

- If a slit lamp is unavailable, the direct ophthalmoscope and a magnifying loupe can be used to view the cornea, but the standard blue light does not cause fluorescence; use a UV light instead.

## Microbial keratitis

This is responsible for at least 1.5 million new cases of blindness every year in the developing world and for significant morbidity in developed countries. It is a sight-threatening emergency.

### Risk factors

- Contact lens wear
- Corneal trauma, especially agriculture trauma
- Corneal surgery
- Postherpetic corneal lesion
- Dry eye
- Corneal anaesthetic
- Corneal exposure (e.g. VII nerve lesion)
- Ocular surface disease, including ulceration

*Pseudomonas aeruginosa* is the most common causative organism in contact lens wearers.

*Acanthamoeba* is associated with bathing or washing in contaminated water.

Urgent referral to an ophthalmologist or eye clinic is needed to avoid rapid corneal destruction with perforation, especially with bacterial keratitis. An appropriate ‘covering’ topical antibiotic is ciprofloxacin 0.3% or ofloxacin 0.3% eye drops.

## Problems with contact lenses

Because a contact lens is a foreign body, various complications can develop and a history of the use of contact lenses is important in the management of a red eye.

### Infection

Infection is more likely to occur with soft rather than hard lenses. They should not be worn when sleeping since this increases the risk of infection 10-fold.<sup>12</sup> One cause is *Acanthamoeba* keratitis acquired from contaminated water that may be used for cleaning the lenses.

## Hard lens trauma

This may cause corneal abrasions with irreversible endothelial changes or ptosis, especially with the older polymethyl-methacrylate-based lenses. Recommend patient should change to modern gas-permeable hard lenses.

## Lost lenses

Patients should be reassured that lenses cannot go behind the eye. The edge of the lens can usually be seen by everting the upper lid.

## Preventive measures<sup>13</sup>

- Wash hands before handling lenses.
- Do not use tap water or saline.
- Clean lenses with disinfecting solution.
- Store overnight in a clean airtight case with fresh disinfectant.
- Change the lens container solution daily.
- Discard disposable lenses after 2 weeks.
- Do not wear lenses while sleeping.
- Do not wear lenses while swimming in lakes, rivers or swimming pools.

Refer to an ophthalmologist if a painful red eye develops, especially if a discharge is present.

## Flash burns

A common problem, usually presenting at night, is bilateral painful eyes caused by UV ‘flash burns’ to both corneas some 5–10 hours previously. The mechanism of injury is UV rays from a welding machine causing superficial punctate keratitis. Other sources of UV light such as sunlamps and snow reflection can cause a reaction.

## Management

- Local anaesthetic (long-acting) drops: once-only application (do not allow the patient to take home more drops).
- Instil homatropine 2% drops statim or other short-acting ocular dilating agent (be careful of glaucoma) or plain tear lubricants.
- Use analgesics (e.g. codeine plus paracetamol) for 24 hours.

- If severe, use chloramphenicol eye ointment in lower fornix (to prevent infection).
- Use firm eye padding for 24 hours, when eyes reviewed (avoid light).

The eye usually heals completely in 48 hours. If not, check for a foreign body.

*Note:* Contact lens ‘overwear syndrome’ gives the same symptoms.

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## ⌚ Cavernous sinus arteriovenous fistula

Such a fistula produces conjunctival hyperaemia but no inflammation or discharge. The lesion causes raised orbital venous pressure. The fistula may be secondary to head injuries or may arise spontaneously, particularly in postmenopausal women. It needs radiological investigation.

The classic symptom is a ‘whooshing’ sound synchronous with the pulse behind the eye, and the sign is a bruit audible with the stethoscope placed over the orbit.

## ⌚ Penetrating eye injuries

These require urgent referral to an ophthalmologist. Do not remove any foreign body.

Consider:

- imaging: X-ray or CT scan
- tetanus prophylaxis
- transport by land (i.e. full atmospheric pressure)
- injection of anti-emetic (e.g. metoclopramide)

Use no ointment or eye drops, including local anaesthetic.

If significant delay is involved, give one dose (in adults) of:<sup>8</sup>

gentamicin 1.5 mg/kg IV plus

cefotaxime 1 g or ceftriaxone 1 g IV (can give ceftriaxone IM but with lignocaine 1%)

or

vancomycin IV + oral ciprofloxacin

## ⌚ Hyphaema

With hyphaema, a common blunt sporting injury, bleeding from the iris collects in the anterior chamber of the eye (see FIG. 40.15). The danger is that, with exertion, a secondary bleed from

the ruptured vessel could fill the anterior chamber with blood, blocking the escape of aqueous humour and causing a severe secondary glaucoma. Loss of the eye can occur with a severe haemorrhage. It is likely to happen 2–4 days after the injury.



**FIGURE 40.15** Hyphaemia of the eye showing blood in the anterior chamber; this occurred in a 29-year-old man who was struck in the eye by a squash ball

## Management

- First, exclude a penetrating injury.
- Avoid unnecessary movement: vibration will aggravate bleeding. (For this reason, do not use a helicopter if evacuation is necessary.)
- Avoid smoking and drinking alcohol.
- Do not give aspirin (can induce bleeding).
- Prescribe complete bed rest for 5 days and review the patient daily.
- Apply padding over the injured eye for 4 days.
- Administer sedatives as required.
- Beware of ‘floaters’, ‘flashes’ and field defects.

Arrange ophthalmic consultation after 1 month to exclude glaucoma and retinal detachment. No sport before this time.

## § Endophthalmitis<sup>14</sup>

This is an intra-ocular bacterial infection that may complicate any penetrating injury, including intra-ocular surgery. It should be considered in patients with such a history presenting with a red painful eye. Pus may be seen in the anterior chamber (hypopyon).

Urgent referral is mandatory. If significant delay, use ciprofloxacin (o) + vancomycin or gentamicin IV as single doses.<sup>3</sup>

## ⌚ Epiphora (watering eyes)

This has many causes and is more common in older people.

The main causes are drainage obstruction and excessive tear production, which includes physical and chemical irritants, blepharitis and entropion. Management depends on the person's age. Remove any mucoid discharge and massage the nasolacrimal sac.

Antibiotics are indicated only for conjunctivitis, blepharitis or dacryocystitis. Probing of the ducts or even surgery may be required.

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## When to refer

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- Uncertainty about the diagnosis
- Uveitis, acute glaucoma, episcleritis/scleritis or corneal ulceration
- Deep central corneal and intra-ocular foreign bodies
- Prolonged infections, with a poor or absent response to treatment or where therapy may be complicating management
- Infections or severe allergies with possible ocular complications
- Sudden swelling of an eyelid in a child with evidence of infection suggestive of orbital cellulitis—this is an emergency
- Emergency referral is also necessary for hyphaema, hypopyon, penetrating eye injury, acute glaucoma, severe chemical burn
- Herpes zoster ophthalmicus: if the external nose is involved then the internal eye may be involved
- Summary for urgent referral:
  - trauma (significant)/penetrating injury
  - hyphaema >3 mm
  - corneal ulcer
  - severe conjunctivitis

uveitis/acute iritis  
Behçet syndrome  
acute glaucoma  
giant cell arteritis  
orbital cellulitis (pre- and post-)  
acute dacryocystitis  
keratitis  
episcleritis/scleritis  
endophthalmitis  
herpes zoster ophthalmicus

*Note:* As a general rule never use corticosteroids or atropine in the eye before referral to an ophthalmologist.

## Practice tips

- Avoid long-term use of any medication, especially antibiotics (e.g. chloramphenicol: course for a maximum of 10 days).<sup>2</sup>

*Note:* Beware of allergy or toxicity to topical medications, especially antibiotics, as a cause of persistent symptoms.

- As a general rule, avoid using topical corticosteroids or combined corticosteroid/antibiotic preparations.
- Never use corticosteroids in the presence of a dendritic ulcer.
- To achieve effective results from eye ointment or drops, remove debris such as mucopurulent exudate with bacterial conjunctivitis or blepharitis by using a warm solution of saline (dissolve a teaspoon of kitchen salt in 500 mL of boiled water) to bathe away any discharge from conjunctiva, eyelashes and lids.
- A gritty sensation is common in conjunctivitis but the presence of a foreign body must be excluded.<sup>6</sup>
- Beware of the contact lens 'overwear syndrome', which is treated in a similar way to flash burns.

## Red eye golden rules

---

- Always test and record vision
- Beware of the unilateral red eye
- Conjunctivitis is almost always bilateral
- Irritated eyes are often dry
- Never use steroids if herpes simplex is suspected
- A penetrating eye injury is an emergency
- Consider an intra-ocular foreign body
- Beware of herpes zoster ophthalmicus if the nose is involved
- Irregular pupils: think iritis, injury and surgery
- Never pad a discharging eye
- Refer patients with eyelid ulcers
- If there is a corneal abrasion look for a foreign body

Source: Based on J Colvin and J Reich<sup>4</sup>

## Patient education resources

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

- Blepharitis
- Bloodshot eye
- Chalazion (meibomian cyst)
- Conjunctivitis
- Flash burns to the eyes
- Foreign body in the eye
- Styte

- Watering eyes

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# 41 Pain in the face

*It's as though the devil suddenly thrust red hot electric needles through my right cheek towards my ear.*

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PATIENT (ANONYMOUS), DESCRIBING 'TIC DOULOUREUX'

When someone complains of pain in the face rather than the head, the physician has to consider foremost the possibilities of dental disorders, sinus disease, especially of the maxillary sinuses, temporomandibular joint (TMJ) dysfunction, eye disorders, lesions of the oropharynx or posterior third of the tongue, trigeminal neuralgia and chronic paroxysmal hemicrania.

The key to the diagnosis is the clinical examination because even the most sophisticated investigation may provide no additional information.

A basic list of causes of facial pain is presented in TABLE 41.1 .<sup>1</sup> The causes can vary from the simple, such as aphthous ulcers, herpes simplex and dental caries, to serious causes, such as carcinoma of the tongue, sinuses and nasopharynx or osteomyelitis of the mandible or maxilla.

**Table 41.1** Diagnoses to consider in orofacial pain

## Positive physical signs

Cervical spinal dysfunction

Dental pathology

Erysipelas

Eye disorders

Herpes zoster

Nasopharyngeal cancer

### Oropharyngeal disorders:

- ulceration (aphthous, infective, traumatic, others)
- cancer
- gingivitis/stomatitis

- tonsillitis
- erosive lichen planus

Paranasal sinus disorders

Parotid gland:

- mumps
- sialectasis
- cancer
- pleomorphic adenoma

TMJ dysfunction

Temporal arteritis

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### Absent physical signs

Atypical facial pain

Chronic paroxysmal hemicrania

Depression-associated facial pain

Facial migraine (lower half headache)

Glossopharyngeal neuralgia

Migrainous neuralgia (cluster headache)

Trigeminal neuralgia (tic dououreux)

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## Key facts and checkpoints

- Dental disorders are the commonest cause of facial pain, accounting for up to 90% of pain in and about the face.<sup>2</sup>
- The most common dental disorders are dental caries and periodontal diseases.
- Dental pain is invariably localised to the dental region of the face.
- The mean age of onset of trigeminal neuralgia is 50 years.
- There is a similarity in the ‘occult’ causes of pain in the ear and in the face (refer to FIGS 39.4 and 39.5 ).
- Sinusitis occurs mainly as part of a generalised upper respiratory infection. Swimming is another common predisposing factor.
- Dental root infection must be sought in all cases of maxillary sinusitis.

# A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 41.2 .

**Table 41.2** Pain in the face: diagnostic strategy model

## Probability diagnosis

Dental pain:

- caries
- periapical abscess
- fractured tooth

Maxillary/frontal sinusitis

TMJ dysfunction

## Serious disorders not to be missed

Cardiovascular:

- myocardial ischaemia
- aneurysm of cavernous sinus
- internal carotid aneurysm
- ischaemia of posterior inferior cerebellar artery

Neoplasia:

- cancer: mouth, sinuses, nasopharynx, tonsils, tongue, larynx, salivary gland
- metastases: orbital, base of brain, bone

Severe infections:

- herpes zoster
- erysipelas
- periapical abscess → osteomyelitis
- acute sinusitis → spreading infection

Temporal arteritis

## Pitfalls (often missed)

TMJ dysfunction

Migraine variants:

- facial migraine
- chronic paroxysmal hemicrania

Atypical facial pain

Eye disorders:

- glaucoma

- iritis
- optic neuritis

Chronic dental neuralgia (odontalgia)

Salivary gland:

- infection, mumps, suppuration, calculus, obstruction, cancer

Acute glaucoma (upper face)

Cranial nerve neuralgias:

- trigeminal neuralgia
- glossopharyngeal neuralgia

### **Seven masquerades checklist**

Depression

Spinal dysfunction (cervical spondylosis)

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

Quite probably. Atypical facial pain has underlying psychogenic elements.

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## Probability diagnosis

The commonest cause of facial pain is dental disorders, especially dental caries. Another common cause is sinusitis, particularly maxillary sinusitis.

TMJ dysfunction causing TMJ arthralgia is a very common problem encountered in general practice and it is important to have some simple basic strategies to give the patient.

### **Red flag pointers for pain in the face**

- Persistent pain: no obvious cause
- Unexplained weight loss
- Trigeminal neuralgia: possible serious cause
- Herpes zoster involving nose
- Person >60 years: consider temporal arteritis, malignancy

## Serious disorders not to be missed

It is important not to overlook cancer of various structures, such as the mouth, sinuses, nasopharynx, tonsils, tongue, larynx and parotid gland, which can present with atypical chronic facial pain.

It is important therefore to inspect these areas, especially in the elderly, but lesions in the relatively inaccessible nasopharynx can be easily missed. Nasopharynx cancer spreads upwards to the base of the skull early and patients can present with multiple cranial nerve palsies before either pain or bloody nasal discharge.<sup>1</sup>

Tumours may arise in the bones of the orbit, for example, lymphoma or secondary cancer, and may cause facial pain and proptosis. Similarly, any space-occupying lesion or malignancy arising from the region of the orbit or base of the brain can cause facial pain by involvement (often destruction) of trigeminal sensory fibres. This will lead to a depressed ipsilateral corneal reflex.

Also, aneurysms developing in the cavernous sinus<sup>1</sup> can cause pain via pressure on any of the divisions of the trigeminal nerve, while aneurysms from the internal carotid arising from the origin of the posterior communicating artery can cause pressure on the oculomotor nerve.

Temporal arteritis typically causes pain over the temporal area but can cause ischaemic pain in the jaws when chewing.

## Pitfalls

Commonly overlooked causes of facial pain include TMJ arthralgia and dental disorders, especially of the teeth, which are tender to percussion, and oral ulceration. Diagnosing the uncommon migraine variants, particularly facial migraine and chronic paroxysmal hemicrania, often presents difficulties, including differentiating between the neuralgias. Glossopharyngeal neuralgia, which is rare, causes pain in the back of the throat, around the tonsils and adjacent fauces. The lightning quality of the pain of neuralgia gives the clue to diagnosis.

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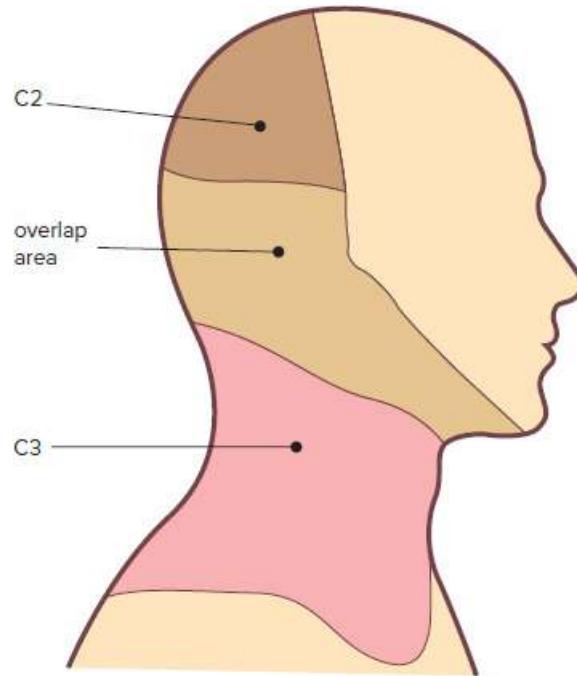
### Common pitfalls

- Failing to refer unusual or undiagnosed causes of facial pain
- Overlooking infective dental causes, which can cause complications
- Failing to consider the possibility of malignant disease of ‘hidden’ structures in the older patient
- Unaware that facial pain never crosses the midline

### Seven masquerades checklist

Of these, depression and cervical spinal dysfunction must be considered. The upper cervical spine can cause facial pain from lesions of C2 or C3 via the lesser occipital or greater auricular

(see FIG. 41.1 ) nerves, which may give pain around the ear. It is important to remember that the C2 and C3 nerves share a common pathway with the trigeminal nerve (see CHAPTER 51 ).



**FIGURE 41.1** Dermatomes of C2 and C3, with the overlap area indicated

Depressive illness can present with a variety of painful syndromes and facial pain is no exception. The features of depression may be apparent and thus antidepressants should be prescribed. Usually the facial pain and the depression subside concomitantly.

## Psychogenic considerations

Psychogenic factors have to be considered in every painful condition. They are considered to be high in patients with atypical facial pain.

## The clinical approach

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### History

Diagnosis of nearly all types of facial pain must be based almost entirely on the history. It is often difficult to delineate the exact nature and distribution of the pain. The history should include the typical analysis of pain, especially noting the site and radiation of the pain.

### Examination

Examination can often confirm the clinical impression given by the history—particularly palpation/percussion/pressure around the teeth, TMJs and sinuses.

The patient's general state and behaviour should be noted. Any sudden jabbing pain in the face causing the characteristic 'tic' may indicate neuralgia.

Palpate the face and neck to include the parotid glands, eyes, regional lymph nodes and the skin. Inspect and palpate the TMJs and cervical spine. Carefully inspect the nose, mouth, pharynx and postnasal space. In particular inspect the teeth, percussing each tooth if dental disorder is suspected. Bimanual palpation of the floor of the mouth is performed to detect induration or submandibular and submental lymph node enlargement.

The sinuses, especially the maxillary sinuses, should be inspected and a (hygienically protected) torch light can be placed inside the mouth to test transillumination of the maxillary sinuses. This works best when one symptomatic side can be compared with an asymptomatic side.

Perform a neurological examination on the cranial nerves with special emphasis on the trigeminal, oculomotor and glossopharyngeal nerves.

## Investigations

If investigations are being contemplated referral may be appropriate, particularly as fibre-optic nasopharyngoscopy is so commonly available as an ENT or maxillofacial 'office test'. The association of multiple sclerosis and tumours with neuralgias may have to be investigated. Radiological investigations include plain X-rays of the paranasal sinuses, CT scans, MRI and orthopantomograms.

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## Facial pain in children

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Apart from trauma, facial pain in children is almost invariably due to dental problems, rarely migraine variants and occasionally childhood infections such as mumps and gingivostomatitis. A serious problem sometimes seen in children is orbital cellulitis secondary to ethmoiditis.

Sinusitis occurs in children, especially older children, and it should be suspected with persistent pain and bilateral mucopurulent rhinorrhoea (beyond 10 days).

## Facial pain in the elderly

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Many of the causes of facial pain have an increased incidence with age, in particular trigeminal neuralgia, herpes zoster, cancer, glaucoma, TMJ dysfunction, sialolithiasis and cervical spondylosis. Glossopharyngeal neuralgia does not seem to have a particular predilection for the elderly. Xerostomia due to decreased secretions of salivary glands may cause abrasion with minor trauma. It may aggravate the pain of glossitis, which is common in the elderly.

# Dental disorders

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## Dental caries

Dental caries, impacted teeth, infected tooth sockets and dental roots can cause pain in the maxillary and mandibular regions. Caries with periapical and apical abscess formation produces pain from infection extending around the apex of the tooth into the alveolar bone. Retention of a fractured root may cause unilateral paroxysmal pain. Impacted third molars (wisdom teeth) may be associated with surrounding soft tissue inflammation (pericoronitis), causing pain that may be localised to the mandible or radiate via the auriculotemporal nerve to the ear. *Candida albicans*, which is an oral commensal, may colonise dentures causing hyperaemia and painful superficial ulceration of the denture-bearing mucosa. Antibiotics are of no proven use.

### Features of dental caries

- Pain is usually confined to the affected tooth but it may be diffuse.
- Pain is almost always aggravated by thermal changes in the mouth:
  - cold—if dental pulp vital
  - hot—if dental pulp is necrotic
- Pain may be felt in more than one tooth.
- Dental pain will not cross the midline.
- Antibiotics are not indicated.

### Treatment of dental pain

- Arrange urgent dental consultation

- Pain relief:<sup>3</sup>

aspirin 600 mg (o) 4–6 hourly

or

ibuprofen 400 mg (o) 4–6 hourly

or

paracetamol 0.5–1 g (o) 4–6 hourly

## Tooth abscess, inflamed wisdom tooth, spreading dental

## infection or root canal infection

Dental treatment will usually alleviate the problem; however, if severe:

metronidazole 400 mg (o) 12 hourly for 5 days

*plus either*

amoxicillin 500 mg (o) tds for 5 days

*or*

phenoxyxymethylpenicillin 500 mg (o) 6 hourly for 5 days

If unresponsive, or if single preparation preferred:

amoxicillin/clavulanate 875/125 mg (o) bd

(adjust all preparations for children's dosages)

For patients hypersensitive to penicillin:

clindamycin 300–450 mg (o) 8 hourly for 5 days

## Gingivitis and periodontitis

Refer to [CHAPTER 61](#).

## Alveolar osteitis (dry socket)

This complicates about 5% of tooth extractions and is due to failure of healing. Refer for toileting (socket irrigation with warm saline) and insertion of dressing to reduce pain. This usually heals naturally in 14–21 days. Antibiotics are of no proven use (see [FIG. 41.2](#) ).<sup>3,4</sup>



**FIGURE 41.2** Dry tooth socket: this is a very painful condition mainly in the lower molars, unrelieved by analgesics following a tooth extraction 1–3 days earlier. The socket has few or no blood clots and sensitive bone surfaces are covered by a greyish layer of necrotic tissue.

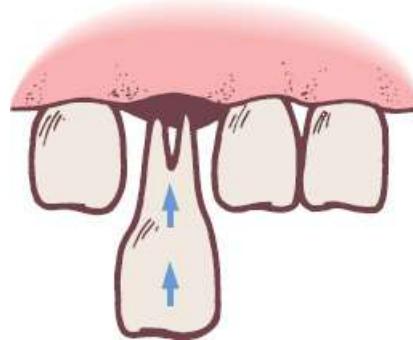
## Knocked-out or broken teeth

If a permanent (second) tooth is knocked out, it can be saved by immediate proper care. Likewise, a broken tooth should be saved and urgent dental attention sought.

### § The knocked-out tooth

- Using a glove, hold the tooth by its crown and place it in its original position, preferably immediately (see FIG. 41.3 ); if dirty, put it in milk or saline before replacement or, better still, place it under the tongue and ‘wash it’ in saliva. Do not use water, and do not wipe or touch the root.
- Fix the tooth by moulding strong aluminium foil over it and the adjacent teeth.
- Refer to a dentist or dental hospital as soon as possible.

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**FIGURE 41.3** Replacement of a knocked-out tooth

*Note:* Teeth replaced within half an hour have a 90% chance of successful re-implantation.

### § Ludwig angina<sup>4</sup>

This is a rapidly swelling cellulitis occurring in both the sublingual and submaxillary spaces without abscess formation, often arising from a root canal infection. It resembles an abscess and should be treated as one. It is potentially life-threatening as it can compromise the airway.

## Management

- Culture and sensitivity testing

- Specialist consultation
- Empirical treatment:  
amoxicillin 2 g IV, 6 hourly

*plus*

metronidazole 500 mg IV, 12 hourly

## Pain from paranasal sinuses

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Infection of the paranasal sinuses may cause localised pain. Localised tenderness and pain may be apparent with frontal or maxillary sinusitis. Sphenoidal or ethmoidal sinusitis causes a constant pain behind the eye or behind the nose, often accompanied by nasal blockage. Chronic infection of the sinuses may be extremely difficult to detect. The commonest organisms are *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis*.

Expanding lesions of the sinuses, such as mucocoeles and tumours, cause local swelling and displace the contents of the orbit—upwards for maxillary, laterally for the ethmoids and downwards for the frontal.

### Maxillary sinusitis

The maxillary sinus is the one most commonly infected.<sup>5</sup> It is important to determine whether the sinusitis is caused by stasis following a URTI or acute rhinitis, or due to dental root infection. Most episodes are of viral origin, and in the first few days this is indistinguishable from bacterial infections.<sup>6</sup>

#### Clinical features (acute sinusitis)

- Facial pain and tenderness (over sinuses)
- Toothache
- Headache
- Purulent postnasal drip
- Nasal discharge
- Nasal obstruction
- Rhinorrhoea
- Cough (worse at night)

- Prolonged fever
- Epistaxis

Suspect bacterial cause if high fever and purulent nasal discharge.

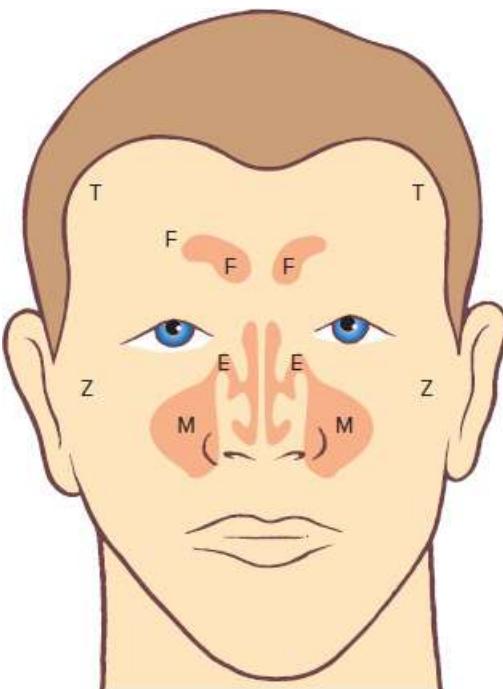
### Clinical features (chronic sinusitis)

- Vague facial pain
- Offensive postnasal drip
- Nasal obstruction
- Toothache
- Malaise
- Halitosis

## Some simple office tests

### Diagnosing sinus tenderness<sup>7</sup>

To differentiate sinus tenderness from non-sinus bone tenderness palpation is useful. Page 503  
This is best done by palpating a non-sinus area first and last (see FIG. 41.4 ), systematically exerting pressure over the temporal bones (T), then the frontal (F), ethmoid (E) and maxillary (M) sinuses, and finally zygomas (Z), or vice versa.



**FIGURE 41.4** Diagnosing sinus tenderness: T (temporal) and Z (zygoma) represent no sinus bony tenderness, for purposes of comparison

Differential tenderness both identifies and localises the main sites of infection (see FIG. 41.4 ).

### Diagnosing unilateral sinusitis

A simple way to assess the presence or absence of fluid in the frontal sinus, and in the maxillary sinus (in particular), is the use of transillumination. It works best when one symptomatic side can be compared with an asymptomatic side.

It is necessary to have the patient in a darkened room and to use a small, narrow-beam torch. For the maxillary sinuses remove dentures (if any). Shine the light inside the mouth (with lips sealed, and torch hygienically covered e.g. plastic bag), on either side of the hard palate, pointed at the base of the orbit. A dull glow seen below the orbit indicates that the antrum is air-filled. Diminished illumination on the symptomatic side indicates sinusitis.

In the first few days, viral and bacterial sinusitis are indistinguishable.

A CT scan may show mucosal thickening without fluid levels. Plain films are not indicated.

### Management (acute bacterial sinusitis)

#### Principles

- Exclude dental root infection.

- Control predisposing factors.
- Consider antibiotic therapy, but remember it is a self-limiting condition that has equal outcomes at day 10 with or without antibiotics.<sup>6</sup> The ‘number needed to harm’ with antibiotic side effects is unfavourable compared to the ‘number needed to treat’ with symptom resolution.
- Establish drainage by stimulation of mucociliary flow and relief of obstruction.

### **Guidelines for antibiotic therapy**

Consider therapy for severe cases that fail to improve over the first 5–7 days and display at least three of the following:

- persistent mucopurulent nasal discharge (>7–10 days)
- facial pain
- poor response to decongestants
- tenderness over the sinuses, especially maxillary
- tenderness on percussion of maxillary molar and premolar teeth that cannot be attributed to by a single tooth

### **Measures**

- Analgesics
- Antibiotics (if indicated):<sup>3</sup>
  - amoxicillin 500 mg (o) tds or 1 g (o) bd for 5 days
  - or (if sensitive to penicillin)
    - doxycycline 100 mg (o) bd for 5 days
    - or
    - cefuroxime 500 mg (o) tds for 5 days
    - or
    - amoxicillin + clavulanate 875/125 mg (o) tds for 5–10 days if poor response to above (indicates resistant *H. influenzae*)
- In complicated or severe disease, use intravenous cephalosporins or flucloxacillin

- Nasal decongestants (oxymetazoline-containing nasal drops or sprays)<sup>5</sup> for 5 days (only if congestion)
- Inhalations (an important adjunct)
- Nasal saline irrigation

Antihistamines and mucolytics are of no proven value. Cefuroxime is preferred to cephalexin or cefaclor because of superior anti-pneumococcal activity.<sup>3</sup>

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## Invasive methods

Surgical drainage may be necessary by atrial lavage or frontal sinus trephine.

## Inhalations for sinusitis

The old method of towel over the head and inhalation bowl can be used, but it is better to direct the vapour at the nose. Equipment needed is a container, which can be an old disposable bowl, a wide-mouthed bottle or tin, or a plastic container. Guard against accidental burns.

For the inhalant, several household over-the-counter preparations are suitable such as Friar's Balsam (5 mL), Vicks VapoRub (1 teaspoon) or menthol (5 mL).

The cover can be made from a paper bag (with its base cut out), a cone of paper or a small cardboard carton (with the corner cut away).

### Method

1. Add 5 mL or 1 teaspoon of the inhalant to 0.5 L (or 1 pint) of boiled water in the container.
2. Place the paper or carton over the container.
3. Get the patient to apply nose and mouth to the opening and breathe in the vapour deeply and slowly through the nose, and then out slowly through the mouth.
4. This should be performed for 5–10 minutes, three times a day, especially before retiring.

After inhalation, upper airway congestion can be relieved by autoinsufflation.

## Chronic sinusitis

Chronic sinusitis (>12 weeks) or recurrent sinusitis may arise from chronic infection or allergy. It may be associated with nasal polyps and vasomotor rhinitis, but is frequently associated with a structural abnormality of the upper airways. Refer to [CHAPTER 48](#).

It does not usually cause pain unless an acute infection intervenes. Initial measures are the same

as for allergic rhinitis;<sup>6</sup> use oral or intranasal antihistamine and add in an intranasal corticosteroid (see CHAPTER 72). Nasal saline irrigation is a useful addition or alternative. After one month, resistant cases (particularly those with nasal polyps) should be referred to a specialist. While waiting, a temporary trial of oral prednisolone 25 mg may be reasonable. Surgical intervention will benefit chronic recurrence with mechanical blockage.

## TMJ dysfunction

This condition is due to abnormal movement of the mandible, especially during chewing. The basic causes are dental malocclusion and masticatory muscle dysfunction. Check for bruxism. The pain is felt over the joint and tends to be localised to the region of the ear and mandibular condyle, but it may radiate forwards to the cheek and even the neck.

### Examination

- Check for pain and limitation of mandibular movements, especially on opening the mouth.
- Palpate about the joint bilaterally for tenderness, which typically lies immediately in front of the external auditory meatus; palpate the temporalis and masseter muscles.
- Palpate the TMJ over the lateral aspect of the joint disc.
- Ask the patient to open the mouth fully when tenderness is maximal. The TMJ can be palpated posteriorly by inserting the little finger into the external canal.
- Check for crepitus in mandibular movement.

### Management

If organic disease such as rheumatoid arthritis and obvious dental malocclusion is excluded, a special set of instructions or exercises can alleviate the annoying problem of TMJ arthralgia in about 3 weeks. Warm packs may help. Provide patient education advice and self-care.

#### Method 1: ‘Chewing’ the piece of soft wood

- Obtain a rod of soft wood approximately 15 cm long and 1.5 cm wide. An ideal object is a large carpenter’s pencil.
- Instruct the patient to position this at the back of the mouth so that the molars grasp the object with the mandible thrust forward.
- The patient then rhythmically bites on the object with a grinding movement for 2–3 minutes at least 3 times a day.

#### Method 2: The ‘six by six’ program

This is a specific program recommended by some dental surgeons. The six exercises should be

carried out six times on each occasion, six times a day, taking about 1–2 minutes.

Instruct the patient as follows:

1. Hold the front one-third of your tongue to the roof of your mouth and take six deep breaths. Page 505
2. Hold the tongue to the roof of your mouth and open your mouth six times. Your jaw should not click.
3. Hold your chin with both hands keeping the chin still. Without letting your chin move, push up, down and to each side. Remember, do not let your chin move.
4. Hold both hands behind your neck and pull chin in.
5. Push on upper lip so as to push head straight back.
6. Pull shoulders back as if to touch shoulder blades together.

These exercises should be pain-free. If they hurt, do not push them to the limit until pain eases.

## Treatments

### Injection into the TMJ<sup>8</sup>

- Indications: painful rheumatoid arthritis, osteoarthritis or TMJ dysfunction not responding to conservative measures. Inject a 1 mL solution of local anaesthetic and corticosteroid in equal parts, around 1 cm anterior to where the top of the tragus meets the face.
- Dental management that may be required for malfunction of the bite includes dental occlusal splinting.
- NSAIDs: a trial of NSAIDs, e.g. ibuprofen 400 mg (o) tds for 10 days, for TMJ inflammation may need consideration. Cease if no response after 10 days.

## Inflammatory or ulcerative oropharyngeal lesions

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A variety of ulcerative conditions and infections of structures such as gingivae, tongue, tonsils, larynx and pharynx can cause facial pain (refer to [CHAPTER 61](#) ). Gingivostomatitis, herpes labialis (cold sores) and aphthous ulceration are common examples. Lesions of the posterior third of the tongue, the oropharynx, tonsils and larynx may radiate to the region of the ear via the tympanic branch of the ninth nerve or the auricular branch of the tenth nerve.

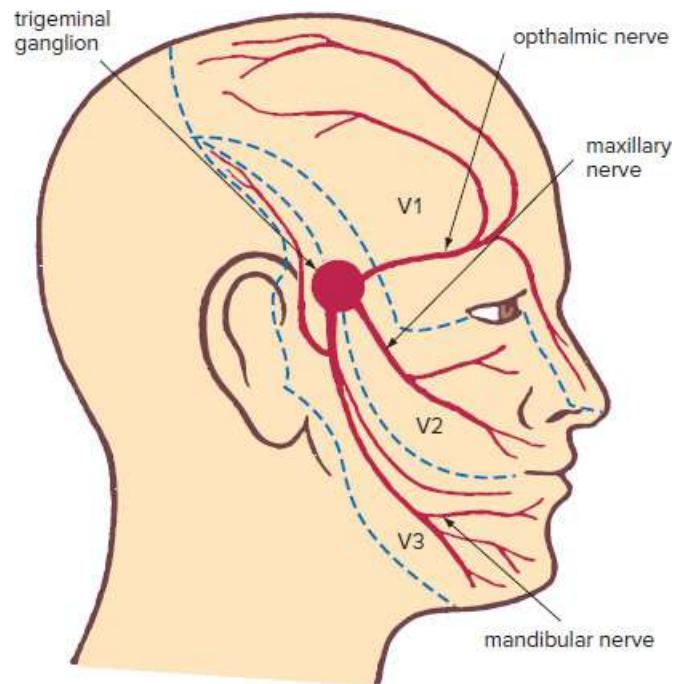
### Trigeminal neuralgia

Trigeminal neuralgia (tic douloureux) is a condition of often unknown cause that typically occurs

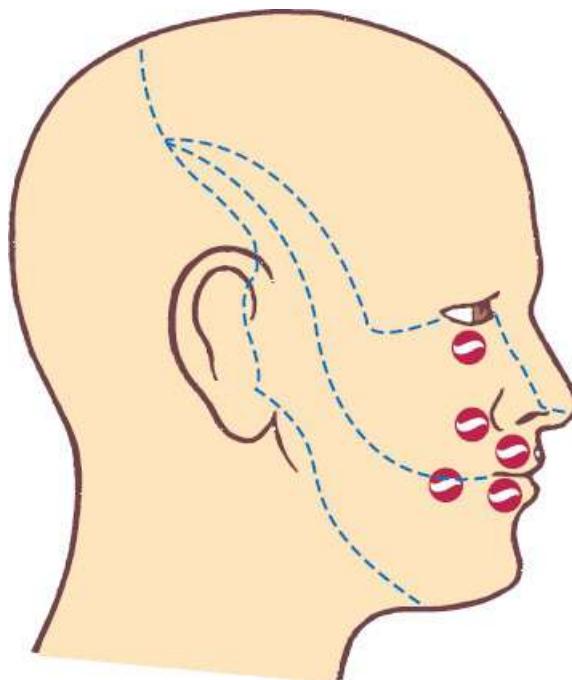
in patients over the age of 50, affecting the second and third divisions of the trigeminal nerve and on the same side of the face. Brief paroxysms of pain, often with associated trigger points, are a feature.

## Clinical features

- Site: sensory branches of the trigeminal nerve (see FIG. 41.5 ) almost always unilateral (often right side)
- Radiation: tends to commence in the mandibular division and spreads to the maxillary division and (rarely) to the ophthalmic division
- Quality: excruciating, searing jabs of pain like a burning knife or electric shock
- Frequency: variable and no regular pattern
- Duration: seconds to 1–2 minutes (up to 15 minutes)
- Onset: spontaneous or trigger point stimulus
- Offset: spontaneous
- Precipitating factors: talking, chewing, touching trigger areas on face (e.g. washing, shaving, eating), cold weather or wind, turning onto pillow
- Aggravating factors: trigger points usually in the upper and lower lip, nasolabial fold or lower eyelid (see FIG. 41.6 )
- Relieving factors: nil
- Associated features: rarely occurs at night; spontaneous remissions for months or years
- Signs: there are no signs, normal corneal reflex



**FIGURE 41.5** Typical cutaneous sensory distribution of the trigeminal nerve and its branches



**FIGURE 41.6** Trigeminal neuralgia: typical trigger points

## Causes

- Unknown
- Local pressure on the nerve root entry zone by tortuous pulsatile dilated small vessels (probably up to 75%)
- Multiple sclerosis
- Neurosyphilis
- Tumours of the posterior fossa

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*Note:* Precise diagnosis of a condition that can become a burdensome ‘label’ is important. MRI may be helpful.

## Treatment

- Patient education, reassurance and empathic support is very important in these patients.

### Medical therapy

carbamazepine (from onset of the attack to resolution)<sup>9</sup> 50 mg (elderly patient) or 100 mg (o) bd initially; gradually increase the dose to avoid drowsiness every 7 days to 400 mg bd

Alternative drugs if carbamazepine not tolerated or ineffective (but question the diagnosis if lack of response):

- oxcarbazepine 300 mg bd
- gabapentin 300 mg at night initially, increasing gradually to 600–1200 mg tds
- lamotrigine 25 mg (o) alternate daily, slowly increasing every 14 days if necessary to 100 mg bd
- phenytoin 300–500 mg daily
- phenytoin 300 mg daily
- baclofen 5 mg bd initially, increasing every 4 days up to 10–20 mg tds

## Surgery

- Refer to a neurosurgeon if medication ineffective
- Possible procedures include:

decompression of the trigeminal nerve root (e.g. gel foam packing between the nerve and

blood vessels)

neuroablative treatment, e.g. thermocoagulation/radiofrequency neurolysis

surgical division of peripheral branches

## **Glossopharyngeal neuralgia<sup>9,10,11</sup>**

This is an uncommon condition of the ninth cranial nerve and branches of the vagus nerve with similar clinical features of severe, lancinating pains, particularly felt in one ear, the base of the tongue or beneath the angle of the jaw. The pain usually lasts 30–60 seconds.

- Sites: back of throat around tonsillar fossa and adjacent fauces deep in ear, back of tongue
- Radiation: ear canal, neck
- Triggers: swallowing (esp. cold liquids), coughing, talking, yawning, laughing
- Treatment: as for trigeminal neuralgia

## **Migrainous neuralgia (cluster headache)**

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As described in [CHAPTER 45](#), the pain is unilateral and centred around the eye with associated lacrimation and stuffiness of the nose.

## **Facial migraine (lower half headache)<sup>9</sup>**

Migraine may rarely affect the face below the level of the eyes, causing pain in the area of the cheek and upper jaw. It may spread over the nostril and lower jaw. The pain is dull and throbbing, and nausea and vomiting are commonly present. The treatment is as for other varieties of migraine with simple analgesics or ergotamine for infrequent attacks.

## **Paroxysmal hemicrania/hemicrania continua**

In the rare condition of chronic or episodic paroxysmal hemicrania, there is a unilateral facial pain that can resemble chronic cluster headache but the duration is briefer, about 15 minutes, and it may recur many times a day even for years. It responds dramatically to indomethacin, e.g. indomethacin 25 mg (o) tds initially, then adjusting.<sup>12</sup> The chronic form is termed hemicrania continua, which persists for more than 3 months.

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## **Herpes zoster and postherpetic neuralgia**

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Refer to [CHAPTER 114](#). Herpes zoster may present as hyperaesthesia or a burning sensation in

any division of the fifth nerve, especially the ophthalmic division.

## ⌚ Atypical facial pain

Also known as persistent idiopathic facial pain, it is mainly a diagnosis of exclusion whereby patients, usually middle-aged to elderly women, complain of diffuse pain in the cheek (unilateral or bilateral) without demonstrable organic disease. The pain does not usually conform to a specific nerve distribution (although in the maxillary area), varies in intensity and duration and is not lancinating as in trigeminal neuralgia. It is usually described as deep-seated and ‘boring’, severe, continuous and throbbing in nature. It is a very confusing and difficult problem to treat. These patients tend to show psychoneurotic tendencies and it can be exacerbated by stress, but caution is needed in labelling them as functional.

### Treatment

Trial of an antidepressant,<sup>9</sup> e.g.:

amitriptyline 10–75 mg nocte (first line)

or

dothiepin 25–150 mg nocte

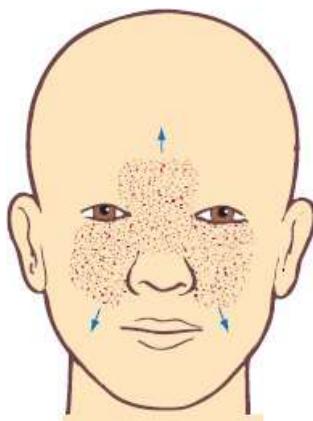
## Temporal arteritis

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This may produce mild or severe unilateral or bilateral headache. There may be ischaemic pain in the jaws when chewing. There may be marked scalp tenderness over the affected arteries. See CHAPTER 21 for management.

## ⌚ Erysipelas

Classical erysipelas is a superficial form of cellulitis involving the face. It usually presents with the sudden onset of butterfly erythema with a well-defined edge (see FIG. 41.7). It often starts around the nose and there may be underlying sinus or dental infection which should be investigated. There is an associated ‘flu-like’ illness and fever. It is invariably caused by *Streptococcus pyogenes*. Treatment is by phenoxymethylpenicillin or di/flucloxacillin for 7–10 days.



**FIGURE 41.7** Erysipelas: typical spreading distribution of the infection

## When to refer

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- Severe trigeminal or glossopharyngeal neuralgia
- Unusual facial pain, especially with a suspicion of malignancy
- Continuing pain of uncertain cause
- Positive neurological signs, such as impaired corneal reflex, impaired sensation in a trigeminal dermatome, slight facial weakness, hearing loss on the side of the neuralgia
- Possible need for surgical drainage of sinusitis—indications for surgery include failure of appropriate medical treatment, anatomical deformity, polyps, uncontrolled sinus pain<sup>5</sup>
- Dental root infection causing maxillary sinusitis
- Other dental disorders

### Practice tips

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- Malignancy must be excluded in the elderly with facial pain.
- Problems from the molar teeth, especially the third (wisdom), commonly present with peri-auricular pain without aural disease and pain in the posterior cheek.
- Facial pain never crosses the midline; bilateral pain means bilateral lesions.<sup>13</sup>
- If no obvious cause of persistent pain, refer to exclude sinister cause: don't overdiagnose sinusitis.

# Patient education resources

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

- Sinusitis
- Temporomandibular joint dysfunction

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## 42 Fever and chills

*Why! The fever itself is nature's instrument.*

THOMAS SYDENHAM (1624–1689), *MEDICAL OBSERVATIONS*

Although fever is a sign of disease and usually occurs in response to infection (mainly viral), its presence is recognised as playing an important role in the individual's defence against infection. The infecting pathogen triggers hypothalamic receptors, causing the thermostatic mechanisms to be reset to maintain core temperature at a higher level. The elevation in temperature results from increased heat production (e.g. shivering) or decreased loss (e.g. peripheral vasoconstriction). The elevation in body temperature activates T-cell production, increases the effectiveness of interferons and limits the replication of some common viruses.<sup>1</sup>

### Key facts and checkpoints

- Fever plays an important physiological role in the defence against infection.
- Normal body temperature (measured orally mid-morning) is 36–37.2°C (average 36.8°C).
- Fever can be defined as an early-morning (6 am) maximal oral temperature >37.2°C or a temperature >37.8°C at other times of the day, typically 4 pm.<sup>2</sup>
- Oral temperature is about 0.4°C lower than core body temperature.
- Axillary temperature is 0.5°C lower than oral temperature.
- Rectal, vaginal and ear drum temperatures are 0.5°C higher than oral and reflect core body temperature.
- There can be a normal diurnal variation of 0.5–1°C.
- Fevers due to infections have an upper limit of 40.5–41.1°C.
- Hyperthermia (temperature above 41.1°C) and hyperpyrexia appear to have no upper limit.

- Infection remains the most important cause of acute fever.<sup>3</sup>
- Symptoms associated with fever include sweats, chills, rigors and headache.
- Causes of fever besides infections include malignant disease, mechanical trauma (e.g. crush injury), vascular accidents (e.g. infarction, cerebral haemorrhage), immunogenic disorders (e.g. drug reactions, SLE), acute metabolic disorders (e.g. gout), and haemopoietic disorders (e.g. acute haemolytic anaemia).<sup>3</sup>
- Drugs can cause fever, presumably because of hypersensitivity.<sup>3</sup> Important examples are allopurinol, antihistamines, barbiturates, cephalosporins, cimetidine, methyldopa, penicillins, isoniazid, quinidine, phenolphthalein (including laxatives), phenytoin, procainamide, salicylates and sulfonamides.
- Drug fever should abate by 48 hours after discontinuation of the drug.<sup>4</sup>
- Infectious diseases at the extremes of age (very young and aged)<sup>3</sup> often present with atypical symptoms and signs. Their condition may deteriorate rapidly.
- Overseas travellers or visitors may have special, even exotic infections and require special evaluation (refer to [CHAPTER 129](#) ).
- Immunologically compromised patients (e.g. those with AIDS) pose a special risk for infections, including opportunistic infections.
- A febrile illness is characteristic of the acute infection of HIV: at least 50% have an illness that presents like glandular fever.

## Chills/rigors<sup>2</sup>

The abrupt onset of fever with a chill or rigor is a feature of some diseases. Examples include:

- bacteraemia/septicaemia
- pneumococcal pneumonia
- pyogenic infection with bacteraemia
- lymphoma
- pyelonephritis
- visceral abscesses (e.g. perinephric, lung)
- malaria

- biliary sepsis (Charcot triad—jaundice, right hypochondrial pain, fever/rigors)

Features of a true chill are teeth chattering and bed shaking, which is quite different from the chilly sensations that occur in almost all fevers, particularly those in viral infections. The event lasts 10–20 minutes.

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Other features:

- shaking cannot be stopped voluntarily
- absence of sweating
- cold extremities and pallor (peripheral vascular shutdown)
- dry mouth and pilo-erection: lasts 10–20 minutes

## Hyperthermia

Hyperthermia or hyperpyrexia is a temperature greater than 41.1°C. A more accurate definition is a state when the body's metabolic heat production or environmental heat load exceeds normal heat loss capacity. Hyperthermia may be observed particularly in the tropics, in malaria and heatstroke. It can occur with CNS tumours, infections or haemorrhages because of their effect on the hypothalamus.

### Heatstroke (sunstroke, thermic fever)<sup>5</sup>

This is the sudden onset of hot, dry, flushed skin with a rapid pulse, temperature above 40°C, and confusion or altered conscious state in a person exposed to a very hot environment. The BP is usually not affected initially but circulatory collapse may precede death. It is a life-threatening emergency. The diagnosis is clinical. Differential diagnoses include severe acute infection, toxic shock, food, chemical and drug poisoning. The elderly and debilitated are susceptible, as are children left in cars.

#### Treatment

- Immediate effective cooling water applied to skin—cool sprays, fanning
- Icepacks at critical points (e.g. axillae, neck, head)
- Full body immersion works, but caution in sick people
- Aim to bring down temperature by 1°C every 10 minutes

### Malignant hyperthermia

This is a rare hereditary disorder characterised by rapidly developing hyperpyrexia, muscular

rigidity and acidosis in patients undergoing major surgery.

## Sweats

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Sweating is a heat loss mechanism, and diffuse sweating that may soak clothing and bedclothing permits rapid release of heat by evaporation. In febrile patients the skin is usually hot and dry—sweating occurs in most when the temperature falls. It is characteristic of only some fevers (e.g. septic infections and rheumatic fever).

## Febrile neutropenia

This is fever usually with a temperature  $\geq 38^{\circ}\text{C}$  in a patient with neutrophils  $<0.5 \times 10^9/\text{L}$ . It is a common complication of people undergoing cancer therapy. If possible, the pathogen should be identified and broad-spectrum antibiotics initiated urgently. Refer to the appropriate hospital or specialist service.

## Factitious fever

Factitious fever is usually encountered in hospitalised patients attempting to malinger. The situation is usually suspected when:

- a series of high temperatures is recorded to form an atypical pattern of fluctuation
- there is excessively high temperature ( $41.1^{\circ}\text{C}$ ) and above
- a recorded high temperature is unaccompanied by warm skin, tachycardia and other signs of fever such as a flushed face and sweating
- there is an absence of diurnal variation

The patient may have surreptitiously dipped the thermometer in warm water, placed it in contact with a heat source or heated the bulb by friction with bedclothes or even mucous membranes of the mouth.

## Neuroleptic malignant syndrome

This is often confused with ‘malignant’ hyperthermia and heat stroke. The syndrome includes high temperature, muscle rigidity, autonomic dysfunction and altered consciousness. It is a rare and potentially lethal reaction in patients taking antipsychotic drugs, particularly occurring with haloperidol alone or with other drugs, especially lithium carbonate. Refer to [CHAPTER 68](#).

## Measurement of temperature

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Temperature can be measured by several methods, including the liquid crystal thermometer, the

electronic probe thermometer, the digital infrared aural device, the forehead skin (temporal area) device and the digital peak-hold thermometer, which is the favoured general instrument for oral and rectal use.

## Oral use

1. Place under the tongue at the junction of the base of the tongue and the floor of the mouth to one side of the frenulum—the ‘heat’ pocket.
2. Ensure that the mouth is kept shut.
3. Remove dentures.

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## Rectal use

This is an appropriate route for babies and young children under the age of 4 years but should be used with care. The rule is ‘2–3 cm in for 2–3 minutes’ and some authorities claim that this method is the gold standard for infants.

### Method

1. Lubricate the stub with petroleum or KY jelly.
2. Insert for 2–3 cm past anal verge.
3. Keep the thermometer between the flexed fingers with the hand resting on the buttocks (see FIG. 42.1).



**FIGURE 42.1** Rectal temperature measurement in infants

Don't:

- dig thermometer in too hard
- hold it too rigidly
- allow the child to move around

### Axillary use

This is unreliable with poor sensitivity and generally should be avoided but may be practical in young children.<sup>6</sup>

If used, place high in the axilla for 3 minutes. Fever is present if the temperature is above 37.2°C.

### Groin use

This route is not ideal but is more reliable than the axilla. It closely approximates oral temperature. In infants, the thigh should be flexed against the abdomen.

### Vaginal use

This is self-administered as an adjunct to the assessment of ovulation during the menstrual cycle. It should be placed deeply in the vagina for 5 minutes before leaving bed in the morning.

### Infrared aural (eardrum) use

Tympanic (otic) thermography is now accepted standard practice. The temperature can be measured in 3 seconds, with the infrared device placed in the ear canal. First, hold the child's head firmly to avoid any movement, then take temperature. The tympanic membrane (TM) accurately reflects hypothalamic temperature, which in turn reflects core body temperature. The TM is also immune from the effects of eating, drinking and smoking. A systematic review in the Cochrane study questions its reliability. It is poor in small infants but useful over the age of 6 months.<sup>7</sup> However, some Australian authorities believe that in general practice the benefits of convenience outweigh the lack of accuracy.<sup>8</sup> The normal range is the same as for rectal temperature.

The same infrared technology is used to measure skin temperature overlying the temporal artery. This no-touch 'forehead' thermometer has markedly increased in popularity since the COVID-19 pandemic.

### Digital electronic pacifier (dummy) thermometer

This is a popular method for infants and younger children, favoured by many paediatricians in the US.

### Skin use

Plastic strip thermometers placed on the forehead are very inaccurate and should not be used.

## The clinical approach

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The initial approach is to evaluate the severity of the problem and the nature of the illness. Some infections, particularly bacterial infections, are life-threatening and this requires urgent diagnosis and hospital admission.

According to Yung and Stanley<sup>3</sup> it is helpful to consider fever in three categories: less than 3 days duration; between 4 and 14 days duration; and protracted fever (more than 14 days).

### Fever of less than 3 days duration

This is very commonly encountered in family practice, often due to a self-limiting viral Page 512 infection of the respiratory tract. It is important, however, to be vigilant for other infections, so evidence of an infectious disease, urinary tract infection, pneumonia or other infection should be sought. Where there is no obvious focus of infection, a urine examination, especially in adult females and children, is an important screening investigation. The majority of patients can be managed conservatively.

### Fever present for 4–14 days

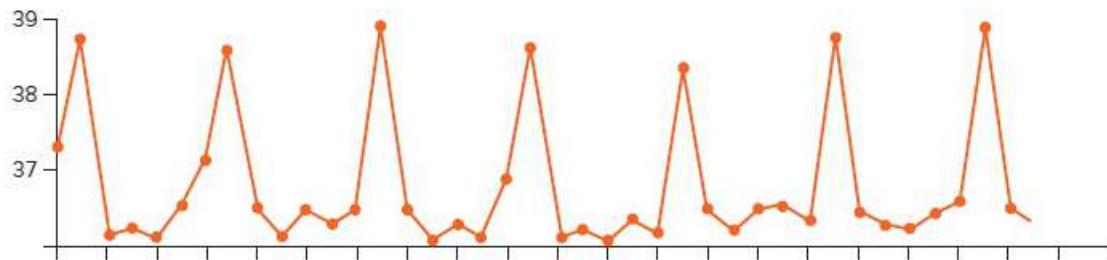
If fever persists beyond 4–5 days a less common infection should be suspected since most common viral infections will have resolved by about 4 days.<sup>3</sup> A careful history is mandatory, as outlined later in the chapter for fever of unknown origin (FUO). The basic examination and investigations are along similar lines.

### Patterns of fever

The patterns of fever may be of diagnostic help as some febrile conditions follow predictable temperature patterns (see FIG. 42.2 <sup>9</sup>).

- *Intermittent fever:* The temperature rises for a few hours each day and then returns to normal. Typical of most pyogenic infections, cytomegalovirus and lymphoma.
- *Relapsing fever:* The fever returns to normal for days before rising again. Malaria is the classic example of periodic relapsing fever: every third day for *Plasmodium malariae* and every second day for *Plasmodium vivax*.
- *Remittent fever:* The temperature returns towards normal for a variable period but is always elevated. Common examples are larger collections of pus: pelvic abscess, wound infection, empyema and also carcinoma.
- *Undulant fever:* Bouts of continuous or remittent fever for several days, followed by afebrile remissions lasting a variable number of days. It is commonly a feature of brucellosis infection but is also seen in the lymphomas, especially Hodgkin lymphoma where ‘Pel–Ebstein’ fever lasts 3–10 days followed by afebrile periods of 3–10 days.
- *Continuous fever:* Common with viral infections such as influenza.

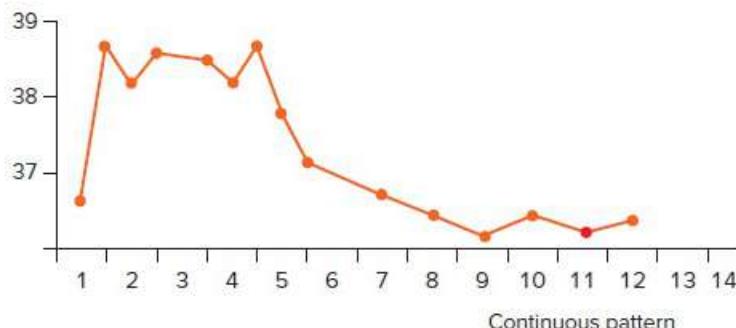
- *Quotidian fever*: The fever recurs daily. Morning spikes are characteristic of *Pseudomonas* infection (e.g. pulmonary superinfection); afternoon spikes are indicative of cytomegalovirus infection; and evening spikes suggest localised collection of pus (e.g. empyema of the gall bladder).



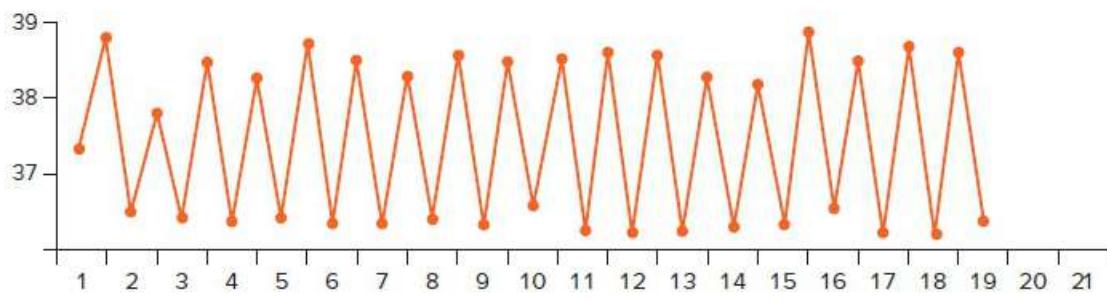
Periodic relapsing fever: quartan fever of malaria (a 4-day pattern with fever peaks every third day)



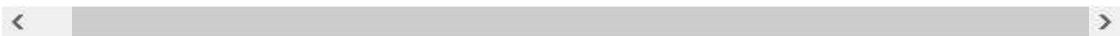
Undulant fever: due to lymphoma



Continuous pattern



Quotidian fever pattern and intermittent fever  
(e.g. empyema of gall bladder)



## FIGURE 42.2 Examples of fever patterns

### Postoperative fever

This is fever occurring within 24 hours after surgery—common with abdominal surgery.

Causes to consider:

- pulmonary atelectasis (common)
- wound haematoma
- deep venous thrombosis
- myocardial infarction
- allergic drug reaction
- transfusion reaction

Septic problems related to the operation usually develop after several days.

### Fever in children

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In children, most authorities would consider a fever of 38.5°C and above to be significant and warrant close scrutiny.<sup>10</sup>

Important causes not to be missed:

- urinary infection
- meningitis/encephalitis
- pneumonia
- septicaemia/bacteraemia
- osteomyelitis
- septic arthritis
- pertussis
- abscess

The fever is usually a response to a viral infection. Fever itself is not harmful until it reaches a level of 41.5°C.<sup>1</sup> Hyperthermia is uncommon in children. Temperatures above 41°C are usually due to CNS infection or the result of human error, for example:

- shutting a child in a car on a hot day
- overwrapping a febrile child

Complications include dehydration (usually mild) and febrile convulsions, which occur in 5% of febrile children between 6 months and 5 years. Febrile convulsions are triggered by a rapid rise in temperature rather than its absolute level. They are not prevented by paracetamol or ibuprofen.

*Note:* Teething does *not* cause fever.

## Approach to the febrile infant

It is important to decide whether the child looks well or seriously ill. Identification of the very ill child is presented in [CHAPTER 89](#).

If the child is well and has no risk factors (such as unreliable caregiver, poor access to treatment, medical risk factors, taking antibiotics) treat expectantly. The only test required is urine microscopy and culture. Educate the caregiver about review if serious signs develop. Treat the fever as outlined in [TABLE 89.1](#), in [CHAPTER 89](#).

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## Management

- Treatment of low-grade fevers should be discouraged.

- Treatment of high-grade fevers includes:

treatment of the causes of the fever (if appropriate)

adequate fluid intake/increased fluids

paracetamol (acetaminophen) is the preferred antipyretic since aspirin is potentially dangerous in young children (use paracetamol if temperature >38.5°C). The usual dose of 10–15 mg/kg every 4–6 hours may represent undertreatment. Use 20 mg/kg as a loading dose and then 15 mg/kg maintenance

ibuprofen 5–10 mg/kg every 6 hours is a suitable antipyretic

Summary: Tepid sponging for the first 30 minutes combined with paracetamol is preferred management.

## Advice to parents

- Dress the child in light clothing (stripping off is unnecessary).
- Do not overheat with too many clothes, rugs or blankets.
- Give frequent small drinks of light fluids, especially water.
- Sponging the forehead with water is optional, but do not immerse in a cold bath.

## Febrile convulsions

Refer to [CHAPTER 89](#).

## Fever in the elderly

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The elderly tend to have a problem with impaired thermoregulation and so they may not develop a fever in response to suppurative infection compared with younger people. This can be misleading in the diagnostic process.

## Important facts

- Any fever in the elderly is significant.
- Viral infection is a less common cause of fever in the elderly.
- Fever in the elderly is sepsis until proven otherwise (common sites are the lungs and urinary tract).

The elderly are more vulnerable to hyperthermia and hypothermia. Heatstroke classically occurs in epidemic form during a heatwave. The syndrome consists of hyperpyrexia, decreased sweating, delirium and coma. The core temperature is usually over 41°C.

## ‘Alarm bell’ signs

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In many patients the existence of a life-threatening infective illness is obvious and prompt action is essential. In others the diagnosis is not clear-cut but there are certain warning signs (see [Red flags box](#)).

These ‘red flag’ symptoms and signs are obviously super ‘sensitive’. Patients with some of these features may have potentially life-threatening diseases, but this list would include many with viral infections. A more targeted subset of this list using specific numerical thresholds is used to create Early Warning Scores (EWS), variations of which include REWS (Remote—Australia), MEWS (Modified), NEWS (National—UK) and PEWS (Paediatric).

## Fever of undetermined origin

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Fever of undetermined origin (FUO), also referred to as pyrexia of unknown origin (PUO), has the following (Petersdorf–Beeson modified) criteria.<sup>11</sup>

- illness for at least 3 weeks
- fevers >38.3°C (100.9°F) on several occasions
- undiagnosed after 1 week of intensive study

### Red flag pointers for fever

- High fever
- Repeated rigors
- Drenching night sweats
- Severe myalgia (?sepsis)
- Severe pain anywhere (?sepsis)
- Severe sore throat or dysphagia (?*Haemophilus influenzae* epiglottitis)
- Altered mental state
- Incessant vomiting
- Unexplained rash
- Jaundice
- Marked pallor
- Tachycardia
- Tachypnoea

Most cases represent unusual manifestations of common diseases and not rare or exotic diseases. Examples are tuberculosis, bacterial endocarditis, hepatobiliary disease and lung cancer.<sup>12</sup>

Keep in mind that the longer the duration of fever, the less likely the diagnosis is to be infectious —fevers that last greater than 6 months are rarely infectious (only 6%). One study showed that 9% are factitious.<sup>13</sup>

Patients with FUO in definite need of further investigation are:

- babies <3 months of age
- children with fever >40°C
- adults >50 years
- people with diabetes
- the immunocompromised
- travellers

## A diagnostic approach

A knowledge of the more common causes of FUO is helpful in planning a diagnostic approach (refer to [TABLE 42.1](#) ).

**Table 42.1** Diagnostic strategy for fever that is prolonged (FUO)

### Probability diagnosis

- Pyogenic abscess (anywhere, e.g. liver, pelvis)
- Pneumonia (viral, bacterial, atypical)
- Epstein–Barr mononucleosis
- Viral upper respiratory tract infection
- Urinary infection (incl. chronic pyelonephritis)

### Serious disorders not to be missed

#### Vascular:

- vasculitides (polyarteritis nodosa, giant cell arteritis/polymyalgia)

#### Infection:

- HIV/AIDS
- malaria and other tropical diseases
- zoonoses (e.g. leptospirosis, Q fever, listeriosis)
- typhoid/paratyphoid fever
- tuberculosis
- osteomyelitis
- chronic septicaemia/bacteraemia
- infective endocarditis
- Lyme disease
- syphilis (secondary)

#### Cancer (up to 30%):

- lymphoma and leukaemia
- solid cancers (e.g. lung, kidney)
- disseminated

Other:

- febrile neutropenia
- inflammatory bowel disease (e.g. Crohn)

### Pitfalls (often missed)

Connective tissue disorder (e.g. rheumatoid arthritis, systemic lupus erythematosus)

Sarcoidosis

Drug idiosyncrasies

*Rarities:*

- factitious fever

*Note:* Up to 20% remain unknown.

## History

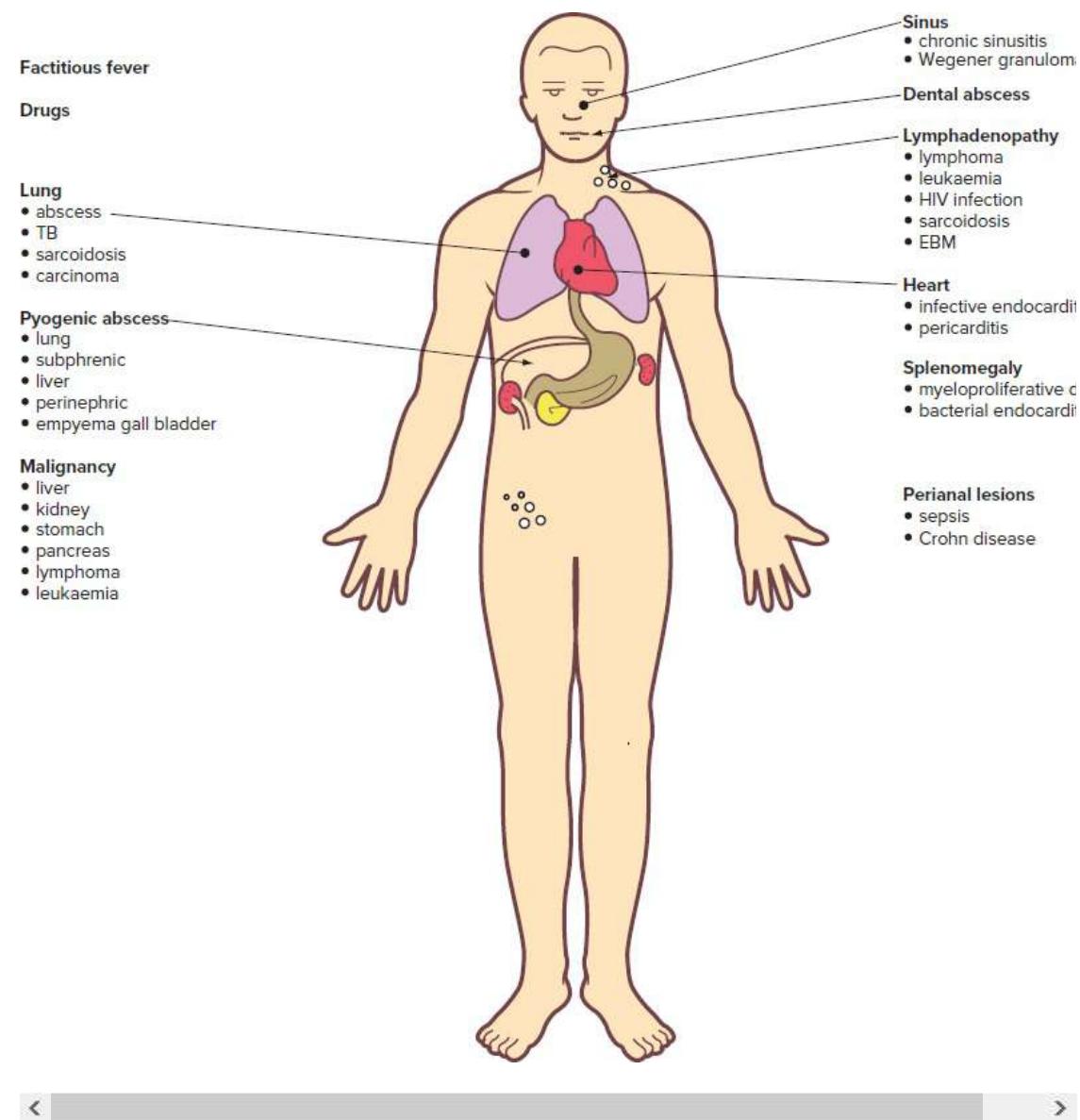
The history should include consideration of past history, occupation, travel history, sexual history, IV drug use (leads to endocarditis and abscesses), animal contact, medication and other relevant factors. Symptoms such as pruritus, a skin rash and fever patterns may provide clues for the diagnosis. The average patient with a difficult FUO needs to have a careful history taken on at least three separate occasions.<sup>14</sup>

## Examination

A common mistake is the tendency to examine the patient only once and not re-examine. The patient should be examined regularly (as for history taking) as physical signs can develop eventually. HIV infection must be excluded. Special attention should be paid to the following (see FIG. 42.3):

- skin—look for rashes, vesicles and nodules
- the eyes and ocular fundi
- temporal arteries
- sinuses and ears (canal and TM)
- teeth and oral cavity—?dental abscess, other signs
- heart—murmurs, pericardial rubs

- lungs—abnormalities including consolidation, pleuritic rub
- abdomen—enlarged/tender liver, spleen or kidney
- rectal and pelvic examination (note genitalia)
- lymph nodes, especially cervical (supraclavicular)
- blood vessels, especially of the legs—?thrombosis
- urine (analysis)



**FIGURE 42.3** Sites to consider in FUO

## Investigations<sup>15</sup>

FUOs in primary care (not hospitalised) should be investigated as a ‘series’, rather than ordering everything at once in ‘parallel’. Start with a broad brush, then focus in on likely possibilities. Basic investigations include:

- haemoglobin, red cell indices and blood film
- white cell count
- ESR/C-reactive protein
- chest X-ray and sinus films
- urine examination (analysis and culture)
- routine blood chemistry
- blood cultures

Further possible investigations (depending on clinical features):

- stool microscopy and culture
- culture of sputum (if any)
- specific tests for malaria, typhoid, EBM, Q fever, brucellosis, psittacosis, cytomegalovirus, toxoplasmosis, syphilis, various tropical diseases and others
- NAAT (e.g. PCR) tests
- HIV screening
- tests for rheumatic fever
- tuberculin test
- tests for connective tissue disorders (e.g. DNA antibodies, C-reactive protein)
- upper GIT series with small bowel follow-through
- CT and ultrasound scanning for primary and secondary neoplasia
- gall bladder functioning
- occult abscesses
- MRI—best for detecting lesions of the nervous system

- echocardiography—for suspected endocarditis
- isotope scanning for specific causes
- aspiration or needle biopsy
- laparoscopy for suspected pelvic infection
- tissue biopsies (e.g. lymph nodes, skin, liver, bone marrow) as indicated

## FUO in children

Fever in children is usually a transient phenomenon and subsides within 4–5 days. At least 70% of all infections are viral. Occasionally a child will present with FUO whose clinical symptoms and signs may be masked from antibiotic administration. Common causes of prolonged fever in children differ from those in adults. Most cases are not due to unusual or esoteric disorders,<sup>16</sup> the majority representing atypical manifestation of common diseases.

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A summary of the common causes (with the most common ranked first) is as follows.<sup>16</sup>

### Infectious causes (40%)

- Viral syndrome
- Urinary tract infection
- Pneumonia
- Pharyngitis
- Sinusitis
- Meningitis

### Collagen–vascular disorders (15%)

- Rheumatic arthritis
- Systemic lupus erythematosus
- Rheumatic fever
- Henoch–Schönlein syndrome

### Neoplastic disorders (7%)

- Leukaemia

- Reticulum cell sarcoma
- Lymphoma

## Inflammatory diseases of the bowel (4%)

### Septicaemia

The diagnosis of septicaemia can be easily missed, especially in small children, the elderly and the immunocompromised, and in the absence of classic signs, which are:

- fever ( $\pm$  shivering)
- muscle pain
- rash (suggestive of meningococcus)
- tachycardia
- tachypnoea
- cool extremities

Patients with septicaemia require urgent referral as it has a very high mortality rate.<sup>17</sup> Investigations should include two sets of blood cultures and other appropriate cultures (e.g. urine, wound, sputum). Empirical initial treatment in adults (after blood cultures) is vancomycin IV and gentamicin IV.<sup>18</sup>

### Glossary of terms

**Bacteraemia** The transient presence of bacteria in the blood (usually implies asymptomatic) caused by local infection or trauma.

**Septicaemia (sepsis)** The multiplication of bacteria or fungi in the blood, usually causing a systemic inflammatory response (SIRS). SIRS is defined as two or more of (in adults):

- temperature  $>38^{\circ}\text{C}$  or  $<36^{\circ}\text{C}$
- respiratory rate  $>20/\text{min}$
- heart rate  $>90/\text{min}$
- WCC  $>12 \times 10^9/\text{L}$  or  $<4 \times 10^9/\text{L}$

**Severe sepsis** Sepsis associated with organ dysfunction, hypoperfusion or

hypotension with two or more of: fever, tachycardia, tachypnoea and elevated WCC.

**Septic shock** Sepsis with critical tissue perfusion causing acute circulatory failure including hypotension that does not respond to IV fluid administrations and peripheral shutdown—cool extremities, mottled skin, cyanosis. Consider *S. aureus* (food poisoning, tampon use) and *S. pyogenes*.

**Pyaemia** A serious manifestation of septicaemia whereby organisms and neutrophils undergo embolisation to many sites, causing abscesses, especially in the lungs, liver and brain.

**Primary septicaemia** Septicaemia where the focus of infection is not apparent, while in secondary septicaemia a primary focus can be identified.

Examples of secondary septicaemia in adults are:

- urinary tract (e.g. *Escherichia coli*)
- respiratory tract (e.g. *Streptococcus pneumoniae*)
- pelvic organs (e.g. *Neisseria gonorrhoeae*)
- skin (e.g. *Staphylococcus aureus*)
- gall bladder (e.g. *E. coli*, *Streptococcus faecalis*)

Patients with septicaemia require urgent referral.

## Patient education resources

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

- Febrile convulsions
- Fever

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## 43 Faints, fits and funny turns

*Persons who have had frequent and severe attacks of swooning, without any manifest cause, die suddenly.*

HIPPOCRATES (?460–377 BCE), *APHORISMS*, 11, 41

When patients present with the complaint of a ‘funny turn’ it is usually possible to determine that they have one of the more recognisable presenting problems, such as fainting, ‘blackouts’, lightheadedness, weakness, palpitations, vertigo or migraine. However, there are some who do present with confusing problems that warrant the label of ‘funny turn’. The most common problem with funny turns is that of misdiagnosis, so it is essential to take a proper and adequate history.

It is important to remember that phrases like ‘funny turn’ or ‘feeling weird’ are ways of communicating subjective symptoms seen through a particular cultural and linguistic lens, often during times of stress.<sup>1</sup> Various causes of faints, fits and funny turns are presented in

TABLE 43.1 . A useful simple classification is to consider them as:

- syncope
- seizures
- sleep disorders—sleep apnoea/narcolepsy/cataplexy
- labyrinthine

**Table 43.1** Faints, fits and funny turns: checklist of causes

### Psychogenic/communication problems

- Breath-holding attack
- Conversion reactions (hysteria)
- Culture/language conflicts
- Fugue states
- Hyperventilation

Malingering  
Personality disorders  
Phobia/anxiety states  
Psychoses/severe depression

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#### Other conditions

Transient ischaemic attacks and strokes  
Complex partial seizure (temporal lobe epilepsy)  
Tonic, clonic or atonic seizures  
Primary absence seizure  
Migraine variants or equivalents, e.g. acute confusional migraine  
Familial periodic paralysis  
Cardiovascular disorders:

- arrhythmias
- Stokes–Adams attacks
- postural hypotension
- long QT syndrome
- aortic stenosis

Vertigo  
Drug reaction  
Alcohol and other substance abuse  
Hypoglycaemia  
Anaemia  
Head injury  
Amnesic episodes  
Metabolic/electrolyte disturbances  
Vasovagal/syncope  
Carotid sinus sensitivity  
Cervical spondylosis  
Sleep disorders:

- sleep apnoea
- narcolepsy/cataplexy

Autonomic failure

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#### Key facts and checkpoints

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- The commonest cause of 'funny turns' presenting in general practice is

lightheadedness, often related to psychogenic factors such as anxiety, panic and hyperventilation.<sup>2</sup> Patients usually call this ‘dizziness’.

- Absence attacks occur with minor forms of epilepsy and with partial seizures such as complex partial seizures.
- The psychomotor attack of complex partial seizure presents as a diagnostic difficulty. The most commonly misdiagnosed seizure disorder is that of complex partial seizures or variants of generalised tonic–clonic seizures (tonic or clonic or atonic).
- The diagnosis of epilepsy is made on the history (or video electroencephalogram/EEG), rather than on the standard EEG, although a sleep-deprived EEG is more effective.
- The triad—angina + dyspnoea + blackout or lightheadedness—indicates aortic stenosis.
- Severe cervical spondylosis can cause vertebrobasilar ischaemia by causing pressure on the vertebral arteries that pass through the intervertebral foramina, especially with head turning or looking up.

## A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 43.2 .

**Table 43.2** Faints, fits and funny turns: diagnostic strategy model

### Probability diagnosis

- Anxiety related/hyperventilation
- Vasovagal syncope
- Postural hypotension
- Breath-holding attacks/febrile convulsions (children)

### Serious disorders not to be missed

Cardiovascular:

- arrhythmias
- aortic stenosis
- postural orthostatic tachycardia syndrome (POTS)

Cerebrovascular:

- TIAs

Neoplasia:

- space-occupying lesions

Subdural/extradural haematomas

Severe infections:

- infective endocarditis

Hypoglycaemia

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### **Pitfalls (often missed)**

Atypical migraine

Cardiac arrhythmias/long QT syndrome

Simple partial seizures

Complex partial seizures

Atypical tonic–clonic seizures

Acute confusional state/delirium

Drugs/alcohol/marijuana

Electrolyte disturbances (e.g. hypokalaemia)

Sleep disorders

Transient global amnesia

Rarities:

- atrial myxoma
- 

### **Seven masquerades checklist**

Depression

Diabetes (hypoglycaemia)

Drugs

Anaemia

Spinal dysfunction (cervical spondylosis)

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

Highly likely. Pseudo-seizures, psychogenic and ‘communication’ disorders quite significant.

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## **Red flag pointers for faints, fits and funny turns**

- Onset in older person
- Neurological symptoms and signs
- Headache

- Tachycardia
- Irregular pulse
- Fever
- Rash
- Drugs: social or prescribed
- Cognitive impairment
- Confusion: gradual onset

## The clinical approach

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### History

The clinical history is of paramount importance in unravelling the problem. A reliable eye-witness account of the ‘turn’ is invaluable, as is the setting or circumstances in which the ‘episode’ occurred.

It is essential at first to determine exactly what the patient means by ‘funny turn’. In the Page 520 process of questioning it is appropriate to evaluate the mental state and personal and social factors of the patient. It may be appropriate to confront the patient about feelings of depression, anxiety or detachment from reality.

It is important to break up the history into three components. First is the lead-up to the episode; second is an adequate description of what took place during the episode; third are the events that took place after the episode.

Apart from the events, note the patient’s feelings, symptoms, circumstances and provocative factors. Search for possible secondary gain.

### Onset

A sudden onset may be due to cardiovascular causes, especially arrhythmias, which may include the more common supraventricular tachycardias in addition to the less common but more dramatic arrhythmias that may cause unconsciousness. Other causes of a sudden onset include the various epilepsies, vasovagal attacks and TIAs.

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### Precipitating factors<sup>2</sup>

Enquire about precipitating factors such as emotion, stress, pain, heat, fright, exertion, suddenly

standing up, coughing, head movement or hypersomnolence:

- emotion and stress suggest hyperventilation
- fright, pain → vasovagal attack
- standing up → postural hypotension
- exertion → aortic stenosis
- head movement → cervical spondylosis with vertebrobasilar insufficiency
- hypersomnolence → narcolepsy

## Associated symptoms<sup>2</sup>

Certain associated symptoms give an indication of the underlying disorder:

- breathing problems and hyperventilation suggest an anxiety state
- tingling in extremities or tightening of the hand → anxiety/hyperventilation
- visual problems → migraine or TIA
- fear or panic → anxiety or complex partial seizure (or realistic fear)
- hallucinations (taste/smell/visual) → complex partial seizure
- speech problems → TIA or anxiety
- sweating, hunger feelings → hypoglycaemia
- related to food → migraine
- first thing in morning → consider ‘hangover’

## Drug history

This requires careful analysis and includes alcohol intake and most illicit drugs, including marijuana, cocaine and amphetamines. Prescribed drugs that can cause lightheadedness or unconsciousness are listed in TABLE 43.3 and those causing seizures in TABLE 43.4 .

**Table 43.3** Typical drugs that may cause lightheadedness or blackouts

Alcohol  
Anti-epileptics

Antihypertensives  
Barbiturates  
Benzodiazepines  
Opioids  
OTC anticholinergic compounds  
Peripheral vasodilators:

- ACE/ARB inhibitors
- glyceryl trinitrate
- hydralazine
- prazosin

Phenoxybenzamine  
Antidepressants

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**Table 43.4** Important causes of convulsive seizures

Epilepsy:

- first presentation
- known epilepsy with recurrence

Cerebral hypoxia

Hypoglycaemia

Poor cerebral perfusion:

- oedema of eclampsia

Neurotrauma

Cerebrovascular accident

CNS infections:

- meningitis
- encephalitis
- septicaemia
- septic emboli
- cerebral abscess

Toxins

Alcohol excess

Hyperthermia

Metabolic disorders

Drugs:

- antidepressants
- theophylline
- amphetamine
- antibiotics, e.g. norfloxacin, ciprofloxacin
- cocaine
- local anaesthetics

Anaphylaxis

Expanding brain lesion:

- neoplasm
  - haematoma
- 

Sudden cessation of certain drugs such as phenothiazine antipsychotics can also be responsible for 'funny turns'.

## Past history

The past history may give an indication of the cause of the 'turn'. Such conditions include hypertension, migraine, epilepsy, rheumatic heart disease, diabetes, atherosclerosis (e.g. angina, vascular claudication), alcohol or other substance abuse and psychiatric disorders.

## Diary of events

If the diagnosis is elusive it may help to get the individual to keep a diary of circumstances in which events take place, keeping in mind the importance of the time period prior, during and post episode.

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## Examination

Important focal points of the physical examination include:

- evaluation of the mental state, especially for anxiety
- looking for evidence of anaemia, alcohol abuse and infection
- cerebrovascular examination: carotid arteries, ocular fundi, bruits
- cardiovascular examination: pulses, BP, heart (the BP should be taken lying and standing)
- the cervical spine

## Various manoeuvres

If the person is currently well and asymptomatic, a number of manoeuvres can try to induce various sensations in order to identify the one that affects them. These include sudden assumption of the erect posture from a squat, spinning around and then a sudden stop, head positioning with either ear down (see FIG. 35.3 , CHAPTER 35 ), Valsalva manoeuvre and hyperventilation for 60 seconds. Children blowing hard on a toy windmill mimics hyperventilation. Ask ‘Which one mimics your complaint?’

## Investigations

Depending on the clinical findings, investigations can be selected from the following tests:

- full blood count: ?anaemia ?polycythaemia
- blood sugar: ?diabetes ?hypoglycaemia
- metabolic studies: urea and electrolytes, calcium, magnesium
- ECG: ?ischaemia ?arrhythmia
- 24-hour ambulatory cardiac (Holter) monitor: ?arrhythmia
- radiology/imaging and neuroimaging:
  - cervical X-ray
  - chest X-ray
  - carotid duplex Doppler scan: ?carotid artery stenosis
  - CT scan
  - MRI scan
- EEG or video EEG; include those recorded with sleep deprivation, hyperventilation or photic stimulation
- positron emission tomography (PET) or single photon emission computerised tomography (SPECT) may show localised brain dysfunction when others are negative

## In neonates and children

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The various forms of seizures are also encountered in childhood, when most epilepsy is first diagnosed. Seizures can be categorised as subtle (staring, eye deviation, abnormal sucking or lip smacking) or tonic and clonic. Benign sleep myoclonus occurs in infants normally only during sleep and usually resolves by 2 months of age. It is appropriate to take a visual recording of the attack.<sup>3</sup>

Neonatal seizures are dangerous and require specialised care to identify the cause and quickly stop the seizure. Pyridoxine may be used.

## Epilepsy syndromes in children

The following are particular age-related epilepsy syndromes seen in children.<sup>4,5</sup>

### ⌚ Febrile seizures

Tonic–clonic seizures occur in 2–5% of children usually aged 3 months to 6 years who have a high fever generally caused by a viral infection. The long-term prognosis is good. For seizures lasting more than 5 minutes, use midazolam or diazepam.

### ⌚ Infantile spasms

Also known as West syndrome or Salaam attacks, these are generalised tonic seizures with sudden flexion of the arms, forward flexion of the trunk and extension of the legs, lasting only a few seconds, with the usual age of onset between 4 and 12 months. They are usually restricted to the first 3 years of life and are replaced by other forms of attacks. ECG shows typical hypsarrhythmias. Prognosis for cognitive development is unfavourable. The most effective therapy is corticotrophin (ACTH), e.g. tetracosactrin IM injection. Otherwise, oral prednisolone, vigabatrin, benzodiazepines or sodium valproate can be used.

### ⌚ Lennox–Gastaut syndrome (myoclonic epilepsy of infancy)

This uncommon syndrome refers to a triad of severe difficult-to-control seizures (usually tonic with drop attack), intellectual disability and characteristic EEG. The seizures usually begin between the ages of 1 and 6 years, with a peak onset at 3–5 years. Prognosis is poor. Sodium valproate is the therapy of choice.

### ⌚ Benign focal childhood epilepsy with centrotemporal spikes

This disorder usually begins in children aged 2–13 years, with a peak age of 5–8 years. Page 523 It is the most common form of childhood focal epilepsy, and there is a strong family history of epilepsy. The main feature is a simple partial motor or somatosensory seizure involving the face and mouth during sleep, producing a typical ‘glugging’ sound. The child usually wakes from sleep, goes to the parents and is unable to speak and has hemifacial contortions. It may progress to a tonic–clonic seizure. There is a characteristic EEG pattern. The prognosis is excellent as remission usually occurs around puberty. Carbamazepine is the therapy of choice.

### ⌚ Childhood and juvenile absence epilepsy

These children present with frequent absence seizures (formerly ‘petit mal’), often over a hundred daily. Peak age of onset is 4–9 years. The absence seizures can be very subtle. Signs include alteration of awareness (usually in the classroom), sudden onset, facial and other automatisms. Juvenile absence epilepsy presents later (10–15 years). First-line treatment is ethosuximide (best tolerated) or sodium valproate.

## **Juvenile myoclonic epilepsy (myoclonic epilepsy of Janz)**

This is a triad of seizures: myoclonic jerks, tonic–clonic seizures and absences. Onset is around puberty but may occur earlier. The myoclonic jerks and tonic–clonic seizures usually occur in the early morning after waking. Intellectual development is usually as normal, but the disorder is generally lifelong and is well controlled with sodium valproate.

## **Medial temporal lobe epilepsy**

This syndrome of complex partial seizures, which usually last 1–3 minutes, is seen in childhood. Transient postictal confusion and speech dysfunction is common. Those with medically intractable seizures respond well to surgery.

## **Dravet syndrome**

Known as severe myoclonic epilepsy of infancy, it is a catastrophic form with a very poor prognosis. It is largely inherited. Early diagnosis and treatment are essential.

## **Non-epileptic events resembling epileptic seizures in children<sup>4</sup>**

Many normal and abnormal behaviours seen in children resemble seizures but are unrelated to epilepsy. A careful history is very important. The following are examples:

- *Postures of spasticity and movement disorders.* These occur in children with neurological disorders such as cerebral palsy.
- *Syncope.* The child may describe a ‘sinking feeling’, or ‘everything getting louder’ prior to the loss of consciousness.
- *Breath-holding.* This often occurs after a crying spell and clonic movements may be seen at the end of the event.
- *Masturbation.* This behaviour leads to a tonic-like posture of the legs and preoccupation, especially in young girls.<sup>6</sup>
- *Munchausen by proxy.*<sup>7</sup> This syndrome of fictitious epilepsy described by a parent is becoming more recognised.

- *Psychogenic seizures (pseudo seizures)*. A diagnostic dilemma exists when these coexist with genuine seizures. These should be suspected when they occur in particular circumstances and the description of the ‘seizure’ is bizarre.
- *Shuddering*. Shuddering or shivering spells can resemble myoclonic jerks.
- *Night terrors*.<sup>8</sup> These episodes, which usually affect children aged 2–4 and 6–9, generally develop within 2 hours of sleep onset and last 1–2 minutes (sometimes longer). They are alarming and the child usually cannot be reassured or settled. A 6-week trial of phenytoin or imipramine can be used for severe problems.
- *Tics*. Motor tics can be quite complex but are usually brief involuntary movements involving the face and upper limbs.
- *Cardiac causes*. For example, long QT syndrome.

## Blackouts

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The important causes of blackout include the various syncopes that are listed in TABLE 64.4 (see CHAPTER 64 ) and the various forms of epilepsy. Epileptic seizures are the commonest cause of blackouts.

Important causes of convulsions (tonic–clonic seizures) are listed in TABLE 43.4 .

## Epilepsy

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Epilepsy is defined as a ‘tendency to recurrence of seizures’. It is a symptom, not a disease. A person should not be labelled as having epilepsy until at least two attacks have occurred.<sup>9</sup> Epilepsy is common. Prevalence depends on definition, and is often quoted as 1 in 30 or 50, but a 2017 systematic review found an overall lifetime prevalence of 0.0076 (1 person in 130).<sup>10</sup> It affects both sexes almost equally, with increased rates at the extremes of young and old age. Some forms run in families.

The most important factors in the management of epilepsy are the accurate diagnosis of the type of seizures; identification of the cause and appropriate investigation; the use of first-line drugs as the sole therapy of some weeks; and adjustment of the dose, according to clinical experience and plasma levels, to give maximum benefit.

Once epilepsy is diagnosed, the long-term prognosis falls into three broad categories: spontaneous remission (20–30%), remission with treatment (20–30%) or continuing seizures (30–40%).<sup>11</sup>

To be accurate in diagnosing seizures the diagnosis must be based on:<sup>12</sup>

- the patient’s memory of the seizure

- the patient's history (e.g. family history, toxin exposure, accidents, febrile convulsions, eclampsia)
- the observation of a witness to the seizure
- a general and neurological examination
- an EEG, although this has considerable limitations
- a CT scan or preferably MRI (especially if the EEG is focal and a tumour is suspected)

Long-term ambulatory EEG recording now provides more information and, coupled with video monitoring, provides a permanent record of the seizure which can be reviewed at will. The CT or MRI scan is necessary to exclude a focal cause (such as a cyst, tumour, malformation or abscess) which might be treatable by surgery. The MRI scan can indicate developmental migration disorders.

The scan may identify mesotemporal sclerosis (an abnormality in the hippocampus due to birth hypoxia), thereby making some 'idiopathic' seizures into secondary seizures from a known cause.

Epilepsy usually starts in early childhood.

Optimal management includes adequate psychosocial support with education, counselling, advocacy and appropriate referral. Advice about appropriate lifestyle using the NEAT approach (see [CHAPTER 5](#)) is important.

An underlying organic lesion becomes more common in epilepsy presenting for the first time in adults over the age of 25 years, and thus more detailed investigation is required.<sup>9</sup>

Secondary causes of seizures include:

- intracerebral tumours
- trauma—head injuries
- postcerebral surgery
- metabolic (e.g. calcium and sodium electrolyte disturbances, hypoglycaemia, uraemia, hepatic failure)
- drugs and other toxins (e.g. alcohol, amphetamines including withdrawal)
- intracerebral infections (e.g. meningitis, cysticercosis-specific refugees)
- autoimmune encephalitis ([CHAPTER 20](#))
- vascular—cerebrovascular, arteritis, hypertension

- eclampsia
- MELAS syndrome ([CHAPTER 23](#))
- hypoxia
- degenerative diseases
- sleep deprivation
- prescribed drugs, including antidepressants (e.g. SSRIs, TCAs, venlafaxine, mirtazapine), antibiotics (e.g. norfloxacin, ganciclovir, isoniazid), amantadine, antipsychotics, chloroquine, interferons, pizotifen, promethazine, theophylline

## Types of epileptic seizures/syndromes

Epileptic seizures are classified in general terms as generalised and partial (see [TABLE 43.5](#) ). Some others are unclassifiable. Partial seizures are about twice as common as generalised seizures and are usually due to acquired pathology.<sup>12</sup>

**Table 43.5** Classification of epileptic seizures<sup>12</sup>

### 1 Generalised seizures

Motor—convulsive:

- tonic–clonic (previously called grand mal)
- clonic
- myoclonic
- secondary generalised

Motor—non-convulsive:

- tonic (drop attacks)
- atonic (drop attacks)

Non-motor (absence):

- typical absence—childhood (petit mal) and juvenile
- atypical absence
- eyelid myoclonia

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### 2 Focal (partial) seizures

Simple partial (consciousness retained):

- with motor signs (Jacksonian)
- with somatosensory symptoms

- with psychic symptoms

Complex partial (consciousness impaired)

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## Generalised seizures

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Generalised seizures arise in both cerebral hemispheres simultaneously from the outset. The seizure may be primary with generalised onset (no focus), or secondary, which starts focally and spreads to become secondary and may be due to acquired cerebral pathology.

The main features are:

- abrupt impairment or loss of consciousness
- possible bilateral symmetrical motor events

## Types

- Tonic–clonic (formerly called grand mal) seizure: this is the classic convulsive seizure with muscle jerking
- Tonic seizure: stiffness only, often with a ‘drop’ (hallmark of Lennox–Gastaut syndrome)
- Clonic seizure: jerks only
- Atonic seizure: loss of tone, and ‘drops’
- Absence seizure (formerly called petit mal): involves loss of consciousness with no or only very minor bilateral muscle jerking, mainly of the face<sup>12</sup>
- Myoclonic seizure: bilateral discrete muscle jerks, which may be very severe, and loss of consciousness

### ⌚ Tonic–clonic seizures

The classic seizure sequence: aura—cry—fall—fit (clonic then tonic)—incontinence.

Variants of tonic–clonic seizures are more common than realised. Some individuals may simply stiffen or drop to the ground while others may have one or two jerks or shakes only:

- stiffen and fall = tonic
- floppy and fall = atonic
- shaking only = clonic

The typical features (in sequential order) of a tonic–clonic convulsion are:<sup>12</sup>

- aura (sensory or psychological feelings)
- initial rigid tonic phase (up to 60 seconds)
- convulsion (clonic phase) (seconds to minutes)
- mild coma or drowsiness (15 minutes to several hours)—postictal confusion

Associated features:

- cyanosis, then heavy ‘snoring’ breathing
- eyes rolling ‘back into head’
- ± tongue biting
- ± incontinence of urine or faeces

It should be noted that sphincter incontinence is not firmly diagnostic of epilepsy. In less severe episodes the person may fall without observable twitching of the limbs.<sup>12</sup>

In atonic epilepsy, which occurs in those with tonic–clonic epilepsy, the person falls to the ground and is unconscious for only a brief period.

## Diagnosis

- Check short-term aggravating factors (e.g. lack of sleep, medications, drugs inc. alcohol)
- Investigate and treat any cause. Usual tests—EEG, CT or MRI (preferable) scan, basic biochemistry and haematology

## Management

*Note:* Do not usually treat on one fit. Refer to FIGURE 43.1 .

- Profound psychosocial support
- Education, counselling, advocacy
- Appropriate referral

## Medication

- sodium valproate (first choice for tonic–clonic)

- adults: 500 mg (o)/d for 1 wk, then bd for 1 wk ↑ every 2–4 wks to achieve control (up to 2–3 g/d)

Some prefer carbamazepine or lamotrigine in young women because of risk of teratogenicity with valproate, which, however, is less sedating.

- Carbamazepine (second choice)
- Other choices: phenytoin, lamotrigine, topiramate, levetiracetam (usually added on to achieve optimal control—check interactions)

Continue treatment until fit-free for at least 2 years. Avoid use of prochlorperazine and benzodiazepines. Monitor with annual LFTs and FBE.

The management of status epilepticus is presented in [CHAPTER 120](#).

## Absence seizure

This type of generalised epilepsy typically affects children from 4 years up to puberty:<sup>2</sup>

- child ceases activity and stares suddenly
- child is motionless (may blink or nod)
- no warning
- sometimes clonic (jerky) movement of eyelids, face, fingers
- may be lip-smacking or chewing (called complex absence)
- only lasts few seconds—usually 5–10 seconds
- child then carries on as though nothing happened
- usually several per day (not just one or two)
- may lead to generalised seizures in adulthood
- two types—childhood and juvenile

## Diagnosis

Best evoked in the consulting room by hyperventilation (child—blow a toy windmill).

EEG:

- classic 3 Hz wave and spike

- may be normal
- always include hyperventilation
- easier with sleep deprivation

## Medication

ethosuximide (first choice)<sup>5,13</sup>

*or*

sodium valproate (second choice)

*or* (others) e.g. clonazepam, gabapentin

Childhood absence may not need pharmacotherapy.

*Note:* Beware of hepatotoxicity with sodium valproate, especially in those under 2 years.

## The first seizure

The decision to treat the first seizure with medication is based on several factors. Since 50% of patients will never have another seizure, treatment is not usually recommended. However, if two or more seizures occur over more than 24 hours, patients are likely to experience more and require treatment. The risk is increased in specific types, such as partial (focal) seizures, those with ECG abnormalities and a lesion on neuroimaging.<sup>5,14</sup>

## Partial seizures

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In partial seizures the epileptic discharge begins in a localised focus of the brain and then spreads out from this focus. The clinical pattern depends on the part of the brain affected:

- simple partial seizures: consciousness is retained
- complex partial seizures: consciousness is clouded so that the patient does not recall the complete seizure

Both these types of partial seizure can evolve into a bilateral tonic–clonic seizure; this is termed a secondary generalised seizure and is usually due to diffuse brain pathology.<sup>9</sup>

## Investigations

Standard minimum investigations are:

- serum calcium, magnesium and electrolytes

- fasting glucose
- EEG (usually with sleep deprivation)
- syphilis serology

Other tests may include:

- chest X-ray
- ECG (?↑ QT interval)
- video EEG (limited mainly to frequent seizures or to diagnostic dilemmas)
- MRI
- CT scanning (if MRI unavailable)
- other cerebral imaging (e.g. SPECT and PET)

A person presenting with the first seizure after the age of 25 years will require more detailed investigation.

## Complex partial seizures

In complex partial seizures (known also as temporal lobe epilepsy) the symptomatology varies considerably from patient to patient and is often a diagnostic problem. It is the commonest type of focal epilepsy and the attacks vary in time from momentary to several minutes (usually 1–3 minutes).

### Possible manifestations<sup>2</sup>

- Commonest: slight disturbance of perception and consciousness
- Hallucinations: visual, taste, smell, sounds
- Absence attacks or vertigo
- Illusions—objects/people shrink or expand
- Affective feelings—fear, anxiety, anger
- Dyscognitive effects: *déjà vu* (familiarity), *jamais vu* (unreality), waves emanating from epigastrium
- Objective signs: lip-smacking, swallowing/chewing/sucking, unresponsive to commands or questions, pacing around a room

- Postictal drowsiness

Unreal or detached feelings are common in complex partial seizures. There can be permanent short-term memory loss. The sensation of strange smells or tastes is more common than auditory or visual hallucinations.<sup>1</sup> They can progress to tonic–clonic seizures.

## Diagnosis

- EEG is diagnostic in 50–60% of cases; a repeat EEG will increase rate to 60–80%
- EEG/video telemetry helpful with frequent attacks
- CT or MRI scan—to exclude tumour when diagnosis confirmed

## Medication

carbamazepine (first choice):<sup>5,9</sup> children and adults

or

sodium valproate (second choice): not <2 years

or (others)

vigabatrin, phenytoin, phenobarbitone, tiagabine, benzodiazepines, ethosuximide, levetiracetam

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## Simple partial seizures

In simple partial seizures (Jacksonian epilepsy) there is no loss of consciousness. These include focal seizures, which may proceed to a generalised tonic–clonic seizure or to motor seizures.

### Jacksonian (motor seizure)

Typically, jerking movements begin at the angle of the mouth or in the thumb and index finger and ‘march’ to involve the rest of the body (e.g. thumb → hand → limb → face ± leg on one side and then on to the contralateral side). A tonic–clonic or complex partial seizure may follow.

## Medication

carbamazepine (first choice):<sup>5</sup> children and adults

or

sodium valproate (second choice) not <2 years

*or (others)*

phenytoin, vigabatrin, gabapentin

## The single unprovoked seizure: to treat or not<sup>15</sup>

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Starting drugs after a single afebrile seizure can be a difficult decision. It basically depends on the EEG findings and also on whether the impact of further seizures outweighs the risk of treatment. If not treated, the risk of recurrence over a 3-year period is 40% with a risk of approximately 25% if treated.<sup>5</sup> The lowest recurrence rates are associated with a normal EEG. Drug therapy should be offered to the patient following two or more seizures within 6–12 months except when there is a clear avoidable precipitant.

## Approaches to management

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- Shared care between GP and consultant is optimal management.
- An accurate diagnosis of the seizure type is essential.
- An underlying brain disease has to be investigated and treated.
- Treatment is based on drugs and lifestyle management. Alcohol abstinence is preferable.
- Aim for monotherapy.
- A decision has to be made about whether drug therapy is appropriate. Pharmacotherapy should be offered if the patient has had two or more seizures within 6–12 months.<sup>5,16</sup> Treatment is usually commenced after the second seizure. Most seizures require long-term anti-epileptic (anticonvulsant) drug therapy aimed at suppressing the underlying seizure activity in the hope that it may subside, so that ‘cure’ ultimately occurs and treatment may be ceased. A summary of anti-epileptic drugs is presented in TABLE 43.6 .
- The choice of drug depends on the seizure type, the age and sex of the patient, and efficacy in relation to toxicity.
- Treatment should be initiated with one drug and pushed until it controls the events or causes side effects, irrespective of the medication blood level. The disorder can usually be controlled by one drug, provided adequate serum or plasma concentrations are reached.<sup>16</sup> Seventy to eighty per cent of people will have no seizures after treatment with a first-line drug.
- If a maximum tolerated dosage of this single drug fails to control the seizures, replace it with an alternative agent with a different action. Add the second drug and obtain a therapeutic effect before removing the first drug.
- An example of an initial drug regimen in a young man with idiopathic generalised tonic-clonic seizures is: valproate 500 mg (o) daily for 2 weeks, then bd, up to 3 g or more per day.

*If not controlled, second-line (add on): lamotrigine 12.5 mg (o) once daily for 2 weeks, increasing up to 100 mg.*

- Pay special attention to the adverse psychological and social effects of epilepsy. Emotional and social support is important and advice about epilepsy support groups is appropriate.

**Table 43.6** Anti-epileptic drugs

The following anti-epileptic drugs are used

Benzodiazepines:

- clobazam
- clonazepam
- diazepam
- midazolam
- nitrazepam

Carbamazepine

Tetacosactrin

Phenobarbitone and related drugs:

- methylphenobarbitone
- primidone

Phenytoin

Sodium valproate (valproate)—avoid in reproductive phase. If necessary, ensure adequate contraception.

Succinimides:

- ethosuximide

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**Newer drugs (mainly as 'added on' therapy)**

Gabapentin

Lacosamide

Lamotrigine

Levetiracetam

Oxcarbazepine

Perampanel

Pregabalin

Sulthiame

Tiagabine

Topiramate

Vigabatrin

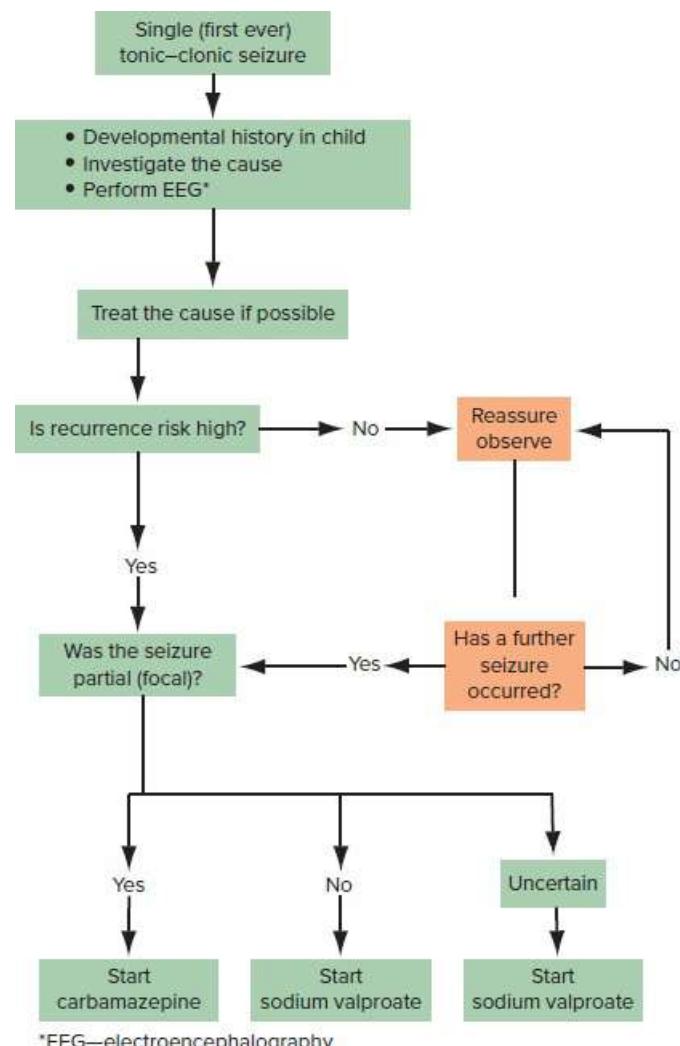
Zonisamide

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Cannabinoids (evidence is emerging, e.g. Dravet syndrome)

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A flow chart for the initial management of epilepsy is presented in [FIGURE 43.1](#) .



**FIGURE 43.1** Initial management of epilepsy

Source: Adapted with permission from Choosing an antiepileptic drug [published 2017 Nov; amended 2021 Mar]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2015 Mar. [www.tg.org.au](http://www.tg.org.au)

## Cessation of drug therapy

It is important to review the need for anti-epileptics every 12 months. The only way to find out if drug therapy is still needed is to withdraw it. It may be ceased if the patient has been free of seizures for at least 2–3 years, particularly if epileptiform activity has disappeared from the EEG (best under specialist supervision). Up to 60% of children have a mild, self-limiting condition and can settle after medication is withdrawn. It is usual to wean children off drugs after 2–3 years (seizure free) and advise about buccal midazolam in the event of a seizure.

## Patient education

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The following points are worth emphasising.

- Most patients can achieve complete control of seizures, but medication compliance is essential.
- Most people lead a normal life—they can expect to marry, have a normal sexual life and have normal children.
- Patients need good dental care, especially if taking phenytoin.
- A seizure in itself will not usually cause death or brain damage unless prolonged or in a risk situation such as swimming.
- Patients cannot swallow their tongue during a seizure.
- Take special care with open fires.
- Encourage patients to cease intake of alcohol. Intoxication is very harmful.
- Adequate sleep is important. Deprivation is harmful.
- Avoid fatigue.
- Advise showering in preference to bathing.

## Driving

One has to be very careful about driving. Most people with epilepsy can drive but each case has to be considered individually. Applicants for a learner's licence need to be seizure-free for 2 years (varies between states/territories), with an annual medical review for 5 years following receipt of the licence. Restrictions range from 1 month to 2 years, depending on the circumstances of the seizures. For a new patient the usual rule is suspension of driving until 3–6 months seizure free.<sup>17</sup>

## Employment

People with epilepsy can hold down most jobs, but if liable to seizures they should not

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work close to heavy machinery, in dangerous surroundings, at heights (such as climbing ladders) or near deep water. Careers are not available in some services, such as the police, military, aviation (pilot, traffic controller) or public transport (e.g. bus driver).

## Sport and leisure activities

Most activities are fine, but people with epilepsy should avoid dangerous sports such as scuba diving, hang-gliding, parachuting, rock climbing, car racing and swimming alone, especially surfing.

**TABLE 43.7** outlines contraindications for sporting activities. These apply to patients who suffer from very frequent seizures, especially the complex partial seizures with prolonged postictal states.<sup>16</sup>

**Table 43.7** Sporting activities: contraindications<sup>18</sup>

### Absolute contraindications

- Flying and parachuting
- Motor racing
- Mountain and rock-climbing
- High diving
- Scuba diving
- Underwater swimming, especially competitive
- Hang-gliding
- Abseiling

### Relative contraindications

- Aiming sports such as archery and pistol shooting
- Contact sports such as boxing, rugby, football, including soccer, where heading the ball is involved
- Competitive cycling for children with absence epilepsy
- Bathing and swimming
- Gymnastics, especially activities such as trampolining and climbing on bars
- Ice skating and skiing
- Javelin throwing

## Avoid trigger factors

- Fatigue
- Lack of sleep
- Physical exhaustion
- Stress
- Excess alcohol
- Prolonged flashing lights if photosensitive (e.g. video games—this applies to those with a proven response to a proper EEG with photic stimulation)

## Epilepsy in children

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### Single (first) episode tonic–clonic seizure in school-aged child

1. Take a comprehensive history: family history, past history, developmental history, any loss of skills, recent head injury, headache, drowsiness, dizziness, recent school performance
2. Investigations:
  - biochemistry (glucose, electrolytes incl.  $\text{Ca}^{++}$ ,  $\text{Mg}^{++}$ )
  - ECG (?QT interval)
  - EEG (specialist input)
  - Imaging—brain CT or MRI
3. Management:
  - Reassure may be one-off event
  - Usually no medication
  - If recurs, start sodium valproate (see FIG. 43.1 )

### Photosensitive epilepsy in children

Some children suffer from photosensitive epilepsy related to exposure to computer and video games and 3D television. There is some evidence that such children may not have seizures if they keep one eye covered. If television provokes seizures, strategies such as watching it with ambient lighting and using the remote control rather than approaching the set will minimise the problem.

## Pregnancy and epilepsy<sup>16,19</sup>

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Pregnancy is associated with a 30% increase in seizures. Although the outcome is successful for more than 90% of women with epilepsy, there is a slightly increased risk of prematurity, low birthweight, mortality, defects and intervention. About 45% of women have an increased number of seizures, due mainly to a fall in anti-epileptic drug levels.

All anti-epileptic drugs are potentially teratogenic, with different drugs being related to different defects: phenytoin has been related to cleft lip and palate and congenital heart disease, while sodium valproate (in particular) and carbamazepine have been associated with spina bifida. Sodium valproate should be avoided unless other drugs are unlikely to prevent seizures. All anti-epileptic drugs are expressed in breast milk but in such reduced concentrations as not to preclude breastfeeding, although it is preferable for mothers taking lamotrigine and levetiracetam to abstain from breastfeeding.<sup>20</sup>

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## Neurosurgical treatment

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Surgical techniques are based on resection such as for temporal lobe epilepsy for a highly select group of patients and also on disconnection techniques. The latter includes corpus callosotomy, multiple subpial transection, hemispherectomy and vagal nerve stimulation. The treatments are limited to a select group with poorly controlled seizures who require detailed evaluation in a specialist centre.

## Pitfalls in management of epilepsy<sup>5</sup>

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### Misdiagnosis

The main pitfall associated with seizure disorders and epilepsy is misdiagnosis. It should be realised that not all seizures are generalised tonic–clonic in type. The most common misdiagnosed seizure disorders are complex partial seizures (an underdiagnosed disorder) or the tonic or atonic seizures.

The diagnosis of epilepsy is made on the history rather than the EEG so a very detailed description of the events from eyewitnesses is important.

The features of complex partial seizure (described in earlier in the chapter) have many variations, the commonest being a slight disturbance of perception or consciousness. The complex partial seizure may evolve to a generalised tonic–clonic seizure. A simple partial seizure may also do this.

In tonic–clonic seizures the patient may become momentarily rigid or fall to the ground and perhaps have one or two jerks only.

## Misdiagnosing behavioural disorders

It is important to differentiate between a fit and a behavioural disorder, but it can be difficult. About 20% of apparently intractable ‘seizures’ are considered to be non-epileptic (pseudoseizures, i.e. emotionally based).<sup>20</sup> These often resemble tonic–clonic seizures but usually there are bizarre limb movements. Ancillary testing, especially with video EEG recording, can help overcome these diagnostic problems but the differentiation may be difficult as the most common situation for pseudoseizures is in the person who has real fits.

## Overtreatment

### Polypharmacy

Polypharmacy may be counterproductive for the patient and the seizure disorder. This is especially applicable to drugs with a high incidence of side effects. If a patient is taking several medications, management of the case needs questioning and perhaps reconsidering with a consultant’s help.

Seizure control may be improved by reducing polypharmacy. When initiating treatment it is best to select one drug and increase its dose until its maximum recommended level, the onset of side effects or appropriate control. If control is not obtained, the drug should be replaced with an alternative agent but a crossover period is essential. Monotherapy is preferred but combination therapy is often acceptable.

### Prolonged treatment

The question should be asked at some stage ‘Does this patient really need medication?’ Some patients are kept on anti-epileptics for too long without any attempt being made to wean them off medication or to transfer them onto anti-epileptics less prone to side effects. Patients should not be left on inappropriate drugs, especially if side effects and drug interactions are a problem.

### Drug interactions

Drug interactions with anti-epileptics should always be kept in mind. The most serious of all is the interaction with the oral contraceptive pill because pregnancy can occur. Erythromycin and carbamazepine interact.

## Narcolepsy

Narcolepsy is characterised by brief spells of irresistible sleep during daytime hours, usually in inappropriate circumstances, even during activity. Its cause is unknown. Although patients are usually aware of their disorder, some may have no insight into the problem and present with the complaint of unusual turns. Narcolepsy can present as ‘attacks’ in which the patient may crumple and fall without losing consciousness. It can be part of a tetrad syndrome (daytime hypersomnolence, cataplexy, hypnagogic and hypnopompic hallucinations, sleep paralysis).

Other features:

- onset in teens or 20s
- can have several attacks per day

Refer to [CHAPTER 60](#).

## Diagnosis

- A clinical diagnosis

If doubtful:

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

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## Medication<sup>21</sup>

dexamphetamine (in slowly increasing doses to 60 mg daily)

*or*

methylphenidate (immediate release) up to 60 mg daily

*or*

modafinil 200–400 mg (o) daily

*or*

tricyclic antidepressants (e.g. clomipramine, imipramine, fluoxetine), for associated cataplexy

## Amnesic episodes

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Amnesic episodes in which people cannot recall events or their own identity can be psychogenic (commonly), such as fugue states, conversion disorder, severe depression and factitious states. They may be related to an organic problem such as epilepsy, sleep apnoea, cerebrovascular disorder, post-trauma, Wernicke–Korsakoff syndromes and drugs (e.g. alcohol, cannabis and anaesthetic agents).

### ⌚ Transient global amnesia<sup>5,22,23</sup>

This is a benign, self-limited profound amnesic episode of unknown aetiology tending to occur

in middle-aged or elderly people. Proposed causation includes transient ischaemia or dysfunction of the temporal lobes, similar to temporal lobe epilepsy or a migraine variant.

## Clinical features

- Typically last 4–8 hours (up to 24 hours)
- Identity and conscious state preserved
- Agitated, perplexed, anxious
- State of bewilderment (e.g. ‘where am I?’)
- Anterograde and retrograde amnesia
- Frequent repetition of questions
- Able to perform complex motor skills (e.g. driving)
- Usually single episode (20% recurrence)
- Complete resolution: good prognosis
- No abnormal neurological signs
- All investigations unhelpful

## Four diagnostic criteria (Caplan)<sup>14</sup>

- Witnessed onset of attack (essential for diagnosis)
- Dysfunction during attack limited to amnesia and repetitive queries
- No other neurological features
- Memory loss should be transient, lasting no longer than 24 hours

## Management

- Reassuring explanation/education
- Special investigations, including angiography, not needed if conform to above criteria
- No active treatment usually needed

## Cerebrovascular disorders

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Cerebrovascular disease is one of the major causes of mortality and morbidity in developed countries and can cause recurrent attacks of ischaemia in the carotid and vertebrobasilar systems (particularly vertebrobasilar insufficiency), which may present as ‘funny turns’. In particular, brain-stem ischaemia causes ‘funny turns’ such as impaired consciousness, including transient global amnesia, drop attacks and the ‘locked-in’ syndrome.

## Orthostatic intolerance and syncope

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There are three main syndromes of autonomic instability with orthostatic intolerance leading to syncope.

1. *Reflex (vasovagal) syncope*. This affects 30% of the population, has a strong family history and an onset in the young. There is a hypotensive response. The multiple precipitants include coughing, micturition, fright, standing, heat and defecation (straining). Although consciousness quickly returns, recovery can be delayed (e.g. malaise for 12–24 hours). See [CHAPTER 64](#).
2. *Postural orthostatic tachycardia syndrome (POTS)*.<sup>24</sup> This is orthostatic intolerance with dysautonomia upon changing from the supine to the upright position or head up tilt. Tachycardia with decreased ventricular filling is a feature with hypotension and possibly syncope. There are myriad symptoms including dizziness, fatigue, blurred vision, chest pain and cognitive impairment. It is sometimes found while investigating chronic fatigue syndrome. It has an adolescent onset and a familial history. Referral to a ‘syncope’ unit is recommended for this complex and debilitating condition. One treatment option is fludrocortisone acetate. Another is the vasopressor/hypertensive agent midodrine to treat dysautonomia and orthostatic hypotension.
3. *Autonomic failure*. This is age related and can be primary (e.g. multiple system atrophy) or secondary (e.g. diabetes, amyloid). It leads to hypoperfusion syncope. Precipitants include orthostasis, meals and alcohol. Recovery from syncope is rapid.

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## Psychogenic or communication disorders

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Psychogenic causes have to be considered. ‘Hysterical fugue’ is one such manifestation. The problem can be a communication disorder, such as an emotional person trying to communicate a problem in a language foreign to them.

Patients with psychiatric disorders such as schizophrenia or depression may experience feelings of depersonalisation or unreality, which can be interpreted as a ‘turn’ or even temporal lobe epilepsy.

Patients who complain of vague and bizarre symptoms, such as ‘queer feelings in the head’, ‘swimming sensation’, ‘unreal feelings’ and ‘walking on air’ quite possibly have an anxiety state.

Severe anxiety or panic attacks typically cause lightheadedness that presents as a ‘funny turn’. Other somatic symptoms include palpitations, sweating, inability to swallow, headache, breathlessness and manifestations of hyperventilation.

## Pitfalls in management<sup>2</sup>

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- The main pitfall associated with seizure disorders and epilepsy is failure to diagnose, particularly seizures not obviously generalised tonic–clonic.
- Failing to place appropriate emphasis on the history in making the diagnosis.
- Misdiagnosing syncope with some involuntary movements as epilepsy.
- Overlooking cardiac arrhythmias as a cause of funny turns, including recurrent dizziness.
- Failing to consider the possibility of aortic stenosis with syncopal attacks.
- Misdiagnosing vertigo and syncope as TIA.
- Mistaking visual or sensory migraine equivalents in young adults for TIA.
- Overlooking drugs (including self-administered drugs) as a cause of lightheadedness.

## When to refer<sup>12</sup>

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- Transient ischaemic attacks, especially if the diagnosis is in doubt
- Clinical suspicion of or proven cardiac arrhythmias
- Evidence of aortic stenosis
- Seizures
- General uncertainty of the diagnosis

### Practice tips

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- A detailed clinical analysis is more important in the first instance than laboratory tests. The key to accurate diagnosis is a very careful history, taking the person second-by-second through the attack and events preceding the turn.
- Talk to as many eyewitnesses as possible in unravelling the cause.
- For ‘undiagnosed turns’, ask the patient to keep a diary with an accurate record of the attack, including preceding events.

- Remember that migraine is a great mimic and can cause confusion in diagnosis.
- Remember that the EEG can be normal in the confirmed epileptic.
- The more bizarre the description of a ‘funny turn’, the more likely a functional problem is the cause.
- Transient hypoglycaemia can mimic a TIA.

## Epilepsy

Specialist referral is advisable under the following circumstances:

- uncertainty of diagnosis
- at onset of seizure disorder to help obtain a precise diagnosis
- when seizures are not controlled by apparently suitable therapy. ?wrong drug, ?suboptimal dose, ?progressive underlying disorder
- when the patient is unwell, irrespective of laboratory investigation
- when a woman is considering pregnancy (preferable) or has become pregnant: to obtain therapeutic guidance during a difficult phase of management
- for assessment of the prospects for withdrawing treatment after some years of absolute seizure control

## Patient education resources

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

- Epilepsy
- Fainting (syncope)

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## 44 Haematemesis and melaena

*The ‘once smelt never forgotten’ sickly smell of melaena can be diagnosed from a distance of 20 metres without trying.*

EMERGENCY ROOM SUPERVISOR, BRISBANE 1985

Acute severe upper GI haemorrhage is an important medical emergency. The dramatic symptom of haematemesis follows bleeding from the oesophagus, stomach and duodenum. More than half the patients are over 60 years of age.<sup>1</sup>

Haematemesis is the vomiting of blood appearing as fresh blood or ‘coffee grounds’. Melaena is the passage of black tarry stools, with 50 mL or more of blood required to produce melaena stool (Greek *melas* = black). Melaena occurs in most patients with upper GI haemorrhage and haematemesis occurs in over 50%.<sup>1</sup>

Although the incidence is declining, the mortality rate of upper GI haemorrhage remains high at approximately 6–8%.<sup>2</sup>

### Key facts and checkpoints

- Chronic peptic ulceration accounts for most cases of upper GI haemorrhage.
- Haematemesis is almost always associated with some degree of blood in the stools, although melaena may not necessarily accompany it, especially if bleeding occurs from the oesophagus.
- Black stool caused by oral iron therapy or bismuth-containing antacid tablets can cause confusion.
- Always check for a history of drug intake, especially aspirin and NSAIDs.
- Corticosteroids in conventional therapeutic doses are thought to have no influence on GI haemorrhage.
- The volume of bleeding is best assessed by its haemodynamic effects rather than relying on the patient’s estimation, which tends to be excessive.

- Melaena is generally less life-threatening than haematemesis.
- Resuscitation of the patient is the first task.
- A sudden loss of 20% or more of circulatory blood volume usually produces signs of haemorrhagic shock such as tachycardia, hypotension, faintness, syncope and sweating. Younger people can compensate better and tolerate a larger loss prior to the development of shock.<sup>1</sup> A useful guide is that shock in a previously well 70 kg male indicates an acute blood loss of at least 1000–1500 mL.

## Causes of upper GI bleeding

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The major cause of bleeding is chronic peptic ulceration of the duodenum and stomach, accounting for about half of all cases.<sup>3,4</sup> The other major cause is acute gastric ulcers and erosions, which account for at least 20% of cases. Aspirin and NSAIDs are responsible for many of these bleeds. Causes are summarised in [TABLE 44.1](#) and illustrated in [FIGURE 44.1](#).

**Table 44.1** Diagnostic strategy model for upper gastrointestinal bleeding (haematemesis)<sup>5,6</sup>

### Probability diagnosis

- Chronic peptic ulcer (stomach and duodenum)
- Acute gastric ulcers/erosions
- Oesophagitis (incl. GORD)/duodenitis
- Mallory–Weiss (emetogenic) syndrome
- Drugs: aspirin, NSAIDs, anticoagulants, clopidogrel
- No obvious cause

### Serious disorders not to be missed

#### Vascular:

- oesophageal varices
- blood dyscrasias
- vascular malformation/angiodyplasia
- hereditary coagulopathy; drugs

#### Cancer:

- gastric or oesophageal

#### Other:

- chronic liver disease

### Pitfalls (often missed)

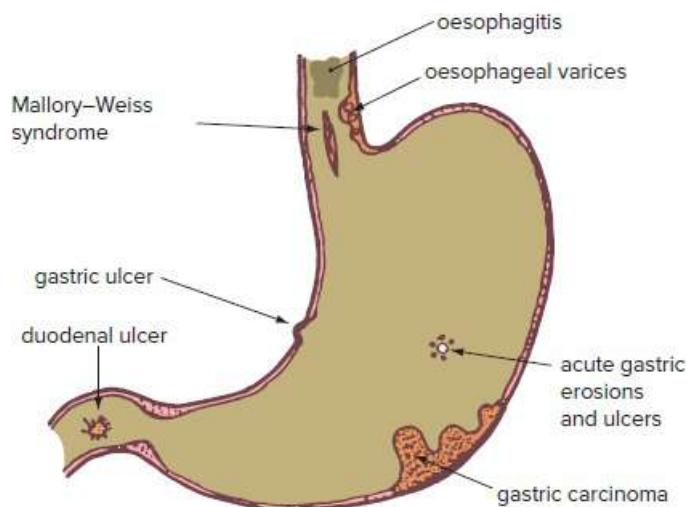
Stomach ulcer

Swallowed blood (e.g. epistaxis)

Collagen diseases (e.g. scleroderma)

### Rarities:

- ruptured oesophagus
- hereditary haemorrhagic telangiectasia
- scurvy
- ingested poisons (e.g. acid, alkali, arsenic)
- gastric antral vascular ectasia



**FIGURE 44.1** Important causes of haematemesis and melaena

## Mallory-Weiss syndrome

In this condition a tear occurs at the lower end of the oesophageal mucosa (at the oesophagogastric junction) because of an episode of severe or protracted vomiting or coughing. Blood appears in the vomitus after a bout of heavy vomiting or dry retching. It is usually seen in alcoholic addiction, and is usually a self-limiting lesion. A definite diagnosis can only be made by endoscopy.

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## Gastro-oesophageal varices

Such varices are caused by portal hypertension, which in turn is usually due to cirrhosis of the liver. There is a raised incidence of peptic ulcer in those with liver cirrhosis, especially in biliary and alcohol-induced cirrhosis, so this should be kept in mind as a possible source of bleeding.

Mortality is about 30%, despite advances, and 70% for untreated patients.<sup>6</sup>

Primary prevention of bleeding from varices is with the use of beta blockers such as propranolol if no contraindications.<sup>7</sup> There is a high bleeding rate.

Management includes injection sclerotherapy, and then intravenous octreotide or terlipressin if it fails. Passage of a Sengstaken–Blakemore or Minnesota tube into the oesophagus and stomach to provide tamponade and the radiological procedure of using a transjugular intrahepatic portosystemic stent are possible options.

## The clinical approach

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### History

It is important to establish the nature of the vomitus and the possibility of bleeding arising from the mouth, nose or pharynx. A coffee grounds vomitus indicates that the blood has been in contact with gastric acid. Oesophageal bleeding tends to lead to vomiting of fresh blood. Patients may confuse vomiting up and coughing up blood.

### Key questions

- What drugs have you been taking? (see TABLE 44.2 )
- Have you been taking aspirin or tablets for arthritis or back pain?
- How much have you vomited?
- What did the vomit look like?
- Did you notice black dots like coffee grounds or any blood clots?
- Have you had any indigestion, heartburn or stomach pains recently?
- Have you lost any weight unexpectedly?
- Have you opened your bowels and if so was it black or unusual in any way?
- How much alcohol do you drink?
- Have you had any previous operations on your stomach for a peptic ulcer?
- Were you vomiting normal vomit before the blood appeared?
- Have you been eating beetroot or other bright red foodstuffs?

**Table 44.2** Drugs associated with gastrointestinal

## haemorrhage<sup>2</sup>

Aspirin  
Clopidogrel  
Other antiplatelet drugs  
Heparin/new oral anticoagulants  
NSAIDs/COX-2 inhibitors  
Prednisolone  
SSRI antidepressants  
Warfarin

## Examination

The patient's general state, particularly the circulation, should be assessed immediately on presentation. It is critical to assess haemodynamic status with vital signs of heart rate, blood pressure and postural changes.<sup>2</sup> A careful abdominal examination should be performed including a digital rectal examination. As a rule abdominal findings are not remarkable except when a mass, hepatomegaly or splenomegaly is found. Other evidence of liver disease should be sought.

## Investigations

Investigations to determine the source of the bleeding should be carried out in a specialist unit. Upper gastrointestinal endoscopy is the single most useful test and will detect the cause of the bleeding in at least 80% of cases.<sup>3</sup> Sometimes no visible cause can be identified.

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The haemoglobin level will not be an appropriate guide to blood loss or the need for transfusion during the early stages because haemodilution occurs gradually over the 24 hours following a severe bleed. However, a level below 90 g/L during this period is usually regarded as an indication for transfusion.

Other tests to consider include FBE, *H. pylori*, LFTs, plain abdominal X-ray, coagulation profile.

## Management

The immediate objectives are:

1. stabilise patient; restore an effective blood volume (if necessary)
2. establish a diagnosis to allow definitive treatment

All patients with a significant bleed should be admitted to hospital and referred to a specialist unit. Urgent resuscitation is required where there has been a large bleed and there are clinical

signs of shock. Such patients require insertion of intravenous lines and rapid infusion of isotonic saline followed by a plasma expander (e.g. Haemaccel) followed by transfusion with blood commenced as soon as possible.

Proton-pump inhibitors should be commenced in most cases especially as 50% of bleeding is from peptic ulceration. Oral administration may be possible in most cases but intravenous PPI (e.g. omeprazole 80 mg over 15–30 minutes followed by IV infusion for up to 3 days or pantoprazole IV)<sup>7,8</sup> is appropriate for the seriously ill.<sup>4</sup> Eventually switch to oral PPIs.

In many patients bleeding is insufficient to decompensate the circulatory system and they settle spontaneously. Approximately 85% stop bleeding within 48 hours.<sup>3</sup>

In some instances intervention via upper endoscopy to achieve haemostasis of bleeding point with electrocautery, a heater probe (e.g. Gold Probe) or injection with adrenaline (or both), or application of clips will be employed. Occasionally surgery will be necessary to arrest bleeding but should be avoided if possible in patients with acute gastric erosion.

## Practice tips

- Ask the patient not to dispose of the evidence of vomitus, if possible.
- Patients with haematemesis should be assessed in the emergency department.
- Upper endoscopy is the gold standard for diagnosis and therapy for acute upper gastrointestinal bleeding.
- Many episodes of gastric bleeding stop spontaneously—possibly as high as 80% in one study.

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## 45 Headache

*When the head aches, all the body is out of tune.*

MIGUEL DE CERVANTES (1547–1616)

Headache, one of the cardinal symptoms known to human beings, is a very common complaint in general practice. When someone presents with ‘headache’ we need to have a sound diagnostic and management strategy as the problem can be confusing. The key to analysing the symptom of headache is to know and understand the cause, for ‘one only sees what they know’.

The patient’s manner of presentation can confuse us because many tend to influence us with preconceived ideas that they will verbalise—‘I think I need my blood pressure checked’ or ‘My eyes need testing’—or they may not mention their anxiety about a cerebral tumour or an impending stroke.

Hypertension is such a rare cause of headache that one is tempted to stress the adage ‘hypertension does not cause headache’, but we do encounter the occasional exception and it is mandatory (and expected by patients) to measure the blood pressure of anyone presenting with headache. However, where headaches and hypertension coexist, assume that the headaches are not due to hypertension.

The diagnosis of serious causes of headache depends on a careful history, a high index of suspicion of the ‘different’ presentations and the judicious use of CT scanning.

### Key facts and checkpoints

- Half to three quarters of adults aged 18–65 years in the world reported a headache in the last year, and of those, 30% reported a migraine.<sup>1</sup>
- Forty per cent of children will have experienced one or more headaches by the age of 7 and 75% by the age of 15.<sup>2</sup>
- Headache can be classified as primary or secondary, e.g. intracranial pathology, eyes, cervical spine, which requires urgent investigation.
- Migraine affects at least 10% of the adult population and one-quarter of these

patients require medical attention for their attacks at some stage.<sup>3</sup> It is under-recognised and poorly managed in the community.<sup>4</sup>

- Five per cent of children suffer from migraine by the age of 11 years.<sup>3</sup>
- Seventy per cent of sufferers have a positive family history of migraine.
- Before diagnosing tension headache, consider underlying disorders of the neck, eyes, teeth, temporomandibular joints or other structures.<sup>3</sup>
- Drug-induced headaches are common and must be considered in the history.
- In children the triad of symptoms—dizziness, headache and vomiting—indicates medulloblastoma of the posterior fossa until proved otherwise.
- A typical triad of symptoms in an adult with a cerebral tumour (advanced) is headache, vomiting and convulsions.
- Eye strain is not a common cause.
- Secondary bronchogenic bronchial carcinoma is the commonest cause of intracerebral malignancy.
- Primary headaches must be differentiated from secondary headaches.<sup>5</sup>

## A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 45.1 .

**Table 45.1** Headache: diagnostic strategy model

### Probability diagnosis

Acute: respiratory infection

Chronic:

- tension-type headache
- combination headache
- migraine
- chronic migraine (transformed migraine)

### Serious disorders not to be missed

Cerebrovascular:

- subarachnoid haemorrhage
- stroke/TIA
- intracranial haemorrhage
- carotid or vertebral artery dissection
- temporal arteritis
- cerebral venous thrombosis, e.g. venous sinus

Neoplasia:

- cerebral tumour
- pituitary tumour

Severe infections:

- meningitis, esp. fungal
- encephalitis
- intracranial abscess

Haematoma: extradural/subdural

Pressor response, e.g. phaeochromacytoma

Glaucoma (acute)

Benign intracranial hypertension

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### Pitfalls (often missed)

Cervical spondylosis/dysfunction

Reversible cerebral vasoconstriction syndrome

Dental disorders

Refractive errors of eye

Sinusitis

Ophthalmic herpes zoster (pre-eruption)

Exertional headache

Paroxysmal hemicrania

Hypoglycaemia

Post-traumatic headache

Post-spinal procedure (e.g. epidural, lumbar puncture)

Sleep apnoea

Rarities:

- Paget disease
- post-sexual intercourse
- Cushing syndrome
- Conn syndrome
- Addison disease
- dysautonomic cephalgia

### **Seven masquerades checklist**

Depression  
Diabetes  
Drugs  
Anaemia  
Thyroid/endocrine disorder  
Spinal dysfunction (cervicogenic/occipital neuralgia)  
UTI

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

Quite likely if there is an underlying psychogenic problem.

## **Probability diagnosis**

The commonest cause of headache presenting in general practice is respiratory infection.<sup>6</sup> The most common causes of chronic recurrent headache are chronic migraine, tension-type headache and combination headaches. Combination headaches, typified by relatively constant pain lasting for many days, have a mix of components such as tension, depression, cervical dysfunction, vascular headache and drug dependence. Neurologists may refer to these headaches as ‘tension-vascular headache’. Tension headache is less common than previously promulgated.<sup>3</sup>

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### **Chronic migraine<sup>7</sup>**

Previously called ‘transformed migraine’ because of the progressive increase in frequency of migraine attacks until the headache recurs daily. The typical migraine features become modified so that the pattern resembles that of tension headache but with the unilateral situation of migraine. Analgesic abuse can transform episodic migraine into chronic daily headache.

## **Serious disorders not to be missed**

For the acute onset of headache it is vital not to miss subarachnoid haemorrhage (SAH) or meningitis. Intracranial haemorrhage, especially involving cerebellar, intraventricular and frontal lobe areas, needs to be considered.

### **Acute ‘thunderclap’ headache<sup>7,8</sup>**

This is a sudden severe headache that can be caused by the following:

- enlarging aneurysm—an enlarging aneurysm or vascular malformation can cause acute headache
- SAH—the pain is typically occipital, localised at first then generalised and may vary in

intensity

- reversible cerebral vasoconstriction
- arterial dissection
- meningitis—must be considered if the headache is generalised, especially in the presence of malaise, fever and neck stiffness: the ache, which is constant and severe, may begin abruptly

For chronic headache, space-occupying lesions including subdural haematomas must be considered. Since headaches tend to decrease with age, headaches developing in the elderly should be viewed with suspicion and this includes considering temporal arteritis (TA). Idiopathic intracranial hypertension should be considered, especially in young obese women. The dangerous cryptococcal meningitis can be difficult as the CT scan may be normal.

### Tips on sinister causes of headache<sup>4</sup>

- The most important indicator is time course: beware of acute or subacute tempo.
- Be suspicious of any focal symptoms or signs (except for typical migraine aura).
- Beware of fever, confusion, altered mental state or neck stiffness.
- New onset headache, esp. sudden and first episode, patient >50 years, post trauma.

## Pitfalls

The list (see TABLE 45.1) contains some causes of headache that are hard to pin down, although some should be obvious if a careful history is elucidated. These include post-traumatic headache, postprocedural headache (e.g. lumbar puncture and spinal anaesthesia) and exertional headache. Sinusitis can be overlooked in the absence of respiratory signs. Refractive errors of the eye, although an uncommon cause of headache, do warrant consideration.

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### General pitfalls

- Overinvestigating the patient with headache, especially as a substitute for a careful history and examination
- Failing to appreciate that a combination of factors and cervical dysfunction are common causes of headache
- Omitting to measure the blood pressure in the person complaining of headache
- Rushing in with antibiotics for a patient (especially children) with fever and headache—bacterial meningitis may be masked