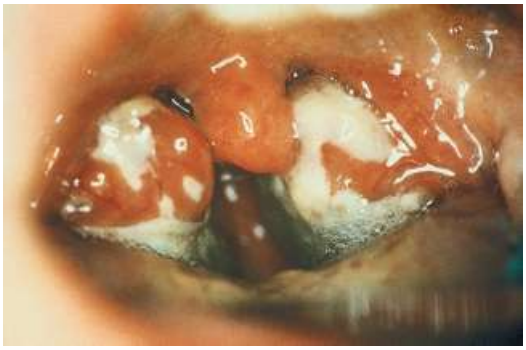

FIGURE 62.3 Tonsillitis of Epstein–Barr mononucleosis showing swollen red tonsils, with a whitish-yellow membranous exudate, swollen uvula and petechiae on the soft palate

Photo courtesy Hugh Newton-John

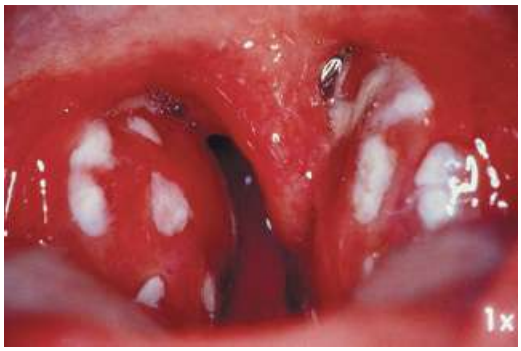

FIGURE 62.4 Acute follicular tonsillitis due to *Streptococcus pyogenes:* the tonsils are red and swollen with pockets of pus

Photo courtesy Hugh Newton-John

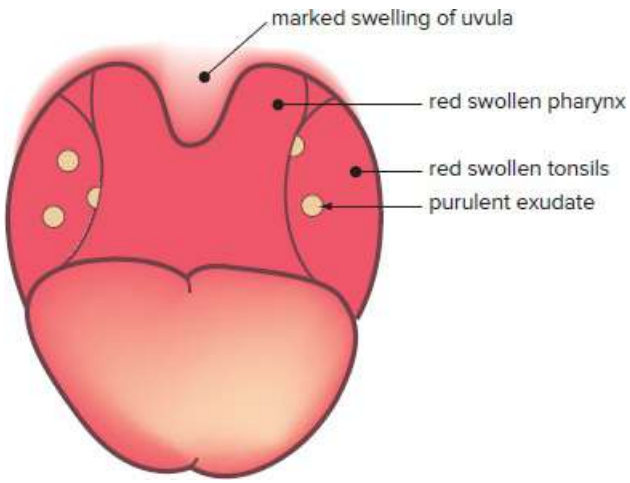

FIGURE 62.5 Streptococcal tonsillopharyngitis: severe inflammation involves both tonsils and pharynx with marked redness, swelling and exudate. Consider herpes simplex and mononucleosis as alternative diagnoses.

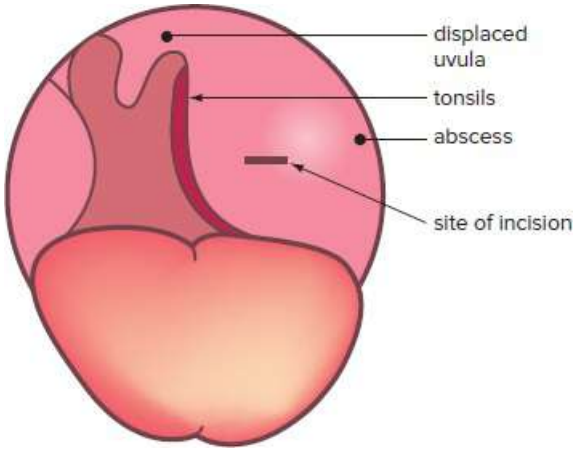

FIGURE 62.6 Peritonsillar abscess (quinsy): a tense red bulging mass is noted and the uvula is displaced from the mid line; a site of incision for drainage is indicated

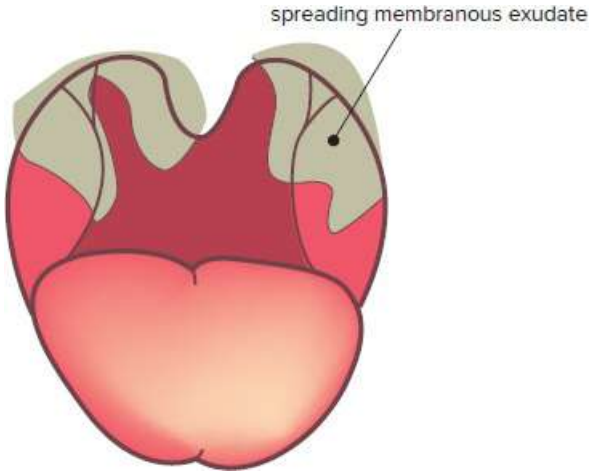

FIGURE 62.7 Diphtheria: tonsils and pharynx are red and swollen; a thick grey–green exudate forms on the tonsils as a spreading membrane

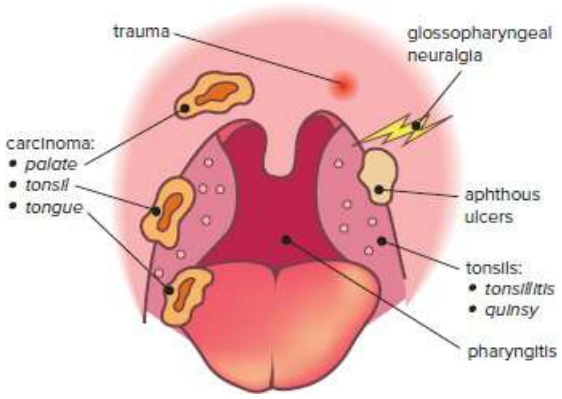

FIGURE 62.8 General causes of a sore throat: note the importance of excluding cancer

Guidelines

- Small patches of exudate on the palate or other structure indicate *Candida albicans* (oral thrush) (see FIG. 62.2).
- A large whitish-yellow membrane virtually covering both tonsils indicates EBM (see FIG. 62.3).
- A generalised red, swollen appearance with exudate indicates GABHS infection (see FIGS. 62.4 and 62.5).

Investigations

Investigations are usually not required, but can be selected from:

- throat swab
- haemoglobin, blood film and white cell count
- mononucleosis test
- random blood sugar (?diabetes)
- biopsy of suspicious lesions

To swab or not to swab

Throat swabs are about 90% effective in isolating GABHS from the infected throat. Authorities are divided about management. Some recommend that throat cultures be performed for all sore throats and antibiotics given only when GABHS is found. Others regard throat cultures as being unnecessary and recommend therapy based on clinical judgment. Swabs are

seldom helpful because the isolation of GABHS often represents asymptomatic carriage.[5] Still others recommend throat cultures for selected patients only.[6]

Generally, throat cultures are not necessary except to verify the presence of *S. pyogenes*, especially in closed institutions such as boarding schools, or if diphtheria is suspected in the non-immunised. One study has found that toothbrushes harbour GABHS and should not be shared.[7] A positive culture and a fourfold rise in the ASO titre are necessary for a precise diagnosis.

Epstein–Barr mononucleosis screening

It is important initially if tonsillar exudate is present to consider the possibility of EBM. If suspected, an IgM antibody test should be ordered, rather than the older tests, such as a Paul–Bunnell test.

Supportive symptomatic treatment for sore throats and common colds

Supportive measures can be useful for their modest benefit, and also as an alternative to the temptation to over-prescribe antibiotics.

- Adequate soothing fluids, including icy poles.
- Analgesia: adults—2 soluble aspirin; children—paracetamol elixir or ibuprofen.
- Commonly recommended are soothing gargles (e.g. soluble aspirin used for analgesia) and rest.
- OTC throat lozenges (Strepsils,[8] lignocaine[9]) and topical benzydamine spray (Difflam[10]) have weak evidence for modest, temporary pain reduction.
- For nasal congestion, the limited use (3 days) of decongestants is occasionally helpful.
- Oral zinc and vitamin C have weak evidence of some small benefit for common cold symptoms.
- Oral corticosteroids have been shown to decrease the intensity and duration of pain in those with severe pain, dysphagia and drooling. Use prednisolone (adult 50 mg daily; child 1 mg/kg daily) for 1–2 days, or a single dose of dexamethasone (adult 10 mg, child 0.6 mg/kg).[5]

Sore throat in children

An acute sore throat in a child usually means a viral or, less commonly, bacterial infection of the tonsillopharynx. A bacterial cause is more common in children aged 3–13 years than in children <3 years. Other causes to consider are:

- gingivostomatitis, especially primary herpes simplex

- epiglottitis
- laryngotracheobronchitis (croup)
- laryngitis
- oral candidiasis (more a bad taste than pain)
- aphthous ulcers
- foreign bodies
- postnasal drip (e.g. allergic rhinitis)
- irritation: low environmental humidity, smoke (e.g. household smoke)

Sore throat in the elderly

Sore throat in the elderly may be caused by a viral infection but otherwise needs to be treated with considerable respect. It is important to exclude pharyngeal cancer which can present with the classic triad.

DxT painful swallowing + referred ear pain + hoarseness → pharyngeal cancer

Oropharyngeal lesions may occur with herpes zoster but vesicles are usually present on the face.

A metallic taste in the mouth with or without a complaint of a sore throat indicates *C. albicans* and hence diabetes must be excluded.

Bacterial causes of sore throat

Streptococcal tonsillopharyngitis

This infection may involve the pharynx only and vary from mild to severe, or it may involve both tonsils and pharynx. It is uncommon under 3 years or over 40 years.[11]

Guidelines for streptococcal throat

The four diagnostic features are the following; however, because viral infections are so common, the presence of all four features still has a predictive value of around 50% for streptococcus.[5]

- Constitutional symptoms:

fever ≥38°C

toxicity

- Tender anterior cervical lymphadenopathy

- Tonsillar swelling and exudate
- Absence of cough.

Other symptoms include:

- difficulty in swallowing
- significant pain, including pain on talking
- foul-smelling breath

Examination

- Pharynx very inflamed and oedematous
- Tonsils swollen with pockets of yellow exudate on surfaces (see FIGS. 62.4 and 62.5)
- Very tender enlarged tonsillar lymph nodes

Treatment

Indications for antibiotic therapy:[5]

- severe tonsillitis with above features of GABHS
- existing rheumatic heart disease at any age
- scarlet fever
- peritonsillar cellulitis or abscess (quinsy)
- patients 2–25 years with presumptive GABHS from vulnerable communities (e.g. remote Indigenous) with a high background incidence of acute rheumatic fever

Treatment should be with penicillin or an alternative antibiotic (see TABLE 62.3).[5]

Table 62.3 Treatment for streptococcal throat (proven or suspected)[5]

Children

phenoxymethyl penicillin 50 mg/kg/day (o) in 2
divided doses for 10 days (to max. 1 g/day)
or
if non-adherent, benzathine penicillin IM single dose according to weight
or (if hypersensitive to penicillin)
azithromycin 12 mg/kg up to 500 mg (o) daily for 5 days
or
cephalexin 25 mg/kg up to 1 g (o) bd for 10 days

Adults

phenoxymethyl penicillin 500 mg (o) 12 hourly for
10 days (can initiate treatment with one injection of procaine penicillin)
or (if hypersensitive to penicillin)
azithromycin 500 mg (o) daily for 5 days
or
cephalexin 1 g bd for 10 days

In those with low likelihood of adherence or oral treatment not tolerated:

benzathine penicillin 900 mg IM as a single dose in adults

In severe cases:

procaine penicillin 1–1.5 g IM daily for 3–5 days *plus* phenoxymethyl penicillin (as above) for 10 days

---

Note: Although symptoms and most evidence will disappear within 1–2 days of treatment, a full course of 10 days should be given to provide an optimal chance of eradicating *S. pyogenes* from the nasopharynx and thus minimising the risk of recurrence or complications such as rheumatic fever.[5] Some studies indicate that 7 days may be sufficient. Corticosteroids can be added in adults if very severe symptoms, e.g. restricted swallowing.

Antibiotic treatment has a variable effect on the resolution of symptoms. It does not protect against glomerulonephritis but does protect against rheumatic fever.[11] Amoxicillin should be avoided in tonsillitis because of confusion caused should mononucleosis be present. Frequent fluids are advisable and paracetamol for pain. Corticosteroids may be added if symptoms very severe, e.g. restricted swallowing, drooling.

Recurrent tonsillitis[5,11]

Recurrent sore throat is common in childhood and usually has a viral cause. Recurrent tonsillitis tends to be overdiagnosed. A throat swab taken during an acute episode can be helpful.[12] Only patients with more than five episodes of presumptive or proven bacterial tonsillitis in a year should be treated with prophylactic penicillin. The decision should be based on the severity of the episode, time lost from work or school, infectivity and response to antibiotics. Tonsillectomy in children results in only a modest reduction in the number of subsequent sore throats.[13]

Quinsy

Quinsy is a peritonsillar abscess characterised by marked swelling of the peritonsillar area with medial displacement of tonsillar tissue (see FIG. 62.6). It is usually caused by GABHS or anaerobes, occasionally *Staphylococcus aureus.* A typical picture of tonsillitis with severe unilateral throat pain and high fever is followed by increasing difficulty in swallowing and trismus.

Treatment

Antibiotics (e.g. procaine penicillin IM or clindamycin) plus aspiration or drainage in hospital under local anaesthetic if it is pointing. Oral penicillin treatment is likely to fail. Subsequent tonsillectomy may, but not always, be necessary. The cover can be broadened by adding metronidazole.[5]

Acute epiglottitis

In children this is a life-threatening infection. It may be overlooked in adults where, unlike children, the airway is usually not obstructed and the patient presents with a severe sore throat, dysphagia, drooling of saliva and a tender neck. Examination of the throat may appear quite normal. However, it is a severe infection requiring hospitalisation and parenteral antibiotics (e.g. cefotaxime).

Diphtheria

Due to the bacterium *Corynebacterium diphtheriae*, the potentially fatal form of this disease almost always occurs in non-immunised people. The clinical presentation may be modified by previous immunisation or by antibiotic treatment.

Clinical features

- Insidious onset
- Mild to moderate fever
- Mild sore throat and dysphagia
- Patient looks pale and ill
- Enlarged tonsils
- Pharynx inflamed and oedematous
- Pseudomembrane (any colour but usually grey–green) can spread beyond tonsils to fauces, soft palate, lateral pharyngeal wall and downwards to involve larynx (see FIG. 62.7)
- Enlarged cervical lymph nodes

- Soft tissue swelling of neck → ‘bull neck’ appearance

Management

- Throat swabs
- Antitoxin
- Penicillin or erythromycin 500 mg qid for 10 days
- Isolate patient

Viral causes of sore throat

Epstein–Barr mononucleosis

The angiose form of EBM is a real trap and must be considered in those aged 15–25 years (peak incidence) with a painful throat that takes about 7 days to reach its peak. Refer to CHAPTER 18 .

Clinical features

- Sore throat
- Prodromal fever, malaise, lethargy
- Anorexia, myalgia
- Nasal quality to voice
- Skin rash

Examination

- Petechiae on palate (not pathognomonic)
- Enlarged tonsils with or without white exudates (looks, but isn’t, purulent)
- Peri-orbital oedema
- Lymphadenopathy, especially posterior cervical
- Splenomegaly (50%)
- Jaundice ± hepatomegaly (5–10%)

The rash

- Primary rash (5%)
- Secondary rash:

  with ampicillin, amoxicillin (90–100%)

  with penicillin (50%)

Note: This rash is not synonymous with penicillin hypersensitivity.

Diagnosis

- Blood film—atypical lymphocytes
- White cell count—absolute lymphocytosis
- Heterophil antibodies

  or

  Monospot test (Paul–Bunnell)

  or

- EBV IgM test (more specific)

Treatment[5]

Symptomatic, e.g. paracetamol for pain. Parenteral corticosteroids only in the most severe cases.

Herpangina

An uncommon infection caused by the Coxsackie virus. Presents as small vesicles on soft palate, uvula and anterior fauces. These ulcerate to form small ulcers. The problem is benign and rapidly self-limiting.

Herpes simplex pharyngitis

In adults primary infection is similar to severe streptococcal pharyngitis but ulcers extend beyond the tonsils.

Other viral pharyngitis

Typically, the signs are fewer than with other causes. The typical case has mild redness without exudate and prominent (sometimes pale) lymphoid patches on the posterior pharynx (see FIG. 62.1). Tonsillar lymph nodes are usually not enlarged or tender. This picture is the

commonest encountered in general practice.

Candida pharyngitis

Oral candidiasis typically presents as milky-white growths on the palate, buccal and gingival mucosae, pharynx and dorsum of the tongue (see FIG. 62.2). If scraped away, a bleeding ulcerated surface remains. A bad (metallic) taste is a feature but the patient may complain of a sore throat and tongue and dysphagia.

Causes or predisposing factors to consider:

- HIV infection
- diabetes mellitus
- broad-spectrum antibiotics
- corticosteroids, including inhalers
- dentures
- debility

Management

- Determine underlying cause.

  nystatin suspension 1 mL (100 000 units/mL), rinse and swallow qid

  or

  amphotericin 10 mg lozenge dissolved slowly in oral cavity, 6 hourly, for 7–14 days

  or

  miconazole oral gel qid

When to refer

- Acute epiglottitis in children (a medical emergency)
- Inaccessible foreign body
- Abscess: peritonsillar or retropharyngeal
- Recurrent attacks of tonsillitis and adenoid hypertrophy for an opinion about tonsillectomy and/or adenoidectomy

- Suspicion or evidence of HIV infection or diphtheria
- Patients not responding to treatment
- Patients with more generalised disorders that are not yet diagnosed[12]

Guidelines for tonsillectomy[13]

- Repeated attacks of acute tonsillitis
- Enlarged tonsils and/or adenoids causing airway obstruction, including OSA
- Chronic tonsillitis
- More than one attack of peritonsillar abscess
- Biopsy excision for suspected new growth

Antibiotic treatment is aimed primarily at streptococcal pharyngitis and this is often based on clinical judgment.

Practice tips

- Consider severe tonsillitis with a covering membrane as EBM.
- If an adult presents with an intensely painful throat with a heavy exudate and that seems toxic, consider primary herpes simplex as well as streptococcal throat.
- Reserve swabs of the throat for verification of a streptococcal throat where it is important to do so, for suspected diphtheria and for suspicion of other serious infections such as tuberculosis.
- Be aware of possible complications, such as febrile convulsions in children and abscess formation.
- Do not misdiagnose unusual causes of a sore throat, such as cancer (see FIG. 62.8).
- The triad in an adult: hoarseness, pain on swallowing and referred ear pain = possible pharyngeal cancer.
- In managing the acute sore throat, consider whether the benefits of antibiotic use outweigh the disadvantages.

Patient education resource

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

- Tonsillitis

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63 Tiredness/fatigue

Chronic fatigue syndrome is not tiredness: there is a difference between feeling tired and 'fatiguey'. Fatigue involves a heaviness in the limbs, a sense of inability to think or move, pain in muscles and joints, nausea etc. Please understand the difference.

PERSON WITH CFS TO AUTHOR, JANUARY 1995

Tiredness, which basically means 'a desire to rest', or fatigue, which is derived from the latin 'fatigare'—to tire, is not a diagnosis but rather a symptom of illness: it may occur as either a presenting or a supporting symptom. Tiredness is interchangeable with terms such as weariness, lethargy, loss of energy, listlessness and exhaustion. It is a common and difficult presenting symptom often known by the acronym TATT—'tired all the time'. The symptom of tiredness is likely to be 'hidden' behind the request for a tonic or a physical check-up.[1]

Tiredness can be a symptom of a great variety of serious and uncommon diseases, including malignant disease. The challenge for the family doctor is to diagnose such disorders quickly without extravagant investigation.

Key facts and checkpoints

- The commonest cause of tiredness is psychological distress, including anxiety states, depression and somatisation disorder. It peaks in ages 20–40.
- An Australian study showed that fatigue presents at a rate of 1.4 per 100 GP encounters.[2]
- A survey in four NSW general practices by Hickie et al.[3] showed that 25% of adult attendees reported prolonged fatigue. Of these, 70% had psychological distress.
- In Jerrett's study,[4] no organic cause was found in 62.3% of patients presenting with lethargy; the constant factors were sleep disturbance and the presence of stress in their lives. Many of them turned out to be suffering from psychological problems or psychiatric illnesses, including depression, anxiety state or bereavement.
- An important cause of daytime tiredness is a sleep disorder such as obstructive

sleep apnoea, which results in periodic hypoventilation during sleep. It occurs in 2% of the general population in all age groups and in about 10% of middle-aged men.[4] Obesity and a history of snoring are pointers to the problem. See CHAPTER 60 .

- Underlying disorders that need to be considered as possible causes of prolonged fatigue are endocrine and metabolic disorders, malignancy, chronic infection, autoimmune disorders, primary psychiatric disorders, neuromuscular disorders, anaemia, drugs and cardiovascular disorders.
- Prolonged or chronic tiredness is characterised clinically by disabling tiredness, typically lasting more than 2 weeks, associated with non-restorative sleep, headaches and a range of other musculoskeletal and neuropsychiatric symptoms.[3]
- Sociodemographic correlates are concurrent psychological distress, female sex, lower socioeconomic status and fewer total years of education.[3]
- Chronic fatigue syndrome (CFS) is defined as debilitating fatigue, persisting or relapsing over 6 months, associated with a significant reduction in activity levels of at least 50%, and for which no other cause can be found.

Causes of tiredness

Analysing the symptom and reaching a diagnosis demands considerable skill, since tiredness may indicate the first subtle manifestation of a serious physical disease or, more commonly, may represent a person's difficulty dealing with the problems of their everyday life. Chronic tiredness or fatigue is a feature of the 'high pressure' nature of many people's lifestyles.

Careful consideration must be given to the differentiation of physiological tiredness, as a result of excessive physical activity, from psychological tiredness. Furthermore, before diagnosing tiredness as psychological, pathological as well as physical causes must be excluded.

A summary of causes of chronic tiredness is presented in TABLE 63.1 .

Table 63.1 Causes of chronic tiredness/fatigue

Psychogenic/non-organic
Psychiatric disorders: • anxiety states • depression/dysthymia • other primary disorders • bereavement

- somatisation disorder

Lifestyle factors:

- workaholic tendencies and ‘burnout’
- lack of exercise/sedentary lifestyle
- mental stress and emotional demands
- exposure to irritants (e.g. carbon monoxide, ‘lead’ fumes)
- inappropriate diet
- obesity
- sleep deprivation

Organic

Congestive cardiac failure

Anaemia

Malignancy

HIV/AIDS

Subacute to chronic infection (e.g. hepatitis, malaria)

Endocrine: various, especially thyroid (hyper and hypo), Addison disease and diabetes mellitus

Nutritional deficiency

Kidney failure

Liver disorders: chronic liver failure, chronic active hepatitis

Respiratory conditions (e.g. asthma, COPD)

Neuromuscular (e.g. MS, myasthenia gravis, Parkinson disease)

Metabolic (e.g. hypokalaemia, hypomagnesaemia

Drug toxicity, addiction or side effects (see TABLE 63.3)

Autoimmune disorders

Sleep-related disorders

Postinfectious fatigue syndrome (e.g. influenza, mononucleosis, COVID-19)

Unknown

Fibromyalgia

Chronic fatigue syndrome

Somatisation disorder

Irritable bowel syndrome

A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 63.2 .

Table 63.2 Tiredness/chronic fatigue: diagnostic strategy model

Probability diagnosis

Stress and anxiety
Depression
Inappropriate lifestyle and psychosocial factors
Viral/postviral infection
Sleep-related disorders (e.g. sleep apnoea)

Serious disorders not to be missed

Vascular:
- cardiac arrhythmias
- cardiomyopathy
- incipient CCF

Infection:
- hidden abscess
- HIV/AIDS
- hepatitis B and C
- others

Cancer, any malignancy
Other:
- anaemia
- haemochromatosis

Pitfalls (often missed)

'Masked' depression
Coeliac disease
Chronic infection (e.g. Lyme disease)
Incipient CCF
Fibromyalgia
Lack of fitness
Drugs: alcohol, prescribed, withdrawal
Menopause syndrome
Pregnancy
Neurological disorders:
- post head injury
- CVA

- Parkinson disease

Kidney failure

Metabolic (e.g. hypokalaemia, hypomagnesaemia)

Chemical exposure (e.g. occupational)

Rarities:

- hyperparathyroidism
- Addison disease (see CHAPTER 14)
- Cushing syndrome
- narcolepsy
- multiple sclerosis
- autoimmune disorders

Seven masquerades checklist

Depression

Diabetes

Drugs

Anaemia

Thyroid disease (other endocrine)

Spinal dysfunction

UTI

Is the patient trying to tell me something?

Highly likely.

Probability diagnosis

The most probable diagnoses to consider are:

- tension, stress and anxiety
- depression
- inappropriate lifestyle and psychosocial factors
- viral or postviral infection
- sleep-related disorders

Research studies have reported that over 50% (and in some cases as many as 80%) of reported cases of fatigue have been of psychological causation.[3] Overwork is a common cause of fatigue and is often obvious to everyone but the patient. The modern approach to sleep-related disorders has revealed several important factors causing excessive tiredness.

Serious disorders not to be missed

Many serious disorders such as anaemia, malignant disease and subacute or chronic infections (e.g. hepatitis, bacterial endocarditis and tuberculosis) can be ‘hidden’ or masked in the initial stages or not readily apparent. Neuromuscular diseases such as myasthenia gravis and multiple sclerosis, connective tissue disorders and HIV infection also have to be considered.

Pitfalls

The symptom of tiredness is fraught with pitfalls. Common ones include depression and other psychoneurotic disorders, and incipient congestive cardiac failure. Drug intake is a very common pitfall, whether it be by self-administration (including alcohol) or iatrogenic.

Tiredness is a feature of pregnancy in many women, so this association is worth keeping in mind, especially in the early stages when a change in menstrual history is not given or a young single woman will attempt to conceal the fact. It is also a presenting symptom of the menopause syndrome, which should not be misdiagnosed. Two classic causes of tiredness are haemochromatosis and coeliac disease.

Despite the impressive list of possibilities, GPs should also avoid the pitfall of ‘investigating for everything’ in the mistaken belief that a diagnosis can only be ruled out by throwing every known test or referral at it.

Seven masquerades checklist

All these important problems are capable of being responsible for tiredness, especially depression, diabetes, drugs, anaemia and urinary infection. Thyroid disorder could certainly be responsible. Drugs that commonly cause tiredness are listed in TABLE 63.3 .

Table 63.3 Drugs that can cause tiredness

Alcohol
Analgesics
Antibiotics
Anticonvulsants
Antipsychotics
Antidepressants
Anti-emetics
Antihistamines
Antihypertensives, e.g. beta blockers
Anxiolytics
Corticosteroids

Digoxin
Ergot alkaloids
Hormones (e.g. oral contraceptives)
Hypnotics
Nicotine
NSAIDs
Vitamins A and D (early toxic symptoms)

Note: Most drugs have a considerable capacity to cause tiredness.

Drug withdrawal, especially for illicit drugs such as amphetamines, marijuana, cocaine and heroin, has to be considered.

Psychogenic considerations

Tiredness is a symptom that may represent a 'ticket of entry': a plea for help in a stressed, anxious or depressed patient. Any of the primary psychiatric disorders can present as tiredness.

Red flag pointers (with examples) for tiredness

- Unexplained weight loss (malignancy, HIV, diabetes, hyperthyroidism)
- Recent onset in well elderly person (malignancy, anaemia, arrhythmia, diabetes, renal failure)
- Persistent fever or lymphadenopathy (HIV, hidden abscess, previous infection)
- Dyspnoea (cardiac failure, arrhythmia, anaemia, COPD)
- Recent onset and progressive (autoimmune disease, malignancy, coeliac disease, MS, haemochromatosis, Parkinson disease)
- Symptoms of depression
- Drug and alcohol abuse

The clinical approach—key history

Careful history taking, often followed up at a second appointment, is the key to diagnosing and managing tiredness. Perfunctory history-taking risks both missing an important cause, and over-investigating using a scattergun approach that leads down 'blind alleys'.

It is mandatory that questions be asked about the following if the information is not volunteered by the patient:

- sleep pattern (it is not uncommon for patients to say they sleep well and yet on questioning it is found they have initial insomnia, or middle insomnia, or both, with or without early morning waking). It is most relevant to talk to any sleeping partners to obtain a history of sleep disturbance
- weight fluctuations
- energy—performance—ability to cope
- sexual activity/sexual problems
- suicidal ideas
- self-medication—OTC preparations (e.g. bromides, stimulants, analgesics, alcohol, cigarettes, other drugs); this is particularly important in the drug addiction-prone group: doctors, chemists, nurses, workers in the liquor industry, truck drivers
- fears (including phobic symptoms, hypochondriasis)
- precipitating factors (present in over 50% of patients with depressive illness):

    postpartum

    postoperative

    associated with chronic physical illness

    bereavement

    pain—chronic pain conditions

    retirement

    medication

    post trauma (e.g. motor vehicle accident)

    postviral infections, especially hepatitis, mononucleosis, influenza, COVID-19
- work history—determine whether the person is a workaholic; ask about bullying at work
- dietary history—determine pattern, including fad diets or skipped meals
- psychological history—stress, anxieties, phobias, depression
- menstrual history and symptoms related to the menopause syndrome

- final questions: ‘Is there anything else you feel you should tell me?’ ‘Do you have any explanation for your tiredness?’
- self-question: ‘Is this patient depressed?’

Physical examination—key features

- General inspection noting facial features, skin appearance and colour, hyperpigmentation, conjunctivae
- Vital signs
- Anthropometric measurements
- Basic respiratory and cardiovascular
- Abdominal examination with focus on masses and inguinal lymphadenopathy
- Urinalysis

Key investigations (screening) guidelines[5]

Only 4% of people reporting unexplained fatigue to their GP have a significant abnormal pathology result.[5] A ‘comprehensive’ investigative work-up for every person who presents with unexplained tiredness will result in numerous positive test results, most of which are false positives. This can lead to wasted time, effort, expense and possibly harmful invasive investigations, and can distract from commencing a reasonable management plan.

Most non-urgent presentations benefit from a few simple initial tests and one or more further consultations over time; further tests can be ordered if any category of diagnoses becomes more likely.

Initial tests could include:

- Office tests: finger-prick glucose, urine dipstick
- FBE
- Serum electrolytes, creatinine
- Liver function tests
- Ferritin
- TSH

Further tests, if indicated (not a comprehensive list):

- Calcium, magnesium
- ESR/CRP (evidence varies as to the usefulness of these)
- Gluten sensitivity screening blood tests
- Faecal occult blood
- CXR

Consider others according to clinical features, e.g. chronic infection and HIV screening, autoimmune disorders, cancer markers and sleep disorder studies.

The diagnosis of CFS can be made only when the minimum investigations have been shown to be normal or to demonstrate minor abnormalities in liver function or blood film (atypical lymphocytes).

Tiredness in children

Tiredness in children is caused by a range of predictable conditions, such as physiological factors (excessive exercise, lack of sleep, poor diet), infections, allergies including asthma, drugs, depression and various illnesses in general.

Overweight children are likely to fatigue more rapidly than children of normal weight.[6] Page 770 Any bacterial, viral or other infection may be associated with tiredness. Chronic EBV infection causing recurrent episodes of fever, pharyngitis, malaise and adenopathy is a significant cause in adolescents, who present with chronic exhaustion that can be mistaken for malignancy.[6] Tonsillar–adenoidal hypertrophy may be large enough to compromise air exchange, particularly during sleep. Snoring may be a feature plus tiredness and lethargy in the waking state.

Tiredness is a presenting feature of depression in adolescents, a serious problem that often goes unrecognised.

Tiredness in the elderly

Elderly people tend to tire more quickly and recover more slowly and incompletely than younger ones. Sleep in older people is generally shorter in duration and of lesser depth, and they feel less refreshed and sometimes irritable on awakening.

Fatigue may be present as a result of emotional frustration. Whenever the prospect of gratification is small, a person tends to tire quickly and to remain so until something stimulating appears. Since the prospects for gratifying experience wane with the years, easy ‘fatigueability’ or tiredness is common in this age group.

However, the onset of marked tiredness in an older person who was previously well raises the possibility of a number of underlying pathologies.

See Red flag pointers .

Bereavement

Although a bereavement reaction is common and a normal human response that occurs at all ages, it is more frequently encountered in the elderly, with the loss of a spouse or a child (young or middle-aged). Fatigue that occurs during the initial mourning period is striking and might represent a protective mechanism against intense emotional stress. With time, usually around 6 to 12 months, a compensated stage is reached, fatigue gradually abates and the patient resumes normal activities as the conflicts of grief are gradually resolved. Freud pointed out the complexities of mourning as the bereaved person slowly adjusts to the loss of the loved one. In others, various symptoms persist as an ‘abnormal grief reaction’, including persistence of fatigue. Some factors that may lead to this include:

- unexpected death
- high dependence upon the dead person
- guilt feelings, especially in a love/hate relationship

Studies in general practice have shown that widows see their family doctors for psychiatric symptoms at three times the usual rate in the first 6 months after bereavement. The consultation rate for non-psychiatric symptoms also increases, by almost 50%.

Role of the family doctor

Following bereavement, it is important to watch for evidence of depression, drug dependency (especially alcohol) and suicidal tendencies. In cases of expected death, management should, if possible, start before the bereavement. Supportive care and ongoing counselling are very important.

General related disorders

Burnout

Definition

Burnout is a clinical syndrome recently officially recognised in the World Health Organization’s ICD-11 classification.[7] It has three dimensions:

- exhaustion
- feelings of negativity, and cynicism towards one’s job
- reduced professional efficacy

It is similar to stress-related depression but mood lowering is temporary and work-specific. Burnout is a prolonged response to chronic occupational stressors.

When a person describes themselves as feeling ‘burnt out’, this may reflect a whole constellation of psychogenic symptoms, such as exhaustion, boredom and cynicism, paranoia, detachment, heightened irritability and impatience, depression and psychosomatic complaints, such as headache and tiredness. It is important to clarify the nature of the problem with care.

At work, are they overextended, disengaged and ineffective? Or do they have an underlying psychoneurotic disorder such as hypomania, anxiety state or depression, or a personality disorder?

Occupations particularly prone to burnout are doctors and other health professionals, musicians, authors, teachers, athletes, engineers, emergency service workers, soldiers, reporters and high-technology professionals.

Management involves appropriate counselling including mindfulness and a holistic approach, which aims to help the person to identify work and life stressors, set realistic personal goals and develop good support mechanisms. There may be a place for helping advocate for workplace changes, or supporting the realisation that the current job is not for them.

Chronic fatigue syndrome

This complex syndrome, which causes profound and persistent tiredness, is also referred to as myalgic encephalomyelitis, chronic neuromuscular viral syndrome,[8] postviral syndrome, chronic EBV syndrome, viral fatigue state, epidemic neuromyasthenia, neurasthenia, Icelandic disease, Royal Free disease and Tapanui disease. CFS is not to be confused with the tiredness and depression that follow a viral infection such as infectious mononucleosis, hepatitis or influenza. These postviral tiredness states are certainly common but resolve within 6 months or so.

Typical features of CFS (see FIG. 63.1):[8]

- extreme exhaustion (with minimal physical effort)
- headache or a vague ‘fuzzy’ feeling in the head
- aching in the muscles and legs
- poor concentration and memory
- hypersomnia or other sleep disturbance
- waking feeling tired
- emotional lability/anxiety
- depressive-type illness, mood swings

- arthralgia (without joint swelling)
- sore throat
- subjective feeling of fever (with a normal temperature)
- shortness of breath
- tender, swollen lymph nodes
- usually occurs between 20 and 40 years of age

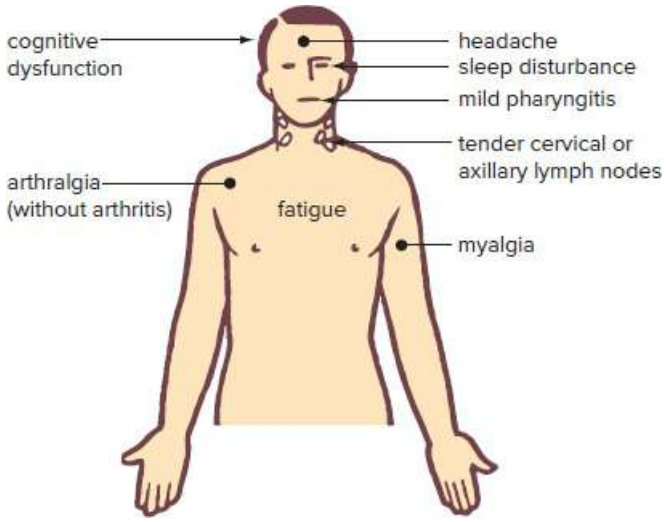

FIGURE 63.1 Chronic fatigue syndrome: characteristic symptoms

Epidemiologically it has been related to Coxsackie B virus infections. The responsible organism is referred to as a slow virus infection by some authorities.[9]

In approximately two-thirds of people the illness follows a clearly defined viral illness. However, no single virus has been consistently associated with the development of the syndrome, which is known to develop following a wide range of viral and non-viral infective illnesses. Immune system dysfunction with chronic overproduction of cytokines (e.g. interferon) is a possible pathogenetic mechanism.

Every family doctor probably has patients with this disorder and the syndrome has been observed in isolated endemics from time to time. Hickie et al.[3] found that only 0.3% of those with prolonged fatigue had a diagnosis of CFS according to their family doctor. Veterans of the Gulf War show a 10-fold incidence of CFS compared with non-deployed military personnel.[10]

There is no doubt that the syndrome is real in these patients. One of the major problems confronting clinicians is that there is no diagnostic test for this illness, so it remains a clinical diagnosis backed up by normal baseline investigations.

Diagnostic criteria for CFS have been published[11] (see TABLE 63.4), which emphasise the positive clinical features of the syndrome and the chronicity of symptoms (greater than 6 months), in addition to the need for careful exclusion of alternative diagnoses by history, physical examination and laboratory investigation.

Table 63.4 Criteria for the diagnosis of chronic fatigue syndrome[11]

Fatigue

Clinically evaluated, unexplained, persistent or relapsing fatigue persistent for 6 months or more, that:

- is of new or definite onset
- is not the result of ongoing exertion
- is not substantially alleviated by rest
- results in substantial reduction in previous levels of occupational, educational, social or personal activities

and

Other symptoms

Four or more of the following symptoms that are concurrent, persistent for 6 months or more and which did not predate the fatigue:

- impaired short-term memory or concentration
- sore throat
- tender cervical or axillary lymph nodes
- muscle pain
- multi-joint pain without arthritis
- headaches of a new type, pattern or severity
- unrefreshing sleep
- post-exertional malaise lasting more than 24 hours

Examination and investigation

Apart from mild pharyngeal infection, cervical lymphadenopathy or localised muscle tenderness, the physical examination is normal.

Investigations should be directed towards excluding possible diagnoses for that patient, such as chronic infection, autoimmune disorders, endocrine and metabolic disorders, primary neuromuscular disorders, malignancy and primary psychiatric disorders. The last mentioned is the most difficult of the differential diagnoses and psychiatric referral will often need to be considered.

Management

Patients who have CFS are really suffering and unhappy people, similar to those with fibromyalgia (see CHAPTER 27). They require considerable understanding and support. Multidisciplinary intervention is recommended. Symptoms last approximately 2½ years.

Management strategies include:[8]

- CFS recognition—explain that the illness is real but the cause unknown and tests are likely to be normal
- explanation and reassurance that the illness is usually self-limiting with no permanent complications; and that a slow, steady improvement can be anticipated, with most CFS patients returning to normal health
- provide continued psychological support
- review for diagnostic reappraisal (examine at least every 4 months)
- avoid telling patients they are depressed
- treat symptomatically—pain relief, consider NSAIDs and antidepressants if significant depression
- refer to counselling and support groups
- provide a realistic, regular, graduated exercise program, which shows improvement in functional work capacity[12]
- promotion of sleep hygiene and an optimal healthy diet
- reduce relevant stress factors (map a realistic living program)
- psychiatric referral if appropriate
- ask the patient to keep a diary of exercise/stress and symptom severity, in particular
- avoid long-distance travel, which is poorly tolerated

Cognitive behaviour therapy appears to help some people, as do relaxation therapy, meditation, stress management and psychotherapy, where indicated.

The emphasis should be placed on caring, rather than curing, until a specific evidence-based intervention is found.

A systematic review has found that cognitive behaviour therapy administered by skilled therapists and exercise are beneficial. Although few patients are cured with CBT, the therapeutic effect is substantial.[13] There is insufficient data or evidence to support the use of antidepressants,

corticosteroids, complementary therapies and dietary supplements, including vitamins B12 and C and co-enzyme Q_{10}.[5] Prolonged rest and immunotherapy is unlikely to be beneficial.[14]

Fibromyalgia

The fibromyalgia syndrome (see CHAPTER 27) bears a clinical resemblance to CFS. Musculoskeletal pain is more prominent although tiredness (fatigue) and sleep disturbance are features. According to Schwenk,[15] fibromyalgia affects 5% of the American population (as for so many disease prevalence studies, treat this figure with caution) with a peak age of 35 years (range 20–60) and a female:male ratio of 10:1. The management is similar to CFS but the prognosis is less optimistic.

Practice tips

- Always consider underlying psychological distress, especially a depressive disorder.
- Do not overlook a sleep disorder.
- Believe the person's symptoms.
- Ask the person what they believe may be the cause of the tiredness.
- Be careful of labelling a person as having CFS.
- Restrict investigations to those that are more likely to be relevant, therapeutic and reassuring.
- A practical approach to the tired patient is to take a comprehensive history and examination, consider a period of watchful waiting in the absence of red flags and the judicious use of investigations once that decision is adopted.

Patient education resource

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

- Chronic fatigue syndrome

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64 The unconscious patient

In whatever disease sleep is laborious, it is a deadly symptom; but if sleep does good, it is not deadly.

HIPPOCRATES

The state of arousal is determined by the function of the central reticular formation, which extends from the brain stem to the thalamus. Coma occurs when this centre is damaged by a metabolic abnormality or by an invasive lesion that compresses this centre. Coma is also caused by damage to the cerebral cortex.[1]

The word ‘coma’ is derived from the Greek *koma*, which means deep sleep. However, the deeply unconscious patient is not in a deep sleep. Coma, which is commonly defined as unrousable or unresponsive, is best defined as ‘lack of self-awareness’.[2]

The various levels of consciousness are summarised in TABLE 64.1 ; the levels vary from consciousness, which means awareness of oneself and the surroundings in a state of wakefulness,[3] to coma, which is a state of unrousable unresponsiveness. Rather than using these broad terms in clinical practice it is preferable to describe the actual state of the patient in a sentence.

Table 64.1 The five conscious levels

	State	Clinical features	Simplified classification
	1 Consciousness	Aware and wakeful	Awake
	2 Clouded consciousness	Reduced awareness and wakefulness ‘Alcohol effect’ Confusion Drowsiness	Confused
	3 Stupor	Unconscious	Responds to shake and

		Deep-sleep-like state Arousal with vigorous stimuli	shout
	4 Semicomatose	Unconscious (deeper) Responds only to painful stimuli (sternal rubbing with knuckles) without arousing	Responds to pain
	5 Coma	Deeply unconscious Unrousable and unresponsive	Unresponsive coma

Key facts and checkpoints

- Always consider hypoglycaemia or opioid overdose in any unconscious person, especially of unknown background.
- If a person is unconscious and cyanosed, consider upper airway obstruction until proved otherwise.
- The commonest causes of unconsciousness encountered in general practice are reflex syncope, especially postural hypotension, concussion and cerebrovascular accidents (CVAs). The main causes are presented in TABLE 64.2 . Brain pathology can be considered as supratentorial or infratentorial.
- Do not allow the person who accompanies the unconscious patient to leave until all relevant details have been obtained.
- Record the degree of coma as a baseline to determine improvement or deterioration.

Table 64.2 Main causes of loss of consciousness

Episodic causes—blackouts

Epilepsy

Orthostatic intolerance and syncope
Drop attacks
Cardiac arrhythmias (e.g. Stokes–Adams attacks)
Vertebrobasilar insufficiency
Psychogenic disorders, including hyperventilation
Breath-holding (children)
Silent myocardial infarction
Hypoxia

Coma

(COMA provides a useful mnemonic for four major groups[1] of causes of unconsciousness)

C = CO_2 narcosis: respiratory failure; hypoxia

O = Overdose of drugs:

- alcohol
- opioids
- tranquillisers and antidepressants
- carbon monoxide
- analgesics
- others

M = Metabolic:

- diabetes:
    - hypoglycaemia
    - ketoacidosis
- hypothyroidism
- hypopituitarism
- hepatic failure
- Addison disease
- kidney failure (uraemia)
- others

A = Apoplexy:

- intracerebral haemorrhage
- haematoma: subdural or extradural
- head injury
- cerebral tumour

- cerebral abscess

Infratentorial (posterior fossa):

- pressure from above
- cerebellar tumour
- brain-stem infarct/haemorrhage
- Wernicke encephalopathy

Meningismus (neck stiffness):

- subarachnoid haemorrhage
- meningitis

Other:

- encephalitis
- overwhelming infection, e.g. septicaemia

Trauma

Urgent attention

The initial contact with the unconscious patient is invariably sudden and dramatic and demands immediate action, which should take only seconds to minutes. The primary objective is to keep the patient alive until the cause is determined and possible remedial action taken.[3]

History

A history can be obtained from relatives, friends, witnesses, ambulance officers or others. The setting in which the person is found is important. Evidence of discs or cards identifying an illness such as diabetes or epilepsy should be searched for. Is there a known history of hypertension, heart disease, respiratory disease, psychiatric illness or overdose?

Questions to be considered[4]

- Does the person have diabetes?
  Do they use insulin?
  Have they had a recent infection?
  Have they recently reduced their food intake (hypoglycaemia)?

- Is drug overdose possible? Have they:
    - suffered recent stress or personal ‘mishaps’
    - suffered recent depression or self-harm
    - been prescribed any centrally acting medications?
- Is opioid usage possible?
    - Are the presenting circumstances unusual?
- Is epilepsy possible?
    - Did the person pass urine or faeces?
    - Was twitching in the limbs observed?
    - Has the tongue been bitten?
- Is head injury possible? Have they:
    - had complaints of headache
    - any recent accident?
- Has a stroke or subarachnoid haemorrhage occurred? Has the patient:
    - had a history of hypertension
    - suffered recent severe headache
    - complained of weakness of the limbs?

Examination

General features requiring assessment:

- breathing pattern:
    - Cheyne–Stokes respiration: periodic breathing = cerebral dysfunction
    - ataxic respiration: shallow irregular breathing = brain-stem lesion
    - Kussmaul respiration: deep rapid hyperventilation = metabolic acidosis
- breath odour: may be a characteristic feature of alcohol, diabetes, uraemia and hepatic coma

- level of consciousness: degree of coma (see TABLE 64.1); the Glasgow coma scale (see TABLE 64.3) and AVPU scale (see TABLE 64.4) are frequently used as a guide to the conscious state
- skin features: look for evidence of injection sites (IV drug use, diabetes), snake bite marks, colour (cyanosis, purpura, jaundice, rashes, hyperpigmentation) and texture
- circulation
- pulse oximetry
- temperature: consider infection such as meningitis and hyperpyrexia if raised and hypothermia (e.g. hypothyroidism) if low
- hydration: dehydration may signify conditions such as a high fever with infections, uraemia, hyperglycaemic coma

Examination	Action
Is the person breathing?	If not, clear airway and ventilate.
Note chest wall movement.	Perform cardiopulmonary resuscitation if necessary.
Check pulse and pupils.	Consider naloxone.
Is there evidence of trauma?	Consider extradural haematoma.
Is the person hypoglycaemic?	Obtain glucometer estimation of blood sugar.
Evidence of diabetes	
Are vital functions present yet immediate correctable causes eliminated?	Place in coma position.

Table 64.3 Glasgow coma scale

	Score
Eye opening (E)	
Spontaneous opening	4
To verbal command	3
To pain	2
No response	1
Verbal response (V)	
Orientated and converses	5

Disoriented and converses	4
Inappropriate words	3
Incomprehensible sounds	2
No response	1
Motor response (M)	
Obeys verbal command	6
Response to painful stimuli	
Localises pain	5
Withdraws from pain stimuli	4
Abnormal flexion	3
Extensor response	2
No response	1
Coma score = E + V + M	
Minimum 3	
Maximum 15	
If 8–10: take care—monitor the airway	

Table 64.4 AVPU scale

Awake	The person is awake
Verbal	The person responds to verbal stimulus
Pain	The person responds to a painful stimulus
Unconscious	The person is unresponsive to any stimulus

Examination of the head and neck[3,4]

Consider the following:

- facial asymmetry
- the skull and neck: palpate for evidence of trauma and neck rigidity
- eyes, pupils and ocular fundi: look for constricted pupils in opioid overdose

- tongue
- nostrils and ears
- auscultation of the skull for bruits

Examination of the limbs

Consider:

- injection marks (IV drug use, diabetes)
- tone of the limbs by lifting and dropping (e.g. flaccid limbs with early hemiplegia)
- reaction of limbs to painful stimuli
- reflexes—tendon reflexes and plantar response

General examination of the body

This should include assessment of the pulses and blood pressure.

Urine examination

Catheterisation of the bladder may be necessary to obtain urine. Check the urine for protein, sugar and ketones.

Diagnosing the hysterical ‘unconscious’ patient

One of the most puzzling problems in emergency medicine is how to diagnose the unconscious patient caused by a conversion reaction (hysteria). These patients really experience their symptoms (as opposed to the pretending patient) and resist most normal stimuli, including painful stimuli.

Method

- Hold the patient’s eye or eyes open with your fingers and note the reaction to light.
- Now hold a mirror over the eye and watch closely for pupillary reaction. The pupil should constrict with accommodation from the patient looking at his or her own image.

Investigations

Appropriate investigations depend on the clinical assessment. The following is a checklist.

- Blood tests:

  – All patients: blood sugar
  urea and electrolytes

  – Selected patients: FBE
  blood gases
  liver function tests
  blood alcohol
  serum cortisol
  thyroid function tests
  serum digoxin

- Pulse oximetry
- Urine tests:

  a urine specimen is obtained by catheterisation

  test for glucose and albumin

  keep the specimen for drug/toxin screening

- Stomach contents: aspiration of stomach contents for analysis
- Radiology: brain CT or MRI are the investigations of choice (if available). If unavailable, X-ray of the skull may be helpful.
- Cerebrospinal fluid: lumbar puncture, necessary with neck stiffness, has risks in the comatose patient. A preliminary CT scan is necessary to search for coning of the cerebellum. If clear, the lumbar puncture should be safe and will help to diagnose subarachnoid haemorrhage and meningitis.
- Electroencephalograph
- ECG; look for ↑ QT interval, etc.

Blackouts—episodic loss of consciousness

Episodic or transient loss of consciousness is a common problem. The important causes of blackout are presented in TABLE 64.5 . The history is important to determine whether the patient is describing a true blackout or episodes of dizziness, weakness or some other sensation.

Table 64.5 Clinical features of blackouts

Cause	Precipitants	Subjective onset	Observation	Recovery
Reflex syncope	Posture Stress Haemorrhage Micturition Dehydration	Warning of feeling ‘faint’, ‘distant’, ‘clammy’, ‘sweaty’	Very pale Sweating	Gradual Feels ‘terrible’ Fatigue Nausea
Cardiac syncope including POTS syndrome	Various	May be palpitations	Pale	Rapid May be flushing
Autonomic syncope	Postural change orthostasis, food, alcohol	Warning (feels faint)	Pale	Rapid
Respiratory syncope	Cough Weight-lifting ‘Trumpet playing’	Warning (feels faint)	Pale	Rapid
Carotid sinus syncope	Carotid pressure (e.g. tight collar + turning neck) Postendarterectomy	Warning (feels faint)	Pale	Rapid
Migrainous syncope	Foods Stress Sleep deprivation	Scotomas	Pale	Nausea and vomiting Throbbing headache
Epilepsy	Stress Sleep deprivation Alcohol withdrawal Infection Menstruation Drug non-compliance	Aura with complex partial seizures (CPS)	Automatism (e.g. fidgeting, lip smacking) with CPS	• Slow • Confused

The clinical features of various types of blackouts are summarised in TABLE 64.5 .

Epilepsy

Epilepsy is the commonest cause of blackouts. There are various types, the most dramatic being the tonic–clonic seizure, which involve a sudden loss of consciousness without warning. See CHAPTER 43 .

Locked-in syndrome

This is a state of awareness and wakefulness with quadriplegia and paralysis of the lower cranial nerves. There is an inability to speak, move or show facial expression. Communication is possible by coded eye movements. It is invariably caused by a CVA (see CHAPTER 121).

Orthostatic intolerance and syncope

In syncope there is a transient loss of consciousness but with warning symptoms and rapid return of alertness following a brief period of unconsciousness (seconds to 3 minutes). The three main syndromes that are outlined in CHAPTER 43 are reflex syncope, postural orthostatic tachycardia syndrome (POTS) and autonomic failure.

Reflex syncope

Relevant features of reflex syncope or vasovagal or common faint (see TABLE 64.5):

- occurs with standing or, less commonly, sitting
- warning feelings of dizziness, faintness or true vertigo
- nausea, hot and cold skin sensations
- fading hearing or blurred vision
- sliding to ground (rather than heavy full-length fall)
- rapid return of consciousness
- pallor, sweating and bradycardia
- often trigger factors (e.g. emotional upset, pain)

The person invariably remembers the onset of fainting. Most syncope is of the benign vasomotor type and tends to occur in young people, especially when standing still (e.g. choir boys). It is the main cause of repeated fainting attacks.

The treatment is to avoid precipitating causes (e.g. prolonged standing, especially in the sun) and if premonitory signs occur, lie down if possible, or bend forwards with the head down. ‘Smelling salts’ (ammonium carbonate) can be carried and used in these circumstances.

Other forms of syncope

Micturition syncope

This uncommon event may occur after micturition in older men, especially during the night when they leave a warm bed and stand to void. The cause appears to be peripheral vasodilatation associated with reduction of venous return from straining.

Cough syncope

Severe coughing can result in obstruction of venous return with subsequent blackout. This is also the mechanism of blackouts with breath-holding attacks.

Carotid sinus syncope

This problem is caused by pressure on a hypersensitive carotid sinus (e.g. in some elderly people who lose consciousness when their neck is touched).

Effort syncope

Syncope on exertion is due to obstructive cardiac disorders, such as aortic stenosis and hypertrophic obstructive cardiomyopathy.

Choking

Sudden collapse can follow choking. Examples include the so-called ‘barbecue coronary’ when the person, while eating meat, suddenly becomes cyanosed, is speechless and grasps the throat. This is caused by inhaling a large bolus of meat that obstructs the larynx. To avoid death, immediate relief of obstruction is necessary. An emergency treatment is the Heimlich manoeuvre, whereby the person is grasped from behind around the abdomen and a forceful squeeze applied (may be repeated) to try to eject the food. If this fails, the foreign body may have to be manually removed from the throat, or CPR commenced.

Drop attacks

Drop attacks are episodes of ‘blackouts’ in which the person suddenly falls to the ground and then immediately gets up again. These involve sudden attacks of weakness in the legs. Although there is some doubt about whether loss of consciousness has occurred, most individuals cannot remember the process of falling. Drop attacks occur typically in middle-aged women and are considered to be brain-stem disturbances producing sudden changes in tone in the lower limbs. Other causes of drop attacks include vertebrobasilar insufficiency, Parkinson disease and epilepsy.[5]

Cardiac arrhythmias

Stokes–Adams attacks (see CHAPTER 59) and cardiac syncope are manifestations of recurrent episodes of loss of consciousness, especially in the elderly, caused by cardiac arrhythmias. These arrhythmias include complete heart block, sick sinus syndrome and ventricular tachycardia. The blackout is sudden with the person falling straight to the ground without warning and without convulsive movements. They go pale at first and then flushed.

Twenty-four-hour ambulatory cardiac monitoring may be necessary to confirm the diagnosis.

People with aortic stenosis are prone to have exercise-induced blackouts. Consider the prolonged QT interval syndrome in all age groups when the person presents with dizziness or blackouts.

Vertebrobasilar insufficiency

Loss of consciousness can occur rarely with vertebrobasilar insufficiency (VBI) transient ischaemic attack. Typical preceding symptoms of VBI include dysphasia, dysarthria, vertigo, vomiting, hemisensory loss, ataxia and transient global amnesia.

Hypoglycaemia

Hypoglycaemia can be difficult to recognise but must be considered as it can vary from a feeling of malaise and lightheadedness to loss of consciousness, sometimes with a convulsion. There are usually preliminary symptoms of hunger, sweating, shaking or altered behaviour. Hypoglycaemic attacks are usually related to diabetes and can occur with sulfonylureas as well as insulin. Causes of hypoglycaemia are presented in TABLE 64.6 . Refer to CHAPTER 120 .

Table 64.6 Causes of hypoglycaemia (adults)

Diabetes-related including insulin and oral hypoglycaemics
Drugs (e.g. quinine, salicylates, sulfonylureas, beta blockers)
Alcohol
Fasting
Tumours (e.g. insulinomas)
Addison disease
Hypopituitarism
Liver disease
Hypoglycaemic artefacta
Pregnancy
Post bariatric surgery
Gastric ‘dumping’ syndrome

Glycogen storage disease
Autoimmune: antibodies to insulin or insulin receptors

Head injuries and unconsciousness

Some non-life-threatening head injuries are sufficiently serious to cause significant loss of consciousness and retrograde amnesia. The clinical terms used to describe brain injury—concussion, contusion and laceration—simply indicate minor to major degrees of a similar injury. Severe individual cases of the above can certainly result in fatal outcomes.

Concussion[6]

Concussion is a transient disturbance of neurological function (occasionally with loss of consciousness) induced by head injury and usually resulting in no persistent abnormal neurological signs. However, depending on definition, 1–10% may develop post-concussive symptoms such as headaches, balance problems, fatigue or noise/light sensitivity.[7] The features of the various grades of concussion are shown in TABLE 64.7 .

Table 64.7 Classification of concussion

Grade	Clinical features
Mild (grade 1)	Stunned or dazed Sensorium clears in <60 seconds No post-traumatic amnesia ± Loss of consciousness
Moderate (grade 2)	Stunned or dazed Sensorium cloudy >60 seconds Headache Amnesia <60 minutes ± Loss of consciousness Loss of colour vision
Severe (grade 3)	Sensorium cloudy >60 seconds Irritable Persistent headache Unsteady gait ± Loss of consciousness

Take extra care in children who are more sensitive to head injury and need to be kept home for around three days.

Post-concussion syndrome

Occasionally, a person who has an episode of concussion has persistence of headaches and dizziness for a number of weeks. Poor memory and concentration and sluggish decision making indicate impaired mental capacity. Patients with this problem should be investigated with neuropsychological testing and CT scanning or MRI of the brain.

Chronic traumatic encephalopathy

This is a progressive brain disease related to repeated traumatic brain injuries, including concussion and other blows to the head. Symptoms, which are progressive, include cognitive impairment, apathy, depression, short-term memory loss and similar cerebral dysfunction. It is associated with dementia. Diagnosis is clinical and by cerebral imaging, especially MRI.

Links to sports concussion tests[8]

- Pocket Concussion Recognition Tool (CRT5)
- Sports Concussion Assessment Tool 5th edn (SCAT5), including Child SCAT5 5–12 years

Extradural (epidural) haematoma

This life-threatening head injury is caused by arterial bleeding between the skull bone and dura mater (see FIG. 64.1). Following injury there may be a short lucid interval followed by loss of consciousness. The patient is restless, confused, irritable (see FIG. 64.2), has severe headaches and develops neurological signs such as seizures, ipsilateral pupil dilatation and facial weakness. A skull X-ray and CT scan should demonstrate the haematoma. Lumbar puncture is contraindicated. Urgent decompression of the haematoma is required.

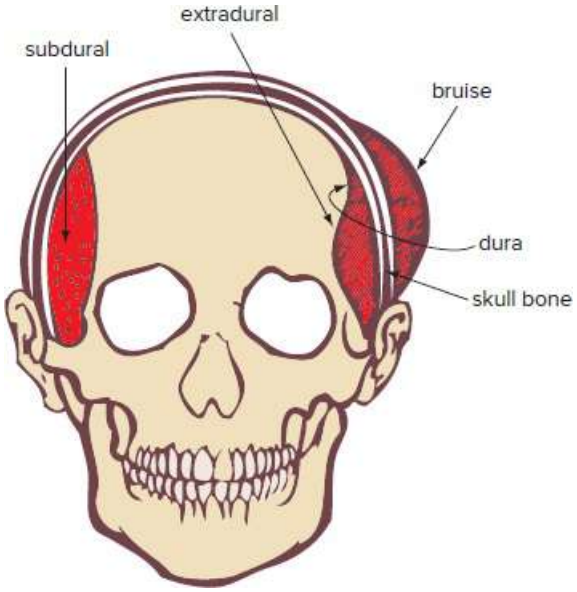

FIGURE 64.1 Illustration of sites of subdural and extradural haematomas in relation to the dura, skull and brain

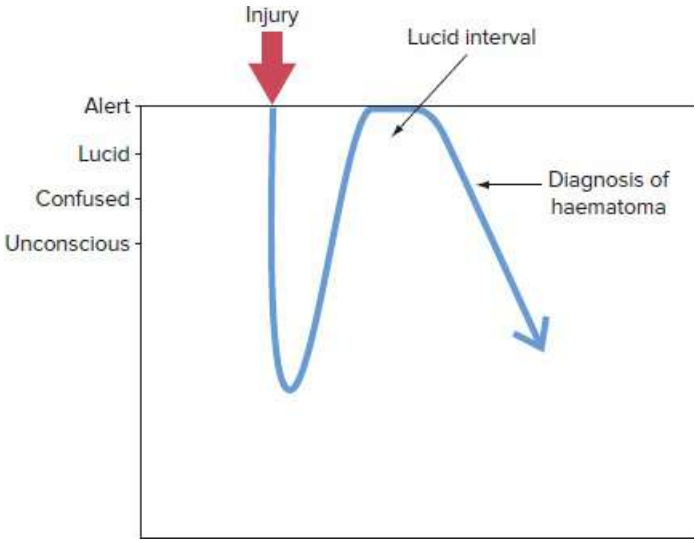

FIGURE 64.2 Classic conscious states leading to extradural haematoma after injury

Subdural haematoma

This is due to a venous bleed between the dura and the arachnoid. It follows injury, which may be seemingly trivial, especially in the elderly, and may be acute, subacute and chronic. Consider it in a person with personality change, slowness and unsteadiness of movement, headache, irritability and fluctuating conscious level. A CT scan or MRI should reveal the haematoma

and/or a midline shift. Neurological referral is urgent.

Psychogenic factors

Psychogenic factors leading to blackouts represent a diagnostic dilemma, especially if occurring in those with tonic–clonic epilepsy. If the attacks are witnessed by the practitioner, then the possibility of functional origin can be determined.

Hysterical blackouts or fits are not uncommon and have to be differentiated from hyperventilation. It is unusual for hyperventilation to cause unconsciousness but it is possible to get clouding of consciousness, especially if the person is administered oxygen.

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Other features that suggest psychogenic rather than organic factors are:

- labile affect
- rapidly changing levels of consciousness
- well-articulated speech
- bizarre thought control

The person found unconscious[8]

Most likely causes to consider

- Drug overdose, including alcohol
- Head injury
- Postictal state (epilepsy or CVA)
- Hypoglycaemia or ketoacidosis
- Subarachnoid haemorrhage
- Respiratory failure
- Hypotension, including cardiac arrhythmias or myocardial infarction
- Infection, e.g. meningitis
- Psychogenic

Basic investigations

- Oxygen saturation
- Blood glucose
- Urine or blood drug profile
- Brain CT scan
- Lumbar puncture (CT scan permitting)
- Routine blood chemistry
- ECG

Initial management of the unconscious patient

The first principle of management of a person found unconscious is to keep them alive by maintaining the airway and the circulation. The basic management essentials are summarised in TABLE 64.8 .

Table 64.8 Basic management essentials

Keep the person alive (maintain airway and circulation)
Get history from witnesses
Examine the person
Give ‘coma cocktail’ (TONG)
Take blood (for investigations)
CT scan (if diagnosis doubtful)

Before embarking on a secondary survey always consider giving the ‘coma cocktail’ (also called TONG[2] or DONT (dextrose, oxygenation, naloxone, thiamine)), which refers to the combination of:

T = **T**hiamine	100 mg IM or IV
O = **O**xygenation	
N = **N**aloxone	0.1–0.2 mg IV
G = **G**lucose	i.e. 50 mL, 50% dextrose

The rapid administration of these agents should be considered for any patient[3,9] with an altered level of consciousness because they may lessen or reverse metabolic insult to the brain. Some emergency physicians recommend adding flumazenil to the cocktail, but this is not supported by

others due to possible adverse effects including airway problems.[10]

In the presence of hypoventilation, constricted pupils[11] or circumstantial evidence of opioid use, naloxone (the specific opiate antagonist) should be given intravenously. If there is no response, the patient should be intubated before further naloxone is given. Use a nasogastric tube to prevent acute gastric dilatation.

Catheterise to relieve urinary distension, send a urine sample for micro and culture, pregnancy test and drug screen.

Use of flumazenil

Flumazenil is a specific benzodiazepine antagonist and may have an important use in the assessment of the unconscious patient. It can have a dramatic effect on benzodiazepine overdosage. After an initial dose of 0.2 mg IV, if benzodiazepine overdose is a reasonable possibility, 0.3–0.5 mg boluses should be given every 1–2 minutes with caution, until a response is observed.[12]

Opioid (heroin) overdose

In a known overdose, treat initially with both IV and IM naloxone:

- naloxone 0.4 mg IV (repeat in 3 minutes if necessary)
- naloxone 0.4 mg IM (to maintain cover)

Practice tips[2]

- An unconscious person who is hypotensive is bleeding until proved otherwise.
- The presence of a head injury should not prevent rigorous resuscitation of the hypotensive person.
- Always suspect cervical injury in persons who are victims of time-critical trauma.
- Tachypnoea is a sign of inadequate oxygenation and not a sign of central nerve damage.
- Always suspect opioid overdosage in the ‘unknown’ person brought in with an altered conscious state.
- Consider administration of TONG—the ‘coma cocktail’.

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65 Urinary disorders

As men draw near the common goal,
Can anything be sadder
Than he who, master of his soul,
Is servant to his bladder?

ANONYMOUS, *SPECULUM*, 1938

Disturbances of micturition are a common problem in general practice, with an annual incidence of about 20 per 1000 patients at risk.[1] Such disturbances include dysuria, frequency of micturition, difficulty or inability to initiate micturition, stress incontinence and haematuria. These symptoms are three times as common in women as in men.[1] The combination of dysuria and frequency is the most common of the symptoms with an incidence of about 14 per 1000 patients and a female:male ratio of 5:1.[1]

Dysuria is present at least occasionally in approximately 3% of adults older than 40 years.[2] With the exception of enuresis (CHAPTER 84), disturbances of micturition are uncommon in children.

Dysuria and frequency

Dysuria (difficult and/or painful micturition [uralgia]) is characterised mainly by urethral and suprapubic discomfort, and indicates mucosal inflammation of the lower genitourinary tract (i.e. the urethra, bladder or prostate). The passage of urine across inflamed mucosa causes pain. Frequency can vary from being negligible to extreme. It can be ‘habit frequency’ or associated with anxiety, which is typically long term and worse with stress and cold weather. In these conditions urinalysis is normal. Sometimes haematuria and systemic symptoms can accompany dysuria and frequency.

A summary of the diagnostic strategy model for dysuria is presented in TABLE 65.1 .

Table 65.1 Dysuria: diagnostic strategy model

Probability diagnosis

UTI, esp. cystitis (female)
Urethritis
Urethral syndrome (female)
Vaginitis

Serious disorders not to be missed

Neoplasia:

- bladder
- prostate
- urethra

Severe infections:

- gonorrhoea
- chlamydia
- genital herpes

Reactive arthritis
Calculi (e.g. bladder)

Pitfalls (often missed)

Menopause syndrome
Adenovirus urethritis
Prostatitis
Foreign bodies in lower urinary tract (LUT)
Acute pelvic or retrocaecal appendicitis
Acidic urine
Acute fever
Interstitial cystitis
Urethral caruncle/diverticuli (usually postmenopausal)
Vaginal prolapse
Obstruction:

- benign prostatic hyperplasia
- urethral stricture
- phimosis
- meatal stenosis

Seven masquerades checklist

Depression
Diabetes
Drugs
UTI

Is the patient trying to tell me something?

Consider psychosexual problems, anxiety and hypochondriasis.

Key facts and checkpoints[1,3]

- Strangury = difficult and painful micturition with associated spasm.
- Inflammation usually results in the frequent passage of small amounts of urine and a sense of urgency.
- Urethritis usually causes pain at the onset of micturition.
- Cystitis usually causes pain at the end of micturition.
- Suprapubic discomfort is a feature of bladder infection (cystitis).
- Vesicocolonic fistulas (e.g. prostatic cancer) cause severe dysuria, pneumaturia and foul-smelling urine.
- Dysuria and frequency are most common in women aged 15–44 years.
- They are four times more common in sexually active women.
- Vaginitis is an important cause and must be considered.
- Dysuria and discomfort is a common feature of postmenopausal syndrome, due to atrophic urethritis. The urethra and lower bladder are oestrogen-dependent.
- Have a low threshold for testing for *Chlamydia* urethritis.
- Urinary infection and other disorders can be quite asymptomatic.

Is it really a urinary tract infection?

Although UTIs account for the majority of cases of dysuria in women it must be remembered that vaginitis and postmenopausal atrophic vaginitis can cause dysuria (see FIG. 65.1). Vaginitis is the most common cause of dysuria in the child and adolescent age group and is a relatively common cause of dysuria in family practice, estimated at around 15%. Postmenopausal oestrogen deficiency can cause recurrent dysuria, so consider prescribing topical oestrogen where appropriate. Acute bacterial cystitis accounts for about 40% of causes of dysuria.

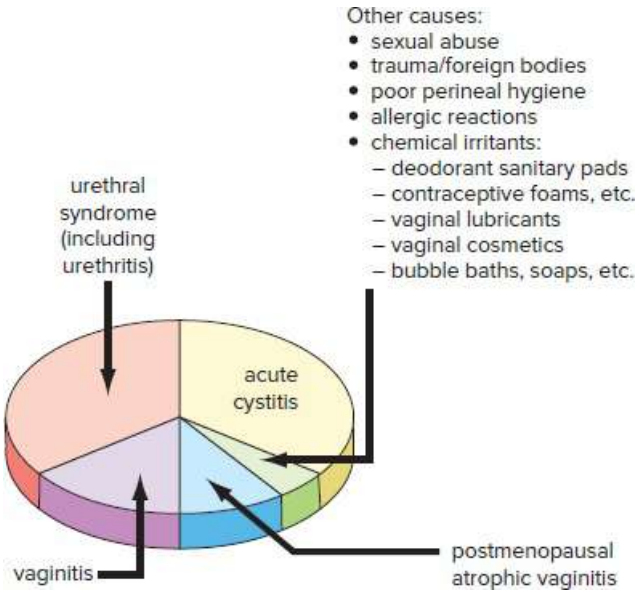

FIGURE 65.1 Relative causes of dysuria in women

The dysuria associated with vaginitis may be described as burning ‘on the outside’ with the discomfort usually felt at the beginning or end of micturition. It may be accompanied by vaginal irritation or discharge. If vaginitis is suspected, a pelvic examination should be carried out to inspect the genitalia and obtain swabs.[2]

Urethral syndrome

Also known as chronic sterile inflammatory disorder, abacterial cystitis, interstitial cystitis or ‘painful bladder syndrome’. It presents with lower urinary tract symptoms (LUTS), particularly suprapubic pain on bladder filling, or symptoms of urethritis (usually frequency and urgency; dysuria is variable). It affects women more than men. Management, which is difficult, is supportive.

The clinical approach

History

It is important to determine whether dysuria is really genitourinary in origin and not attributable to functional disorders, including psychosexual problems. Disturbances of micturition are uncommon in young males, and if present suggest venereal infection.

Key questions

- Could you describe the discomfort?

- What colour is your urine?
- Does it have a particular odour?
- Have you noticed a discharge?
- If so, could it be sexually acquired?
- Do you find intercourse painful or uncomfortable?
- Have you any fever, sweats or chills?

Examination

The general inspection and examination should include measurement of the basic parameters of pulse, temperature and blood pressure. The possibility of underlying kidney disease, especially in the presence of an obstructive component, should be kept in mind.

Abdominal palpation should focus on the loins and suprapubic areas. The possibility of sexually transmitted infections should also be considered; genital and rectal examination may be appropriate. In the menopausal female, a dry atrophic urethral opening, a urethral caruncle or urethral prolapse may give the clue to this important cause of dysuria.

Investigations

Basic investigations include:

- dipstick testing of urine
- microscopy and culture (midstream specimen of urine, or suprapubic puncture in children), and possibly urethral swabs or first pass urine for sexually transmitted infections
- where relevant, first pass urine NAAT (PCR test) for chlamydia and gonorrhoea

Further investigations depend on initial findings and referral for detailed investigation will be necessary if the primary cause cannot be found.

The management of urinary tract infection is presented in CHAPTER 16 .

Haematuria

Haematuria is the presence of blood in the urine and can vary from frank bleeding (macroscopic) to the microscopic detection of red cells. Haematuria can occur in a wide variety of disorders but a careful history and examination can often lead to the source of the bleeding and help with the selection of investigations. Macroscopic haematuria is often a sign of a serious underlying disorder.

Key facts and checkpoints

- Macroscopic haematuria is the presence of blood visible to the naked eye. It is always abnormal except in menstruating women.
- Small amounts of blood (1 mL/1000 mL urine) can produce macroscopic haematuria.
- Microscopic haematuria is the presence of blood in the urine that can be detected only by microscopic or chemical methods.
- Microscopic haematuria includes the presence of red blood cells (RBC) >8000 per mL of urine (phase contrast microscopy) or >2000 per mL of urine (light microscopy) representing the occasional RBC on microscopic examination.
- Joggers and athletes engaged in very vigorous exercise can develop transient microscopic or even macroscopic haematuria.
- Microscopic (asymptomatic haematuria) can be classified as either:

  glomerular (from kidney parenchyma): common causes are IgA nephropathy and thin membrane disease[4]

  or

  non-glomerular (urological): the common causes are bladder cancer, benign prostate hyperplasia and urinary calculi
- Common sources of macroscopic haematuria are the bladder, urethra, prostate and kidney.[5]
- Macroscopic haematuria occurs in 70% of people with bladder cancer and 40% with kidney cancer.[5]
- Common urological cancers that cause haematuria are the bladder (70%), kidney (17%), kidney pelvis or ureter (7%) and prostate (5%).[6]
- It is important to exclude kidney damage, so patients should have blood pressure, urinary protein (ACR) and plasma creatinine levels measured as a baseline.
- All patients presenting with macroscopic haematuria or recurrent microscopic haematuria require judicious investigation, which may involve both radiological investigation of the upper urinary system and visualisation of the lower urinary system to detect or exclude pathology.

The clinical approach

History

Is it really haematuria? In many patients the underlying disorder may be suspected from a detailed enquiry about associated urinary symptoms. The presence of blood can be verified rapidly by microscopy so that red discolouration due to haemolysis, beetroot or red food dye can be discounted.

The time relationship of bleeding is useful because, as a general rule, haematuria occurring in the first part of the stream suggests a urethral or prostatic lesion, while terminal haematuria suggests bleeding from the bladder. Uniform haematuria has no localising implications.

It is most unusual for haematuria to cause anaemia unless it is massive. Massive haematuria is a feature of radiation cystitis.

Painful haematuria is suggestive of infection (including sexually acquired urethritis), urethral caruncle, calculi or kidney infarction, while painless haematuria is commonly associated with infection, trauma, tumours or polycystic kidneys. Loin pain can occur as a manifestation of nephritis and may be a feature of bleeding in cancer of the kidney or polycystic kidney.

A drug history is relevant, especially with anticoagulants and cyclophosphamide. A diet history should also be considered.

It is worth noting that large prostatic veins, secondary to prostatic enlargement located at the bladder neck, may rupture when a man strains to urinate.

A summary of the diagnostic strategy model for haematuria is presented in TABLE 65.2 .

Table 65.2 Haematuria: diagnostic strategy model

Probability diagnosis
Infection: • cystitis • urethritis • pyelonephritis • prostatitis Calculi—kidney, ureteric, bladder
Serious disorders not to be missed
Cardiovascular: • kidney infarction

- kidney vein thrombosis
- prostatic varices

Neoplasia:

- kidney tumour
- urothelial: bladder, kidney, pelvis, ureter
- prostate cancer

Severe infections:

- infective endocarditis
- kidney tuberculosis
- blackwater fever

Glomerulonephritis (e.g. post-streptococcal, IgA nephropathy)
Kidney papillary necrosis
Other kidney disease, e.g. polycystic kidneys, medullary sponge kidney

Pitfalls (often missed)

Urethral prolapse/caruncle
Pseudohaematuria (e.g. beetroot, porphyria)
Haemorrhagic cystitis
Benign prostatic hyperplasia
Trauma: blunt or penetrating
Foreign bodies
Bleeding disorders
Vigorous exercise
Radiation cystitis
Menstrual contamination
Rarities:

- hydronephrosis
- Henoch–Schönlein purpura
- bilharzia
- schistosomiasis
- polycystic kidneys
- benign renal masses
- endometriosis (bladder)
- systemic vasculitides

Seven masquerades checklist

Drugs (cytotoxics, anticoagulants)
UTI

Is the patient trying to tell me something?

Consider artefactual haematuria.

Key questions

- Have you had an injury such as a blow to the loin, pelvis or genital area?
- Is the redness at the start or end of your stream or throughout the stream?
- Have you noticed any bleeding elsewhere, such as bruises or nose bleeds?
- Have you experienced any pain in the loin or abdomen?
- Have you noticed any burning or frequency of your urine?
- Have you had any problems with the flow of your urine?
- Have you been having large amounts of beetroot, red lollies or berries in your diet?
- Could your problem have been sexually acquired?
- Have you been overseas recently?
- Have you been exposed to chemicals (e.g. dyes, rubber) in your work?
- Have you been aware of any other symptoms?
- Do you engage in strenuous sports such as jogging?
- Have you had any kidney problems in the past?
- Are you taking blood-thinning drugs?

Examination

The general examination should include looking for signs of a bleeding tendency and anaemia, and recording the parameters of temperature, blood pressure and the pulse (see FIG. 65.2). The heart should be assessed to exclude atrial fibrillation or infective endocarditis with emboli to the kidney, and the chest should be examined for a possible pleural effusion associated with perinephric or kidney infections.

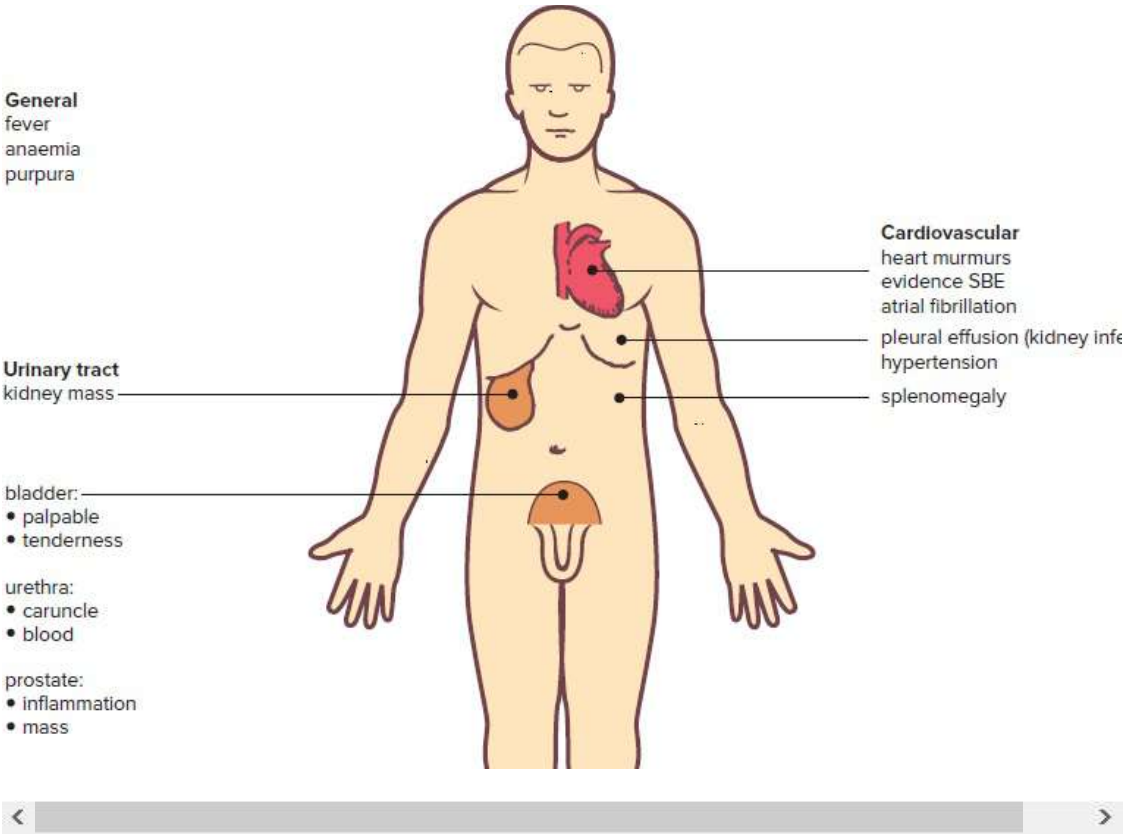

FIGURE 65.2 Features to consider in the physical examination of the patient with haematuria

The abdomen should be examined for evidence of a palpable enlarged kidney or spleen. The different clinical findings for an enlarged left kidney and spleen are shown in TABLE 65.3 . Kidney enlargement may be due to kidney tumour, hydronephrosis or polycystic disease. Splenomegaly suggests the possibility of a bleeding disorder.

Table 65.3 Differences between spleen and left kidney on abdominal examination

	Spleen	Left kidney
Palpable upper border	Impalpable	Palpable
Movement with inspiration	Inferomedial	Inferior
Notch	Yes	No
Ballotable	No	Yes
Percussion	Dull	Resonant (usually)
Friction rub	Possible	Not possible

The suprapubic region should be examined for evidence of bladder tenderness or enlargement. In men the prostate should be examined rectally to detect benign or malignant enlargement or tenderness from prostatitis.

In women, consider a pelvic examination to search for possible pelvic masses. The urethral meatus should be inspected to exclude a urethral caruncle ('raspberry tumour') or urethral prolapse.

Investigations

It is important to identify the cause, especially if a possible sequela is impaired kidney function.

- Urinalysis by dipstick testing (*note:* high vitamin C intake can interfere).
- Urine microscopy:

  formed RBCs in true haematuria

  red cell morphology can assist in determining whether the source is glomerular or urinary tract

  red cell casts and dysmorphic red cells indicate glomerular origin
- Urinary culture: early culture is important because of the common association with infection and consideration of early treatment with antibiotics. If tuberculosis is suspected, three early morning urines should be cultured for tubercle bacilli.
- Urinary cytology: this test, performed on a urine sample, may be useful to detect malignancies of the bladder and lower tract but is usually negative with kidney cancer. Sensitivity can be increased by testing mid morning or random specimens from three separate voids.
- Blood tests: appropriate screening tests include a full blood count, ESR and basic kidney function tests (urea and creatinine). If glomerulonephritis is suspected, antistreptolysin O titres and serum complement levels should be measured. PSA is appropriate if prostate cancer is suspected.
- Radiological techniques—available tests include:[7]

  intravenous urography (IVU); intravenous pyelogram (IVP)—previously the key investigation, largely superseded by CT

  ultrasound (less sensitive at detecting LUT abnormalities)

  CT (with or without contrast), e.g. KUB (kidney, ureter, bladder)

  MRI

kidney angiography

retrograde pyelography

- Direct imaging techniques: these include urethroscopy, cystoscopy and ureteroscopy. Unless a renal cause is identified, referral to urologist for consideration of cystoscopy is advisable.
- Kidney biopsy: indicated if glomerular disease is suspected.

Red flags for urgent surgical intervention

- Anuria
- Single kidney
- Bilateral obstruction
- Concurrent UTI and urolithiasis

Pseudohaematuria

Pseudohaematuria is red urine caused by pigments other than red blood cells that simply stain the culture red.

Causes include:

- anthocyanins in food (e.g. beetroot, berries)
- red-coloured confectionery
- porphyrins
- free haemoglobin (e.g. haemoglobinuria)
- myoglobin (red-black colour)
- drugs (e.g. pyridium, phenolphthalein—alkaline urine)

Exercise haematuria

Exercise or sports haematuria is the passage of a significant number of red cells in the urine during or immediately after exercise, particularly long runs with under-hydration. It has been recorded in a wide variety of athletes, including swimmers and rowers. Dipstick testing is usually positive in these athletes. Despite the theory that it is largely caused by the posterior wall of the bladder impacting repetitively on the base of the bladder during running, there are other possible

factors and glomerular disease must be excluded in the athlete with regular haematuria, especially if dysmorphic red cells are found on microscopy.

Artefactual haematuria

Macroscopic haematuria can be a presenting symptom of people with Munchausen syndrome or those seeking opioids by simulating renal colic. If suspected, it is wise to get these people to pass urine in the presence of an appropriate witness before examining the urine.

Urethral caruncle

This is a benign granulomatous tumour about the size of a pea in the distal urethra. Almost exclusive to postmenopausal women, it is very tender and bleeds easily. The main symptom is haematuria. It may require cystoscopy and biopsy for diagnosis. Treatment includes warm salt baths and oestrogen creams. Otherwise obliteration by laser vaporisation, cryotherapy, cautery or even surgical excision is used.

Bladder cancer[8]

Bladder cancer is the seventh most common malignancy, with 90% being transitional cell carcinomas. Other forms include squamous cell carcinoma and adenocarcinoma. Smoking is the most common association. There is little evidence that drinking chlorinated water can predispose.[9]

Clinical features

- Haematuria/microhaematuria
- Irritative symptoms: frequency, urgency, nocturia
- Dysuria

Diagnosis

- Urine cytology: three specimens
- Cystoscopy and biopsy
- Imaging of upper tracts: ultrasound, CT IV urethrogram is the gold standard
- Differential diagnosis is haemorrhagic cystitis

Management

Treatment depends on the staging and grading.

- The common carcinoma in situ is treated with intravesical BCG immunotherapy. This 6-week course and follow-up if necessary leads to 60–75% remission.
- Other intravesical agents used include various cytotoxics (e.g. mitomycin C).
- Other treatments include surgery such as tumour resection plus intravesical agents, bladder resection (partial or total) and radio–chemotherapy.

Regular surveillance, which may be lifelong, is essential.

Glomerulonephritis[10]

Glomerulonephritis means kidney inflammation involving the glomeruli. It can be simply classified into:

- acute nephritic syndrome: haematuria + dysmorphic RBCs/casts + hypertension
- nephrotic syndrome: oedema + hypoalbuminaemia + proteinuria Page 789
- asymptomatic kidney disease

DxT haematuria + dysmorphic RBCs/casts + hypertension → acute nephritic syndrome

The main causes of glomerulonephritis–nephritic syndrome are:

- IgA nephropathy (Berger disease—most common)
- anti-glomerular basement membrane disease (has an AD genetic link), aka Goodpasture disease
- post-streptococcal glomerulonephritis
- systemic vasculitis—ANCA associated
- others, e.g. SLE

Nephritic syndrome

Feature is haematuria + dysmorphic RBCs and casts (microscopy) plus one of following:

- proteinuria (mild to moderate)
- hypertension

- oedema
- ↑ s. creatinine
- oliguria

IgA nephropathy

Typically presents as haematuria (smokey urine) in a young male adult at the time of or within 1–2 days of a mucosal infection (usually throat, influenza or URTI) and persists for several days. Other presentations are as incidentally found microscopic haematuria or as previously unsuspected chronic kidney failure.

Due to deposition of IgA antibody complexes in the glomeruli, it runs a variable course, but prognosis is usually good. There is no specific treatment to date but immune suppression may be used. Refer suspected cases immediately. Diagnosis is by biopsy.

Acute post-streptococcal glomerulonephritis

Typically seen in children (>5 years), especially in Aboriginal and Torres Strait Islander communities following GABHS throat infection or impetigo. Presents after a gap of 1–2 weeks.

Clinical features

- Irritable, lethargic, sick child
- Haematuria: discoloured urine (‘Coke’ urine)
- Peri-orbital oedema (may be legs, scrotum)
- Rapid weight gain (from oedema)
- Scanty urine output (oliguria)
- Hypertension → may be complications

Usual course

- Oliguria 2 days
- Oedema and hypertension 2–4 days
- Invariably resolves
- Good long-term prognosis

Diagnosis

- GABHS antigens
- Blood urea, creatinine, $C_{3\&4}$ (complement), ASOT, anti-DNase B

Treatment

- Hospital admission
- Bed rest
- Strict fluid balance chart
- Daily weighing
- Penicillin (if GABHS +ve)
- Fluid restriction
- Low protein, high carbohydrate, low salt diet
- Antihypertensives and diuretics (as necessary)

Follow-up: monitor BP and kidney function. Regular urinalysis (microscopic haematuria may last for years).

DxT discoloured urine + peri-orbital oedema + oliguria → post-streptococcal glomerulonephritis

Proteinuria

Proteinuria is an important and common sign of kidney disease. The protein can originate from the glomeruli, the tubules or the LUT. Healthy people, however, do excrete some protein in the urine, which can vary from day to day and hour to hour; hence the value of collecting it over 24 hours or comparing the albumin against the standard rate of filtered creatinine (i.e. ACR). While proteinuria can be benign, it always requires further investigation. Important causes of proteinuria are presented in TABLE 65.4 .

Table 65.4 Important causes of proteinuria

Transient
Contamination from vaginal secretions
Urinary tract infection
Pre-eclampsia

Note: These all require exclusion and follow-up.

Kidney disease

Glomerulonephritis

Nephrotic syndrome

Congenital tubular disease, e.g.

- polycystic kidney
- kidney dysplasia

Acute tubular damage

Kidney papillary necrosis, e.g.

- analgesic nephropathy
- diabetic papillary necrosis

Overflow proteinuria, e.g.

- multiple myeloma, monoclonal gammopathy

Systemic diseases/conditions affecting the glomeruli:

- diabetes mellitus
- hypertension
- SLE
- pre-eclampsia
- malignancy
- drugs (e.g. penicillamine, gold salts)
- amyloid
- vasculitides

No kidney disease

Orthostatic proteinuria

Exercise

Emotional stress

Fever

Cold exposure

Postoperative

Acute medical illness (e.g. heart failure)

Key facts and checkpoints[11]

- The amount of protein in the urine is normally less than 100 mg/24 hours.
- Greater than 300 mg/24 hours is abnormal in children and adults.

- If accompanied by dysmorphic haematuria or red cell casts, this tends to confirm glomerular origin.
- Routine dipstick testing will detect levels greater than 300 mg/24 hours (ACR of 30–300 mg/g) only and thus has limitations.[12]
- In diabetics, microalbuminuria is predictive of nephropathy and an indication for early blood pressure treatment.

Urine albumin–creatinine ratio (ACR)

Remember the rough 'rule of 3' for ACR levels (units are mg/g):[13]

- <3 is normal
- 3–30 is mild elevation
- 30–300 is moderate ('trace protein' on dipstick)
- >300 is severe albuminuria.

If proteinuria is confirmed on repeated dipstick testing it should be measured more accurately by measuring the albumin–creatinine ratio (ACR), which is preferable to a 24-hour urinary protein as it avoids finicky 24-hour collections. Refer to CHAPTER 79 . High values require referral for investigation. The minimum investigations are microurine and assessment of kidney function (eGFR). Nephrotic range proteinuria (>3 g/24 hours) is due to one or other form of glomerulonephritis in over 90% of patients.[10] Possible contamination from vaginal secretions or from a low UTI needs to be excluded.

Orthostatic proteinuria

Orthostatic proteinuria is the presence of significant proteinuria after the patient has been standing but is absent from specimens obtained following recumbency for several hours, such as an early morning specimen.

It occurs in 5–10% of people,[6] especially during their adolescent years. In the majority it is of no significance and eventually disappears without the development of significant kidney disease. However, in a small number the proteinuria can foreshadow serious kidney disease.

Diabetic microalbuminuria

The presence of protein in the urine is a sensitive marker of diabetic nephropathy, so regular screening for microalbuminuria in those with diabetes is an important predictor of nephropathy

and other possible complications of diabetes. The use of antihypertensives, particularly ACEi and ARBs, at the microalbuminuria stage may slow the development of overt nephropathy.

Consequences of proteinuria

While proteinuria is usually simply a marker of kidney disease, heavy proteinuria in excess of 3 g/24 hours may have severe clinical consequences, including oedema, intravascular volume depletion, venous thromboembolism, hyperlipidaemia and malnutrition.

Minimal change glomerulonephritis is the commonest cause of the nephrotic syndrome in childhood and accounts for about 30% of adult nephrotic syndrome.[8] It is steroid responsive.

Nephrotic syndrome[10,11]

DxT proteinuria + generalised oedema + hypoalbuminaemia → nephrotic syndrome

Nephrotic syndrome occurs at any age but is more common in children and has primary and secondary causes that require elucidation.

Clinical features

- Proteinuria >3 g/day (+++ or ++++[1] on dipstick)
- Swelling of eyelids and face
- Generalised oedema, especially peripheral oedema
- Hypertension
- Hypoalbuminaemia <30 g/L
- Hypercholesterolaemia >4.5 mmol/L
- Waxy pallor
- Normal BP
- Dyspnoea
- Frothy urine

Predisposes to sepsis (e.g. peritonitis, pyelonephritis, thromboembolism).

Causes

- 1 in 3 (approx.):

  systemic kidney disease (e.g. diabetes, SLE, amyloid, hepatitis B/C)

- 2 in 3 (approx.):

  minimal change disease (most common)

  idiopathic nephrotic syndrome (based on kidney biopsy)

  focal glomerular sclerosis

  membranous nephropathy

  membranoproliferative glomerulonephritis

- Others: drugs, malignancy, infection, e.g. malaria

Glossary of terms

Functional incontinence Loss of urine secondary to factors extrinsic to the urinary tract (e.g. dementia, endocrine causes).

Nocturnal enuresis (or bed-wetting) Involuntary urine loss during sleep.

Overactive bladder (detrusor instability) The most common cause of urge incontinence; synonymous with an irritable or unstable bladder; characterised by involuntary bladder contractions, resulting in a sudden urge to urinate, usually accompanied by frequency and nocturia, with or without incontinence.

Overflow incontinence Escape of urine following poor bladder emptying.

Stress incontinence The involuntary loss of urine on coughing, sneezing, straining or lifting, or any factor that suddenly increases intra-abdominal pressure.

Urge incontinence Involuntary loss of urine associated with urgency.

Urinary incontinence The involuntary loss of urine during the day or night.

Treatment

- Immediate referral to renal physician or unit
- Bed rest
- Treat causative disorder

- Diet modification: low fluid, protein consideration, low salt

Medication may include:

- diuretics
- ACE inhibitors or ARBs
- prednisolone
- pneumococcal vaccination
- penicillin
- statins
- aspirin

Urinary Incontinence

A summary of the types of incontinence and their causes is presented in TABLE 65.5 .

Table 65.5 Types of incontinence and their implied causes

Type of incontinence	Likely cause
Stress incontinence	Pelvic floor or sphincter weakness
Urge incontinence	Detrusor overactivity or low compliance
Mixed urinary incontinence	Both pelvic floor weakness and detrusor overactivity
Quiet dribble incontinence	Variable, may be due to overflow (i.e. incomplete emptying)
Continuous leakage	Fistula, ectopic ureter, patulous urethra
Reflex incontinence (without warning)	Neuropathic bladder

Female urinary incontinence

Urinary leakage affects around 37% of adult women in Australia.[14] Despite its prevalence, many women will not seek treatment. The most common types are stress incontinence, urge incontinence and mixed (both stress and urge).

Assessment

The basic assessment of the person with incontinence requires a careful history and examination, exclusion of infection and the keeping of a micturition or bladder chart. Use of a severity index questionnaire is very helpful. Drugs that adversely affect urinary function are presented in TABLE 65.6 . If central nervous system pathology is suspected, referral to a neurologist is required.

Table 65.6 Drugs that can cause or aggravate incontinence

- ACE inhibitors
- phenoxybenzamine (Dibenyline)
- prazosin
- labetalol

Bladder relaxants → overflow incontinence:

- anticholinergic agents
- tricyclic antidepressants

Bladder stimulants → urge incontinence:

- cholinergic agents
- caffeine

Sedatives → urge incontinence:

- antidepressants
- antihistamines
- antipsychotics
- hypnotics
- tranquillisers

Others → urge incontinence:

- alcohol
- loop diuretics (e.g. frusemide), other diuretics
- lithium

Investigations

Urine microscopy and culture is important to exclude infection, haematuria and glycosuria. Urinary tract ultrasound is often appropriate to measure the post residual volume (>100 mL is abnormal). Urodynamic studies may be required to dispel doubt about the diagnosis, especially with suspected voiding difficulty, neuropathy, failed treatment or when considering surgery.

Management

Stress incontinence[17]

- Pelvic floor muscle training (see below)
- Weight reduction if obese
- Vaginal oestrogen therapy may help some postmenopausal women
- Referral for surgical therapy is appropriate if symptoms are severe or unresponsive to conservative treatment

Causes of incontinence

A mnemonic: $DIAP_2E_2RS_2$:[15,16]

D = **D**elirium/dementia

I = **I**nfection of urinary tract

A = **A**trophic urethritis

P = **P**harmacological (e.g. diuretics)

P = **P**sychological (e.g. acute distress)

E = **E**ndocrine (e.g. hypercalcaemia, diabetes insipidus)

E = **E**nvironmental (e.g. unfamiliar surrounds)

R = **R**estricted mobility

S = **S**tool impaction

S = **S**phincter damage or weakness

The severity index questionnaire

- How often do you experience urine leakage?

  0 = never

  1 = less than once a month

2 = one or several times a month

3 = one or several times a week

4 = every day and/or night

- How much urine do you lose each time?

  1 = drops or little

  2 = more

The total score is the score for the first question multiplied by the score for the second question.

0 = dry, 1–2 = slight, 3–4 = moderate, 6–8 = severe

Pelvic floor muscle training[18,19]

- Evidence is strong. Compared to placebo or no treatment, women with stress incontinence given pelvic floor training were eight times more likely to report a ‘cure’ (56% vs 6%).
- Best in motivated young women with stress incontinence.
- At least 3 months trial with supervision (physiotherapist or continence nurse).

Basic techniques:

- Advise the patient to pull up her pelvic muscles to imagine herself stopping passing urine (or controlling diarrhoea) and hold the ‘squeeze’ for a count of 10. Repeat many times daily. Refer to the *Patient Education* hand-out at the end of this chapter.

Urge incontinence

- Lifestyle intervention:

  Alter fluid intake—decrease to reduce urinary frequency, increase to improve urine concentration

  Reduce intake of bladder irritants—caffeine, alcohol, carbonated beverages

  Avoid constipation

- Bladder retraining

  includes pelvic floor muscle therapy, a scheduled voiding program with gradual increases in the duration between voids, urge suppression techniques with distraction or relaxation

a basic first step is to delay voiding when possible and wait for the urge to pass

best performed with supervision of a physiotherapist or continence nurse

- Anticholinergic/antispasmodic drugs (see below)

- Vaginal oestrogen therapy may help some post menopausal women
- Intravesical treatment with botulinum toxin (second line)
- Neuromodulation—electrical stimulation of the sacral nerve (third line)

Anticholinergic/antispasmodic drugs[18]

These may be worth a trial for overactive bladder or urge incontinence. They are less effective for stress incontinence:

- oxybutynin 2.5–5 mg (o) bd or tds
- oxybutynin transdermal patch 3.9 mg (top) twice weekly
- solifenacin 5–10 mg (o) daily
- tolterodine 2 mg (o) bd
- darifenacin 7.5–15 mg (o) daily

Bladder dysfunction (in women during night)

Women with urethral syndrome constantly wake at night with urge to micturate but produce only a small dribble of urine.

- Instruct patient to perform a pelvic tilt exercise by balancing on upper back, lifting her pelvis with knees flexed and holding position for 30 seconds
- Squeeze pelvic floor inwards (as though holding back urine or faeces)
- Repeat a few times

Uterovaginal prolapse[20]

Uterovaginal prolapse is very common, eventually affecting 50% of parous women. The main complaint is of ‘heaviness’ in the vagina and a sensation of ‘something coming down’. Relevant symptoms that are of considerable distress (depending on the type of prolapse) include voiding difficulties, urinary stress incontinence, faecal incontinence, incomplete rectal emptying and recurrent cystitis. Backache is a common associated symptom, usually relieved by lying down.

Classification of prolapse

See FIGURE 65.3 .

- Cystocele—bladder descends into vagina
- Urethrocele—urethra bulges into vagina
- Rectocele—rectum protrudes into vagina
- Enterocele—loop of small intestine bulges into vagina (usually posterior wall)
- Uterine—uterus and cervix descend towards vaginal introitus:

  first degree—cervix remains in vagina

  second degree—cervix protrudes on coughing/straining

  third degree (procidentia)—uterus lies outside vagina

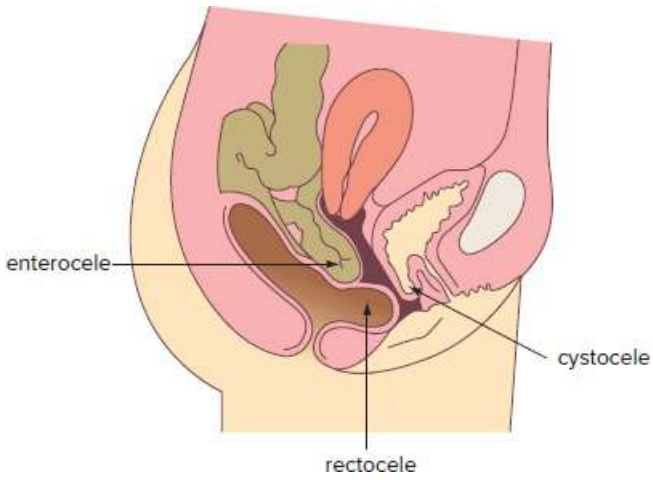

FIGURE 65.3 Uterovaginal prolapse

Examination

This is best performed with women in the left lateral position using a Sims speculum or posterior blade of the Graves speculum. Ask the patient to cough or bear down (several times)—observe anterior, posterior and lateral vaginal walls and descent of cervix.

Management

As a rule asymptomatic prolapse does not need invasive treatment, just basic reassurance and education, including pelvic floor exercises (see earlier in this chapter). Consider referral to a physiotherapist. Lifestyle measures include optimal nutrition, weight loss if obese, smoking

cessation and exercise. Aggravating comorbidities such as constipation, atrophic vaginitis and COPD require optimal treatment. Consider topical oestrogens in post menopausal women.

Prevention

Promote optimal obstetric management, especially postpartum exercises, lifelong pelvic floor exercises, ideal weight and sensible bladder and bowel function.

Ring pessaries

Pessaries are an option for those who are poor anaesthetic risks, too frail for surgery, don't want surgery, are young and have not completed their family or are awaiting surgery. The correct-sized pessary needs to be fitted individually. Topical oestrogens will improve comfort. The pessary needs to be cleaned or changed every 4–6 months.

Surgery

Refer to a gynaecological surgeon if a woman who is fit for surgery has symptomatic prolapse that warrants surgery, especially with associated voiding problems or obstructed defecation. The principles of reconstructive pelvic surgery are to:

- reposition pelvic structures to normal anatomical relationships
- restore and maintain urinary and/or faecal continence
- maintain sexual function
- correct coexisting pelvic pathology

Options include repair procedures (vaginally, sometimes abdominally, per laparoscopy), colpo/vaginal suspension and hysterectomy (vaginal or abdominal).

Patient education resource

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

- Incontinence of urine

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66 Visual failure

All those, therefore, who have cataract see the light more or less, and by this we distinguish cataract from amaurosis and glaucoma, for persons affected with these complaints do not perceive the light at all.

PAUL OF AEGINA (615–690 CE)

The commonest cause of visual dysfunction is a simple refractive error. However, there are many causes of visual failure, including the emergency of sudden blindness, a problem that requires a sound management strategy. Apart from migraine, virtually all cases of sudden loss of vision require urgent treatment.

The ‘white’ eye or uninflamed eye presents a different clinical problem from the red or inflamed eye.[1] The ‘white’ eye is painless and usually presents with visual symptoms and it is in the ‘white’ eye that the majority of blinding conditions occur.

Criteria for blindness and driving

This varies from country to country. The WHO defines blindness as ‘best visual acuity less than 3/60’, while in Australia eligibility for the blind pension is ‘bilateral corrected visual acuity less than 6/60 or significant visual field loss’ (e.g. a patient can have 6/6 vision but severely restricted fields caused by chronic open-angle glaucoma). The minimum standard for driving is 6/12 (Snellen system) in the better eye or bilaterally. Commercial licence standards are stricter (see Austroads guidelines).

The clinical approach

History

The history should carefully define the onset, progress, duration, offset and the extent of visual loss. An accurate history is important because a visual defect (particularly unilateral) may only just have been noticed by the patient, even if it has been longstanding. Two questions need to be answered:

- Is the loss unilateral or bilateral?
- Is the onset acute, or gradual and progressive?

Key facts and checkpoints

- The commonest cause of blindness in the world is trachoma. The other major causes of gradual blindness are cataracts, onchocerciasis and vitamin A deficiency.[2]
- In Western countries, the commonest causes are senile cataract, glaucoma, age-related macular degeneration, trauma and diabetic retinopathy.[2]
- The commonest causes of sudden visual loss are migraine and transient occlusion of the retinal artery (amaurosis fugax).[3]
- 'Flashing lights' are caused by traction on the retina and may have a serious connotation: the commonest cause is vitreoretinal traction, which is a classic precursor to retinal detachment.
- The presence of floaters or 'blobs' in the visual fields indicates pigment in the vitreous: causes include vitreous haemorrhage and vitreous detachment.
- Posterior vitreous detachment is the commonest cause of the acute onset of floaters, especially with advancing age.
- Retinal detachment has a tendency to occur in short-sighted (myopic) people.
- Suspect a macular abnormality where objects look smaller or straight lines are bent or distorted.

The distinction between central and peripheral visual loss is useful. Central visual loss presents as impairment of visual acuity and implies defective retinal image formation (through refractive error or opacity in the ocular media) or macular or optic nerve dysfunction. Peripheral field loss is more subtle, especially when the onset is gradual, and implies extramacular retinal disease or a defect in the visual pathway.

It is important to differentiate the central field loss of macular degeneration from the hemianopia of a CVA.

A drug history is very important (see TABLE 66.1). Tuberculosis treatment with ethambutol or quinine/chloroquine can be oculotoxic. The family history is relevant for diabetes, migraine, Leber hereditary optic atrophy, Tay–Sachs disease and retinitis pigmentosa.

Table 66.1 Systemic drugs that can cause ocular side effects

Alcohol/ethanol/methanol
Amiodarone
Antihypertensives e.g. β Blockers
Bisphosphonates
Chloroquine/hydroxychloroquine/quinine
Cyclosporin
Cytotoxic agents, e.g. vincristine
Corticosteroids
Disulfiram
Erectile dysfunction agents
Ethambutol
Indomethacin
Nitrofurantoin
Phenothiazines
Phenytoin
Tacrolimus
Tamsulosin
Tamoxifen
Tetracyclines, e.g. minomycin
Thiazides
Tricyclics
Topiramate

Questions directed to specific symptoms

- Presence of floaters → normal ageing (especially ≥55 years) with posterior vitreous detachment or may indicate haemorrhages or choroiditis
- Flashing lights → normal ageing with posterior vitreous detachment or indicates traction on the retina (?retinal detachment)
- Coloured haloes around lights → glaucoma, cataract

- Zigzag lines → migraine
- Vision worse at night or in dim light → retinitis pigmentosa, hysteria, syphilitic retinitis
- Headache → temporal arteritis, migraine, benign intracranial hypertension
- Central scotomata → macular disease, optic neuritis
- Pain on moving eye → retrobulbar neuritis
- Distortion, micropsia (smaller), macropsia (larger) → macular degeneration
- Visual field loss:

    central loss—macular disorder

    total loss—arterial occlusion

    peripheral loss

It is worth noting that if a patient repeatedly knocks into people and objects on a particular side (including traffic accidents), a bitemporal or homonymous hemianopia should be suspected.

Diseases/disorders to exclude or consider

- Diabetes mellitus
- Giant cell (temporal) arteritis
- Hypopituitarism (pituitary adenoma)
- Cerebrovascular ischaemia/carotid artery stenosis (emboli)
- Multiple sclerosis
- Cardiac disease (e.g. arrhythmias, and SBE—emboli)
- Anaemia (if severe can cause retinal haemorrhage and exudate)
- Marfan syndrome (subluxated lenses)
- Malignancy (the commonest cause of eye malignancy is melanoma of the choroid)

Examination

The same principles of examination should apply as for the red eye. Testing should include:

- visual acuity (Snellen chart)—with pinhole testing

- pupil reactions, to test afferent (sensory) responses to light
- confrontation fields (using a red pin)
- fundus examination with dilated pupil (ophthalmoscope), noting:

    the red reflex

    appearance of the retina, macula and optic nerve

Depending on circumstances, also consider:

- Amsler grid (or graph paper)
- colour vision (Ishihara chart)
- tonometry

General examination

General examination should focus on the general features of the patient, the nervous system, endocrine system and cardiovascular system.

Slit-lamp examination (biomicroscopy)

Usually performed with fluorescein dye, it provides a precise stereoscopic view of the eyelids, conjunctiva, cornea, iris, sclera, anterior chamber (measures depth), lens, anterior vitreous and retina. It is very useful for identifying corneal lesions, uveitis and scleritis.

Perimetry

Various defects in the visual fields are depicted in FIGURE 66.1 .

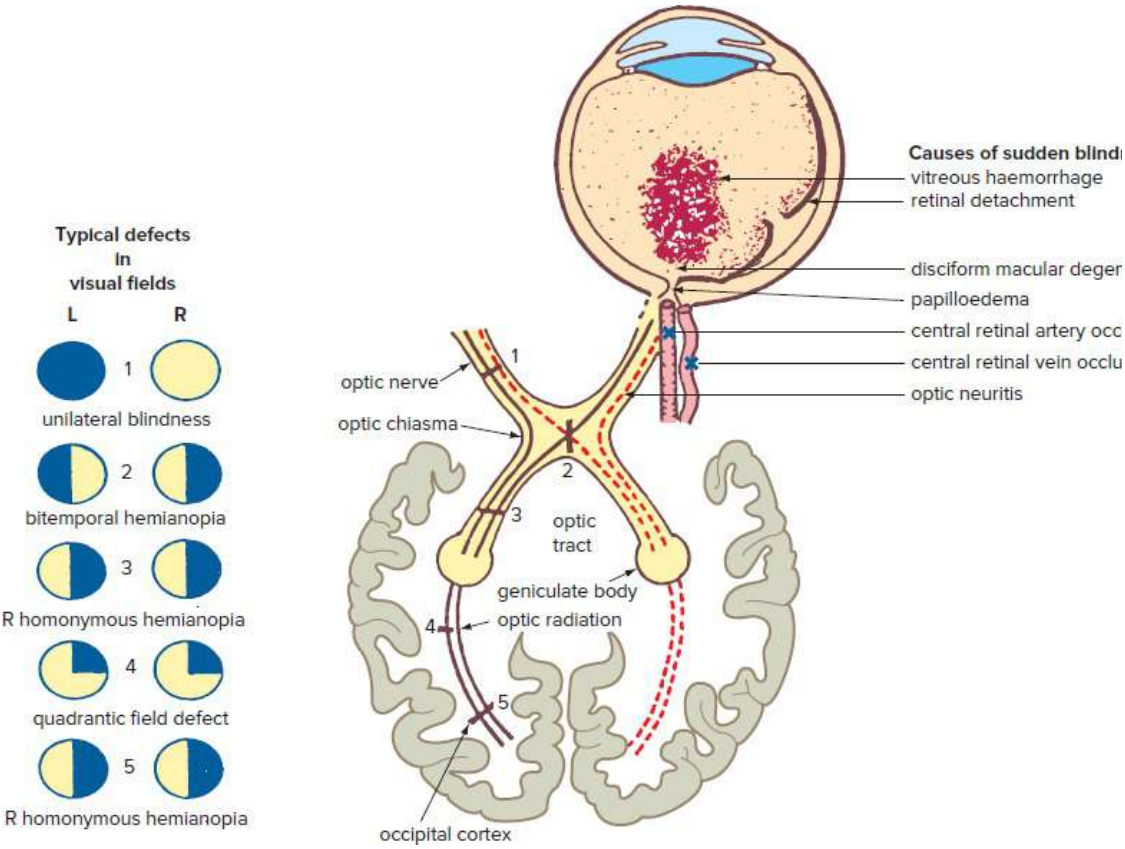

FIGURE 66.1 Diagrammatic representation of important causes of sudden painless loss of vision (right side) and typical defects in the visual fields (left side)

Investigations

Depending on the clinical examination, the following tests can be used selectively to confirm the diagnosis:

- blood tests:

  full blood (?anaemia, lead poisoning, leukaemia)

  ESR (?temporal arteritis)

  blood sugar (?diabetes mellitus)

- temporal artery biopsy (?temporal arteritis)
- CT/MRI scan (?CVA, optic nerve lesions, space-occupying lesions)

- formal perimetry and Bjerrum screen
- fluorescein angiography (?retinal vascular obstruction, diabetic retinopathy)
- visual evoked responses (?demyelinating disorders)
- carotid Doppler ultrasound
- B-scan ocular ultrasound

Visual failure in children

There are long lists of causes for visual failure or blindness in children. An approximate order of frequency of causes of blindness in children is cortical blindness, optic atrophy, choroidoretinal degeneration, cataract and retinopathy of prematurity. Almost half the causes of blindness in Western nations are genetically determined, in contrast to the nutritional and infective causes that predominate in developing countries.[4] About 3% of children will fail to develop proper vision in at least one eye.

The eyes of all babies should be examined at birth and at 6 weeks.

Amblyopia

Amblyopia, referred to as ‘lazy eye’, is defined as a reduction in visual acuity due to abnormal visual experience in early childhood. It is the main reason for poor unilateral eyesight until middle age and is usually caused by interference with visual development during the early months and years of life.

The common causes are:

- strabismus
- large refractive defect, especially hypermetropia
- congenital cataract

Principles of management[5]

- Most cases are treatable.
- Early diagnosis and intervention is fundamental to achieving useful vision.
- No child is too young to have the visual system assessed.
- The good eye should be patched in order to utilise the affected eye.
- Remove a remedial cause such as strabismus.

- Correct any refractive error, usually by prescription of glasses.

Some important guidelines in children

Referral

Refer if any of the following are present in infants:

- nystagmus
- a wandering eye
- a lack of fixation, or lack of following movements
- photophobia
- opacities (seen with ophthalmoscope set on +3, held 30 cm from baby's eye)
- delayed development

Strabismus

- The two serious squints are the constant and alternating ones, which require early referral. Transient squint and latent squint (occurs under stress, e.g. fatigue) usually are not a problem.
- Always refer children with strabismus (squint) when first seen to exclude ocular pathology such as retinoblastoma, congenital cataract and glaucoma, which would require emergency surgery.
- Children with strabismus (even if the ocular examination is normal) need specialist management because the deviating eye will become amblyopic (a lazy eye with reduced vision, i.e. 'blind') if not functioning by 7 years of age. The younger the child, the easier it is to treat amblyopia; it may be irreversible if first detected later than school age. Surgical correction of a true squint is preferred at 1–2 years of age. (See also CHAPTER 85 .)

Cataracts

Children with suspected cataracts must be referred immediately; the problem is very serious as the development of vision may be permanently impaired (amblyopia).[2] Cataracts are diagnosed by looking at the red reflex and this should be a routine part of the examination of a young child. Common conditions causing cataracts are genetic disorders and rubella but most causes are unknown. Rarer conditions, such as galactosaemia, need to be considered.

Refractive errors

Refractive errors, with the error greater in one eye, can cause amblyopia. Detection of refractive errors is an important objective of screening.

Retinoblastoma

Retinoblastoma, although rare, is the commonest intra-ocular tumour in childhood. It must be excluded in any child presenting with a white pupil. Such children also have the so-called ‘cat’s eye reflex’. In 30% of patients the condition is bilateral with an autosomal dominant gene being responsible.

Visual failure in the elderly

Most patients with visual complaints are elderly; failing vision affects perception of the environment and the ability to communicate effectively. Typical causes are cataracts, vascular disease, macular degeneration, chronic simple glaucoma and retinal detachment. Retinal detachment and diabetic retinopathy can occur at any age, although they are more likely with increasing age. Macular degeneration in its various forms is the commonest cause of visual deterioration in the elderly. For the elderly with cataracts the decision to operate depends on the patient’s vision and their ability to cope. Most patients with a vision of 6/18 or worse in both eyes usually benefit from cataract extraction, but some can cope with this level of vision and rely on a good, well-placed (above and behind) reading light.[6]

Sudden loss of vision in the elderly is suggestive of temporal arteritis or vascular embolism, so this problem requires immediate attention.

Floaters and flashes (photopsia)

When the vitreous gel shrinks as part of the normal ageing process, it tugs on the retina (rods and cones), causing flashing lights. When the gel separates from the retina, floaters (which may appear as dots, spots or cobwebs) are seen. Floaters are more commonly seen with age, but are also more common in people who are myopic or who have had eye surgery such as removal of cataracts. It is important to consider retinal detachment but if floaters remain constant there is little cause for concern. The appearance of a fresh onset of flashes or floaters is of concern, with the two important causes being retinal detachment and posterior vitreous detachment.

Haloes

Haloes around lights may result from cataracts, acute angle-closure glaucoma, chronic glaucoma, dry eyes and corneal haziness including excessive tears and/or mucus or drugs, e.g. chloroquine, digoxin.

Refractive errors

Indistinct or blurred vision is most commonly caused by errors of refraction—the commonest being myopia.

In the normal eye (emmetropia) light rays from infinity are brought to a focus on the retina by the cornea (contributing about two-thirds of the eye's refractive power) and the lens (one-third). Thus, the cornea is very important in refraction; abnormalities such as keratoconus may cause severe refractive problems.[6]

The important clinical feature is that where there is a refractive error only, the use of a simple 'pinhole' in a card will usually improve blurred vision or reduced acuity.[1]

Pinhole test

The pinhole reduces the size of the blur circle on the retina in the uncorrected eye. A pinhole acts as a universal correcting lens. If visual acuity is not normalised by looking through a card with a 1 mm pinhole, then the defective vision is not solely due to a refractive error. The pinhole test may actually help to improve visual acuity with some cataracts. Further investigation is mandatory.

Myopia (short/near-sightedness)

Close objects appear clearly but far objects are blurred. The image is focused in front of the retina. This is usually progressive in the teens. Highly myopic eyes may develop retinal detachment, macular degeneration or glaucoma.

Management

- Glasses with a concave lens
- Contact lenses
- Consider radial keratotomy or excimer laser surgery

Hypermetropia (long/far-sightedness)

The image is focused behind the retina. This condition is more susceptible to closed-angle glaucoma. In early childhood it may be associated with convergent strabismus (squint), and spectacle correction alone may straighten the eyes. It is mostly overcome by the accommodative power of the eye, though it may cause reading difficulty. Typically, the long-sighted person needs reading glasses at about 30 years. A positive converging (convex lens) is used for correction.

Presbyopia

The process of accommodation is required for focusing closer objects. This process, which relies on the action of ciliary muscles and lens elasticity, is usually affected by ageing, so that from the age of 45 close work becomes gradually more difficult.[6] There is a need for near correction with loss of accommodative power of the eye in the 40s.

Astigmatism

This results from non-spherical (variable) curvature of the lens or cornea. This creates the need for a corrective lens that is more curved in one meridian than another because the cornea does not have even curvature. If uncorrected, this may cause headaches of ocular origin. Conical cornea is one cause of astigmatism.

Keratoconus

Keratoconus is a bulging, slowly progressive thinning and distortion of the cornea, leading to loss of visual acuity—commonly irregular astigmatism. It usually appears between the ages of 10 and 25 and seems to be genetically determined. Frequent changes of glasses is a feature and contact lenses may help. If not, corneal transplant surgery may be necessary.

Cataract

The term ‘cataract’ describes any lens opacity. The symptoms depend on the degree and the site of opacity. Cataract causes gradual visual loss with normal direct pupillary light reflex.

The prevalence of cataracts increases with age: 65% at age 50–59, and all people aged over 80 have opacities.[3] Significant causes of cataracts are presented in TABLE 66.2 and causes of progressive visual loss in TABLE 66.3 .

Table 66.2 Causes of cataracts

Advancing age
Systemic disease, e.g. diabetes mellitus, myotonic dystrophy
Smoking cigarettes
Steroids (topical or oral)
Radiation: long exposure to UV light or X-rays
TORCH organisms → congenital cataracts
Trauma
Uveitis
Significant alcohol consumption
Malnutrition
Dystrophia myotonica
Galactosaemia → congenital cataract

Progressive bilateral visual loss

Table 66.3 Progressive bilateral visual loss

Globe	Chronic glaucoma Senile cataracts
Retina	Macular degeneration Retinal disease: • diabetic retinopathy • retinitis pigmentosa • choroidoretinitis
Optic nerve	Optic neuropathies Optic nerve compression (e.g. aneurysm, glioma) Toxic damage to optic nerves
Optic chiasma	Chiasmal compression: pituitary adenoma, craniopharyngioma, etc.
Occipital cortex	Tumours Degenerative conditions

Note: Unilateral causes (e.g. cataract, refractive errors, uveitis, glaucoma, progressive optic atrophy and tumours) can affect the second eye.

Typical symptoms:

- reading difficulty
- difficulty in recognising faces
- problems with driving, especially at night
- difficulty with television viewing
- reduced ability to see in bright light
- may see haloes around lights

The type of visual distortion seen by patients is illustrated in FIGURE 66.2 .

FIGURE 66.2 Blurred vision: appearance of a subject through the eyes of a person with cataracts

Photo courtesy Allergan Pharmaceuticals

Examination

- Reduced visual acuity (sometimes improved with pinhole)
- Diminished red reflex on ophthalmoscopy
- A change in the appearance of the lens

The red reflex and ophthalmoscopy

The ‘red reflex’ is a reflection of the fundus when the eye is viewed from a distance of about 60 cm (2 feet) with the ophthalmoscope using a zero lens. This reflex is easier to see if the pupil is dilated. Commencing with the plus 15 or 20 lens, reduce the power gradually and, at plus 12, lens opacities will be seen against the red reflex, which may be totally obscured by a very dense cataract. The setting up of the ophthalmoscope to examine intra-ocular structures is illustrated in FIGURE 66.3 .

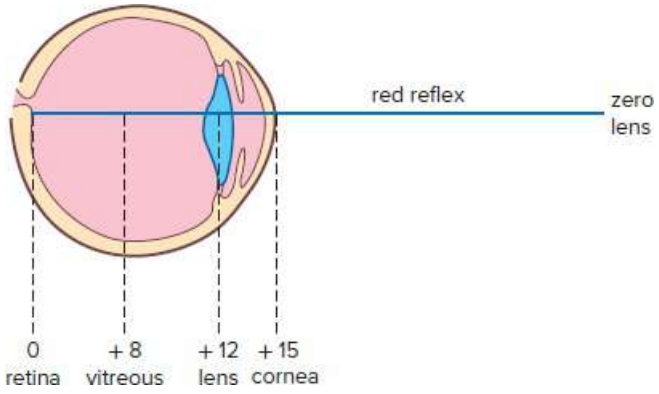

FIGURE 66.3 Settings of the ophthalmoscope used to examine intra-ocular structures

Management

Advise extraction of the cataract when the patient cannot cope. Contraindications for extraction include intra-ocular inflammation and severe diabetic retinopathy. There is no effective medical treatment for established cataracts. The removal of the cataractous lens requires optical correction to restore vision and this is usually performed with an intra-ocular lens implant. Full visual recovery may take 2–3 months. Complications are uncommon yet many patients may require YAG laser capsulotomy to clear any opacities that may develop behind the lens implant.

Postoperative advice to the patient

- Avoid bending over for a few weeks.
- Avoid strenuous exercise.
- The following drops may be prescribed:

  steroids (to reduce inflammation)

  antibiotics (to avoid infection)

  dilators (to prevent adhesions)

Prevention

Sunglasses, particularly those that wrap around and filter UV light, may offer protection against cataract formation.

Glaucoma

Glaucoma, which is caused by raised intra-ocular pressure, is categorised as open-angle or closed-angle and further as primary or secondary and acute or chronic. Open-angle glaucoma, also known as chronic simple glaucoma, is the commonest cause of irreversible blindness in middle age.[1] At a very late stage, it presents as difficulty in seeing because of loss of the outer fields of vision due to optic atrophy (see FIG. 66.4). Acute glaucoma, on the other hand, has a relatively rapid onset over a few days.

FIGURE 66.4 Typical visual field loss for chronic simple glaucoma; a similar pattern occurs with retinitis pigmentosa and functional visual loss (previously termed ‘hysteria’)

Clinical features (chronic glaucoma)

- Familial tendency
- No early signs or symptoms
- Central vision usually normal
- Insidious progressive restriction of visual field resulting in ‘tunnel vision’

Investigations

Tonometry

- Upper limit of normal is 22 mmHg

Ophthalmoscopy

- Optic disc cupping >30% of total disc area

Screening

- Adults 40 years and over: 2–5 yearly (at least 2 yearly over 60)
- Start about 30 years, then 2 yearly if family history

Management

- Treatment can prevent visual field loss
- Medication (for life) usually selected from:[7]

    timolol or betaxolol drops bd

    Note: These beta blockers can cause systemic complications, e.g. asthma

    latanoprost (or other prostaglandin analogue) drops, once daily

    pilocarpine drops qid

    dipivefrine drops bd

    brimonidine drops bd

    acetazolamide (oral diuretics)

- Surgery or laser therapy for failed medication

Retinitis pigmentosa

Primary degeneration of the retina is a hereditary condition characterised by a degeneration of rods and cones associated with displacement of melanin-containing cells from the pigment epithelium into the more superficial parts of the retina.

Typical features

- Begins as night blindness in childhood
- Visual fields become concentrically narrowed (periphery to centre), i.e. tunnel vision
- Blind by adolescence (sometimes up to middle age)
- Irreversible course—may be delayed by vitamin A[8]

Examination (ophthalmoscopic)

- Irregular patches of dark pigment, especially at periphery
- Optic atrophy → disc pallor

Intra-ocular foreign body

A small metal chip may penetrate the eye with minimal pain and the patient may not present with an ocular problem until the history of injury is long forgotten.

If infection does not supervene, presentation may be delayed for months or years until vision deteriorates due to metal degradation. The iris becomes rust-brown. It is important to X-ray the eye if it has been struck by a hammered fragment or if in any doubt at all about the mechanism of the injury.[1]

Chronic uveitis

Pain and redness may be minimal with this chronic inflammation, which often accompanies chronic systemic conditions (e.g. sarcoidosis). If untreated, visual loss often develops from secondary glaucoma and cataract. The pupil is bound to the lens by synechiae and is distorted. Treatment may involve long-term topical corticosteroids.

HIV infection

AIDS is associated with serious ocular complications, including Kaposi sarcoma of the conjunctivae, retinal haemorrhage and vasculitis.[3] Another problem is ocular cytomegalovirus infection, which presents as areas of opacification with haemorrhage and exudates.

Sudden loss of vision

This problem is alarming and distressing to the patient; considerable empathy is needed. Initial presentation may confuse with seemingly inappropriate behaviour; be very careful before diagnosing as psychogenic in origin.

A comparison of bilateral and unilateral causes of sudden loss of vision is presented in TABLE 66.4 , and the diagnostic strategy model in TABLE 66.5 . A simplified classification is:

unilateral:	retinal detachment
	retinal artery occlusion
	retinal vein thrombosis
	temporal arteritis

optic neuritis

migraine

bilateral: bilateral optic nerve lesion

functional (‘hysteria’, conversion reaction)

Table 66.4 Causes of sudden loss of vision[7]

	Bilateral	Unilateral	
		Transient	Permanent
Vascular causes	Occipital cortex ischaemia Pituitary apoplexy Homonymous hemianopia—vascular	Amaurosis fugax Transient ocular ischaemia Retinal emboli Malignant hypertension	Central retinal artery occlusion Central retinal vein occlusion Vitreous haemorrhage Ischaemic optic neuropathy
Other causes	Bilateral optic neuritis Toxic damage to optic nerve: • methanol • ethanol • tobacco • lead Leber optic atrophy Quinine poisoning of retina Cerebral oedema Occipital lobe trauma Craniopharyngioma Functional (‘hysterical’)	Acute angle closure glaucoma Uhthoff phenomenon Papilloedema Posterior vitreous detachment	Optic neuritis Retinal detachment Optic nerve compression Carcinomatous optic neuropathy Intra-ocular tumour

Table 66.5 Acute or subacute painless loss of vision:

Acute or subacute painless loss of vision: diagnostic strategy model

Probability diagnosis

Amaurosis fugax
Migraine
Retinal detachment
'Wet' macular degeneration

Serious disorders not to be missed

Cardiovascular:

- central retinal artery occlusion
- central retinal vein occlusion
- hypertension (complications)
- CVA

Neoplasia:

- intracranial tumour
- intra-ocular tumour:
  - primary melanoma
  - retinoblastoma
  - metastases

Vitreous haemorrhage
AIDS
Temporal arteritis
Acute glaucoma
Benign intracranial hypertension

Pitfalls (often missed)

Papilloedema
Optic neuritis
Intra-ocular foreign body
Posterior uveitis

Seven masquerades checklist

Diabetes (diabetic retinopathy)
Drugs (quinine)
Thyroid disorder (hyperthyroidism)

Is this patient trying to tell me something?

Consider 'hysterical' blindness, although it is uncommon.

A flow chart for the diagnosis of painless loss of vision is presented in FIGURE 66.5 .

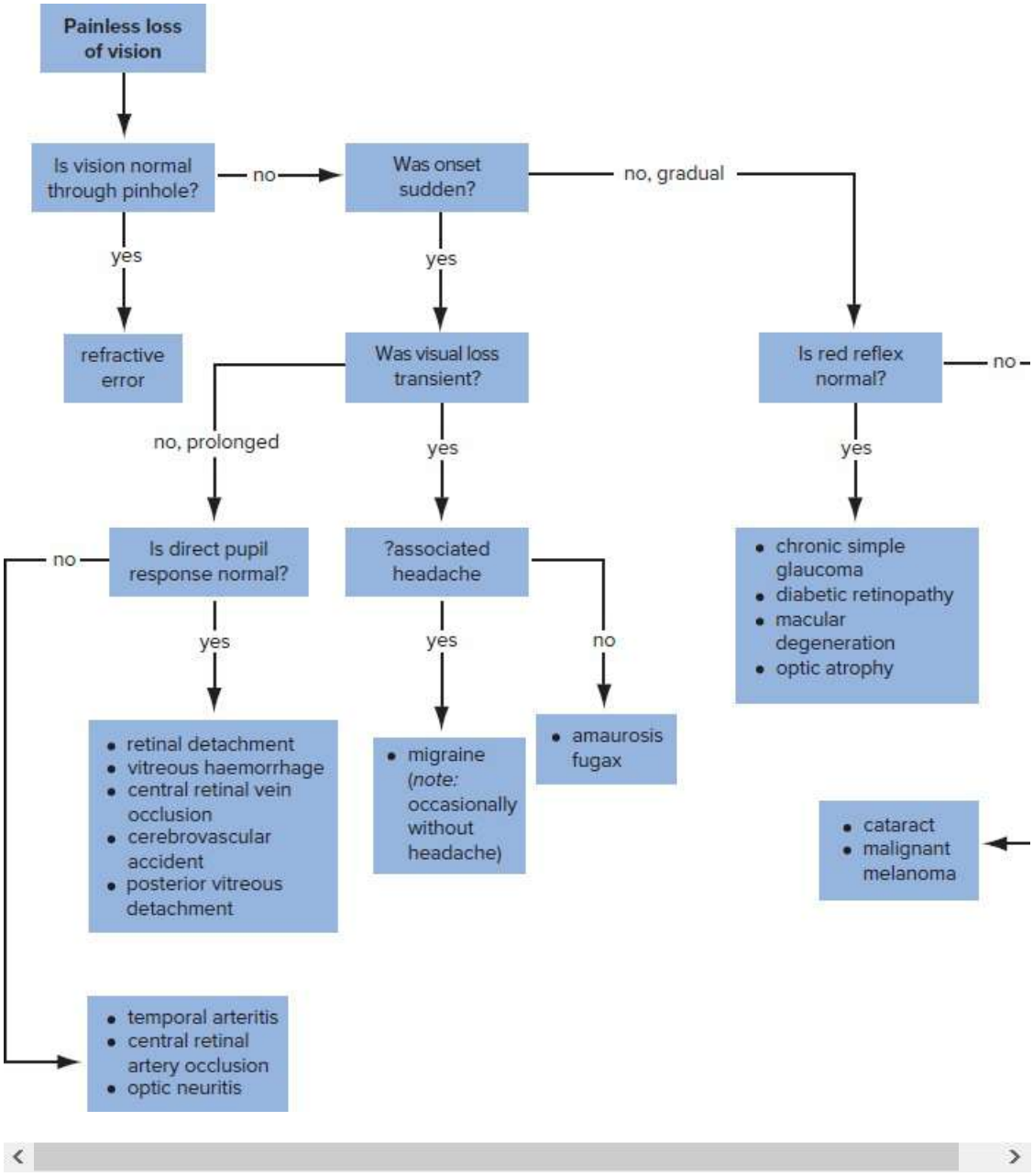

FIGURE 66.5 Diagnosis of painless loss of vision

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Amaurosis fugax

Amaurosis fugax is transient loss of vision (partial or complete) in one eye due to transient