottle).
- Use sodium fluoride 0.5% mouthwash for 5 minutes each day.

- Topical applications of glycerin or paraffin oil to the lips.

Halitosis[4]

Causes

The diagnostic strategy model for chronic halitosis (bad breath) is presented in CHAPTER 9 . The commonest causes are orodental disorders secondary to poor oral hygiene and inappropriate diet. Bacterial putrefaction of dental and food debris, together with inflammation of the gums, is largely responsible for the oral malodour. Smoking, alcohol and a dry mouth will aggravate the problem. (A 1999 survey showed that 87% of patients with halitosis had an oral cause, 8% an ear, nose and throat cause with 5% having other or unidentified causes.)

Red flags for halitosis

- Fever
- Purulent nasal discharge
- Purulent sputum
- Pathological oral lesions on inspection

Management

- First exclude dental disease, malignancy (esp. nasopharyngeal cancer), pulmonary TB, hairy tongue, nasal and sinus infection. Treat underlying disease.
- Refer for a dental check. Treat gingivitis.
- Consider drugs such as isosorbide dinitrate and various antidepressants as a cause.
- If a smoker—cease.
- Avoid or limit onions, garlic, peppers, curries, spicy salami and similar meats.
- Avoid or limit strong cheeses.
- Avoid excessive nips of alcohol.
- Brush teeth regularly during the day—immediately after meals.
- Gently brush the dorsum of the tongue with special, available soft brushes.

- Rinse mouth out with water after meals.
- Avoid fasting for long periods during the day.
- Drink copious amounts of water during the day.
- Chew sugarless gum to help moisten the mouth.
- Gargle with mouthwash regularly (e.g. Listerine; Cepacol Mint mouthwash; 0.2% aqueous chlorhexidine).
- Use dental floss regularly to clean the teeth.

Tip: use an oil/water wash (e.g. equal volumes of cetylpyridinium chloride (Cepacol) and olive oil), gargle a well-shaken mixture and spit out, qid.

Practice tips

- Recurrent herpes simplex ulceration is not common in the oral mucosa and if suspected should be confirmed by laboratory investigation. The treatment is different from aphthous ulceration so clinical distinction is important. Herpes simplex virus is aggravated by topical steroids.
- For unusual mouth ulceration, consider acute leukaemia, cancer, blood dyscrasias, Crohn disease and drug therapy such as anti-epileptics and antihypertensives.

Patient education resources

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

- Aphthous ulcers
- Halitosis
- Hand, foot and mouth disease
- Tongue soreness

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62 Sore throat

I believe there are hundreds of young adults who have erroneously suffered tonsillectomy because of the tonsillitis of undiagnosed glandular fever.

SCEPTICAL GP (ANONYMOUS)

URTIs are the most common symptomatic presentation in general practice (6% of encounters) so a sore or painful throat is one of the most common symptoms a GP will encounter.[1] The most usual cause is viral pharyngitis, which is self-limiting and usually only requires symptomatic treatment.

Glossary of terms

Pharyngitis Inflammation of pharynx ± tonsils.

Quinsy A peritonsillar abscess.

Tonsillitis Inflammation of tonsils only.

Key facts and checkpoints

- In a National Morbidity Survey (UK),[2] nine episodes per annum of acute pharyngitis or acute tonsillitis were diagnosed for every 100 patients.
- Sore throats account for about 5% of consultations in general practice per annum.[3]
- Although throat infections are common from infancy, children under 4 years of age rarely complain of a sore throat.
- Complaints of a sore throat are prevalent in children between 4 and 8 years and in teenagers.
- Sore throats continue to be common up to the age of 45 and then decline

significantly.

- The common causes are viral pharyngitis (approximately 70+%) and tonsillitis due to *Streptococcus pyogenes* (approximately 20%).
- The sore throat may be the presentation of serious and hidden systemic diseases, such as blood dyscrasias, HIV infection and diabetes (due to candidiasis).
- An important cause is tonsillitis caused by Epstein–Barr mononucleosis (EBM). Treating this cause with penicillin can produce adverse effects.
- As a general rule, antibiotics should not be prescribed to treat a sore throat, excluding evidence of group A beta-haemolytic *Streptococcus* (GABHS) infection.[4]

Presentation

Sore throat may be present as part of a complex of the common upper respiratory infections, such as the common cold and influenza. However, sore throat often presents as a single symptom. The pain is usually continuous and aggravated by swallowing. In those under 4 years of age the presentation of acute pharyngitis or tonsillitis may be confusing as the presenting complaints may be vomiting, abdominal pain and fever rather than sore throat and swallowing difficulty.

It is appropriate to consider sore throat as acute or chronic. Most presentations come as acute problems, the causes of which are listed in TABLE 62.1 .

Table 62.1 Causes of acute sore throat

Bacteria
Beta-haemolytic streptococci (GABHS)
Diphtheria (rare)
Gonococcal pharyngitis
Haemophilus influenzae
Moraxella catarrhalis
Quinsy
Staphylococcus aureus (rare)
Syphilis (rare)
Acute ulcerative gingivitis (Vincent angina infection)
Viral
Moderate–severe soreness

Epstein–Barr mononucleosis
Herpangina (Coxsackie A or B)
Herpes simplex pharyngitis
Mild–moderate soreness
Adenovirus
Cytomegalovirus
Coronavirus
Enterovirus
Influenza and parainfluenza virus
Respiratory syncytial virus (RSV)
Picornavirus
Rhinovirus
Human immunodeficiency virus
Varicella (chicken pox)

Other infections
Candida albicans, esp. in infants
Mycoplasma pneumoniae
Chlamydia pneumoniae

Blood dyscrasias
Agranulocytosis
Leukaemia

Irritants
Tobacco smoke
Antiseptic lozenges (oral use)

A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 62.2 .

Table 62.2 Sore throat: diagnostic strategy model

Probability diagnosis
Viral pharyngitis
Streptococcal (GABHS) tonsillitis
Chronic sinusitis with postnasal drip (often not ‘sore’)

Oropharyngeal candidiasis
Epstein–Barr mononucleosis

Serious disorders not to be missed

Cardiovascular:

- angina
- myocardial infarction

Neoplasia:

- cancer of oropharynx, tongue

Blood dyscrasias (e.g. agranulocytosis, acute leukaemia)
Severe infections:

- acute epiglottitis (children and adults)
- peritonsillar abscess (quinsy)
- pharyngeal abscess
- diphtheria (very rare)
- HIV/AIDS
- Vincent angina

Pitfalls (often missed)

Trauma—foreign body (e.g. fish bone)
Epstein–Barr mononucleosis
Candida:

- common in infants
- steroid inhalers

STIs:

- gonococcal pharyngitis
- herpes simplex (type II)
- syphilis

Irritants (e.g. cigarette smoke, chemicals)
Reflux oesophagitis → pharyngolaryngitis
Tonsilloliths
Cricopharyngeal spasm
Kawasaki disease
Angioedema (Quincke oedema)
Chronic mouth breathing
Aphthous ulceration
Thyroiditis
Glossopharyngeal neuralgia
Rarities:

- scleroderma
- Behçet disease
- sarcoidosis
- malignant granuloma
- tuberculosis

Seven masquerades checklist

Depression
Diabetes (*Candida*)
Drugs
Anaemia (possible)
Thyroid disorder (thyroiditis)
Spinal dysfunction (cervical)

Is the patient trying to tell me something?

Unlikely, but the association with depression is significant.

Probability diagnosis

The majority of sore throats, mainly pharyngitis, will be caused by a virus. A viral infection is supported by the presence of coryza prodromata, hoarseness, cough, conjuctivitis and nasal stuffiness. The common viruses include coronavirus, rhinovirus, RSV, parainfluenza, coxsackievirus, adenovirus and enterovirus.

Serious disorders not to be missed

It is vital to be aware of *Haemophilus influenzae* infection causing the sudden development of epiglottitis in children, especially between 2 and 4 years. These patients present with a short febrile illness, respiratory difficulty (cough is not a feature) and are unable to swallow.

It is important not to overlook cancer of the oropharynx or tongue, or the blood dyscrasias, including acute leukaemia (see CHAPTER 17). The severe infections not to be missed include streptococcal pharyngitis with its complications (quinsy, post-strep glomerulonephritis) and HIV infection (including AIDS).

A foreign body may stick in the supraglottic area and may not be seen on oral examination.

Pitfalls

There are many pitfalls, the classic being to diagnose the exudative tonsillitis of EBM as streptococcal tonsillitis and prescribe one of the penicillins, which may precipitate a severe rash.

Primary HIV infection can present with a sore throat along with other symptoms. Adenovirus pharyngitis can also mimic streptococcal pharyngitis, especially in young adults.

Traumatic episodes are important but are often not considered, especially in children. They include:

- a foreign body—may cause a sudden onset of throat pain, then drooling and dysphagia
- vocal abuse—excessive singing or shouting can cause a sore throat and hoarseness
- burns—hot food and drink, acids or alkalis

Red flag pointers for sore throat

- Persistent high fever
- Failed antibiotic treatment
- Medication-induced agranulocytosis
- Mouth drooling: consider epiglottitis (don't examine the throat)
- Stridor or other signs of respiratory distress
- Sharp pain on swallowing (?foreign body)
- Marked swelling of quinsy
- Candidiasis: consider diabetes or immunosuppression

Various irritants, especially cigarette smoke in the household and smoke inhalation from fires, can cause pharyngeal irritation with sore throat, especially in children.

The mouth and pharynx may become dry and sore from mouth breathing, which is often associated with nasal obstruction (e.g. adenoid hypertrophy, allergic rhinitis).

Tonsilloliths are concretions of debris entrapped within deep tonsillar crypts. They are a common cause of halitosis, vague sore throat and possibly recurrent bouts of tonsillitis.

Seven masquerades checklist

Depression may be associated with a sore throat. Diabetes and aplastic anaemia and drugs are indirectly associated through candidiasis, neutropenia and agranulocytosis respectively. NSAIDs can cause a sore throat. The possibility of thyroiditis presenting as a sore throat should be kept in mind.

Making a diagnosis

The issues of making a reliable diagnosis and prescribing antibiotics are rather contentious and at times difficult. It is difficult to distinguish clinically between bacterial and viral causes. The main issue is to determine whether the sore throat has a treatable cause by interpretation of the clinical and epidemiological data.

The appearance of the pharynx and tonsils is not always discriminating. A generalised red throat may be caused by a streptococcal or a viral infection, as may tonsils that are swollen with follicular exudates. On probability, most sore throats are caused by a virus and generally do not show marked inflammatory changes or purulent-looking exudates (see FIG. 62.1).

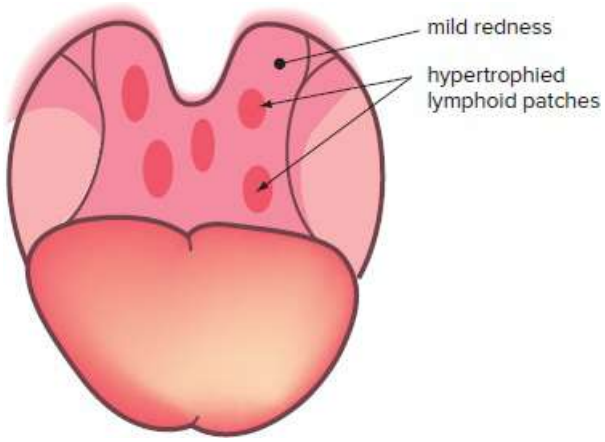

FIGURE 62.1 Viral pharyngitis: the signs may be minimal but mild redness of pharynx and prominent lymphoid patches on the oropharynx are typical

The presence of rhinorrhoea, conjunctivitis or a viral rash adds weight to the viral diagnosis. Viruses are considerably more likely to cause a dry cough and hoarse voice than bacterial causes, so the presence of these also helps discriminate. The majority of sore throats presenting to a general practice exhibit some of these features and should be treated symptomatically.

Reassuringly for this approach, only around 10% of sore throats in adults and 20% in children are caused by the most common bacterial pathogen (*Streptococcus pyogenes*) and even though this benefits from antibiotics, it is usually self-limiting; symptoms last approximately one day longer if no antibiotics are given.[5]

The clinical approach

History

It is necessary to determine whether the patient has a sore throat, a deep pain in the throat or neck pain. Instruct the patient to point to exactly where the pain is experienced. Enquire about relevant

associated symptoms such as metallic taste in mouth, fever, upper respiratory infection, other pain such as ear pain, nasal stuffiness or discharge and cough.

Note whether the patient has asthma and uses a corticosteroid inhaler, is a smoker or is exposed to environmental irritants. Check the immunisation history, enquiring especially about diphtheria.

The history should give a clue to the remote possibility that the painful throat is a manifestation of angina.

Examination

An inspection should note the general appearance of the patient, looking for ‘toxicity’, the anaemic pallor of leukaemia, the nasal stuffiness of infectious mononucleosis, the characteristic halitosis of a streptococcal throat.

Palpate the neck for soreness and lymphadenopathy, inspect the ears and check the sinus areas.

Then inspect the oral cavity and pharynx. Look for ulcers, abnormal masses and exudates. Note whether the uvula and soft palate, tonsils, fauces or pharynx are swollen, red or covered in exudate. The typical appearances of various conditions causing a sore throat are shown in FIGURES 62.1 to 62.7, and important causes to exclude in FIGURE 62.8.

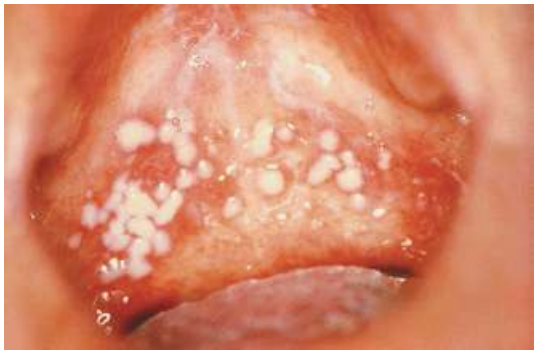

FIGURE 62.2 Oral thrush due to *Candida albicans* in a person with diabetes. Small patches of yellow–white exudate are seen on the palate, dorsum of the tongue, pharynx and mucosa.

Photo courtesy Hugh Newton-John