

- neurological, especially cranial nerve function and muscle weakness disorders or evidence of stroke
- special oesophageal obstruction test:
 - hand the patient a glass of water and place a stethoscope over the left upper quadrant of abdomen
 - measure time between swallowing and murmur produced by bolus passing the cardia (normal: 7–10 seconds)
- assess aspiration risk, e.g. through having a sip of water¹

Investigations

- Full blood examination: ?anaemia
 - Neurological cause: oesophageal motility study (manometry)
 - Mechanical:
 - extrinsic compression (e.g. barium swallow, CT scan, chest X-ray)
 - intrinsic (e.g. endoscopy ± barium swallow)
- PET scan: good for identifying oesophageal cancer and gastro-oesophageal function

The primary investigation in suspected pharyngeal dysphagia is a video barium swallow,⁵ while endoscopy is generally the first investigation in cases of suspected oesophageal dysphagia. Barium swallow should precede endoscopy in the latter when there is a suspected oesophageal ‘ring’ and suspected oesophageal dysmotility. If endoscopy and radiology are negative, consider oesophageal motility studies to look specifically for achalasia or other less common motility disorders.

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Specific conditions

Benign peptic stricture

- Fibrous stricture of lower third oesophagus (can be higher)
- Follows years of reflux oesophagitis
- Usually older people
- Dysphagia with solid food

- Diagnosis confirmed by endoscopy and barium swallow

Treatment

- Dilate the stricture
- Treat reflux vigorously

⌚ Oesophageal cancer

- Dysphagia at beginning of meal
- Progressive dysphagia for solid food steadily progressive over weeks
- Can remain silent and tends to be invasive when diagnosed
- Hiccoughs may be an early sign
- Hoarseness and cough (upper third)
- Discomfort or pain—throat, retrosternal, interscapular
- Weight loss can be striking
- Associations: GORD, tobacco, Barrett oesophagus
- Diagnosis confirmed by barium swallow and endoscopy
- Both SCC upper third (commonest) and adenocarcinoma, distal third
- Adenocarcinoma associated with Barrett mucosa
- Plummer–Vinson syndrome
- Treatment is usually palliative surgery



DxT fatigue + progressive dysphagia + weight loss → oesophageal cancer

⌚ Barrett oesophagus

See [CHAPTER 36](#).

⌚ Achalasia⁶

- A disorder of oesophageal motility

- Widely dilated oesophagus
- Empties poorly through a smoothly tapered lower end
- Gradual onset of dysphagia for both liquids and solids
- Fluctuating symptoms—dysphagia, regurgitation
- Chest discomfort
- Diagnosis confirmed by barium swallow or manometry
- Manometry is the only way to diagnose with certainty¹

Treatment

- Conservative in the elderly (e.g. nifedipine/or endoscopic botulinum toxin injection into the sphincter)
- Pneumatic dilatation of lower oesophageal sphincter or surgical myotomy

Note: Prokinetic drugs have no place in treatment.

⌚ Drug-induced oesophageal injury³

- Tetracycline, especially doxycycline, can cause painful ulceration in all age groups.
- Delayed passage of some drugs (due to pre-existing disorders) can cause local ulceration, even perforation (especially in the elderly) (e.g. iron tablets, slow-release potassium, aspirin, NSAIDs, bisphosphonates, zidovudine, antibiotics).
- The elderly are prone to the problem if they ingest drugs upon retiring to bed with insufficient liquid washdown.

Management

- Stop drugs or swallow them upright with a glass of water
- Take antacids

⌚ Globus sensation (cricopharyngeal spasm)

Also referred to as ‘globus hysterius’ or ‘lump in the throat’, it is the subjective sensation of a lump in the throat. It appears to be associated with psychological stress (e.g. unresolved hurt, grief, non-achievement). Suppression of sadness is most often implicated.⁷ No specific aetiology or physiological mechanism has been established. The symptom can be associated with GORD, from frequent swallowing or an emotionally based dry throat.

Clinical features

- Sensation of being ‘choked up’ or ‘something stuck’ or lump—a very real sensation
- Not affected by swallowing
- Eating and drinking may provide relief
- Normal investigations

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Approach to patient

- Careful history and examination
- Exclude organic cause (refer TABLE 37.2)
- May require investigations if doubtful diagnosis

Management

- Usually settles with education, reassuring support and time (up to several months)
- Avoid swallowing very hot drinks
- No drug of proven value
- Treat any underlying psychological disorder

Odynophagia⁶

Pain on swallowing is basically caused by irritation of an inflamed or ulcerated (in particular) mucosa by the swallowed food bolus, usually meat, which may impact. Food bolus impaction may be very serious. If drinking water or, better still, 25–50 mL of a carbonated drink is ineffective, urgent upper GIT endoscopy may be required.

Important causes include:

- GORD (the commonest cause) with associated oesophagitis
- oesophageal spasm, especially distal oesophagus
- oesophageal candidiasis, especially in the immunosuppressed
- herpes simplex oesophagitis, in the immunosuppressed
- cytomegalovirus oesophagitis, in the immunosuppressed

- pill-induced oesophagitis/ulceration
- oesophageal cancer
- achalasia

Infective oesophagitis

- Common infective causes—candida species, herpes simplex virus (HSV), cytomegalovirus.
- These are more prone to occur in the immunocompromised.
- Present with odynophagia and/or dysphagia.
- Diagnosis is by upper GI endoscopy and biopsy.

Oesophageal candidiasis

- Treat with nystatin 100 000 units/mL suspension, 1 mL (o) 6 hrly for 10–14 weeks.
- If poor response, fluconazole (o), or IV if not tolerated orally.
- If no response to fluconazole, itraconazole (o). Refer to therapeutic guidelines.

Herpes simplex

- Treat with aciclovir IV until oral therapy possible, then famciclovir or valaciclovir.

Eosinophilic oesophagitis^{6,8,9}

Eosinophilic oesophagitis is increasingly being recognised as a cause of dysphagia, gastro-oesophageal reflux and acute food bolus obstruction in both children (particularly) and adults. It may present as infant colic.⁹ It should be considered in those who regularly experience food getting stuck in their throat. It is associated with allergic disorders such as hay fever, cow's milk allergy and asthma. The IgE is elevated. Refer to gastroscopy, which may show eosinophilic infiltrates in the oesophagus on mucosal biopsies. However, symptoms usually resolve within 72 hours of eliminating the offending food. A six-food elimination diet (cow's milk protein, wheat, soy, eggs, seafood and peanuts) has been shown to reduce symptoms in up to 90%.¹ Treatment of the acute attack includes IM buscopan and a swallowed topical corticosteroid aerosol, e.g. fluticasone twice daily for 8 weeks.⁶

Practice tips

-
- Although dysphagia is a common psychogenic symptom, it must always be taken seriously and investigated.

- Mechanical dysphagia represents cancer until proved otherwise.
- Progressive dysphagia and weight loss in an elderly patient is oesophageal cancer until proved otherwise.
- Oesophageal cancer usually causes pain, wasting and regurgitation.
- Globus sensation or hystericus, an anxiety disorder, should not be confused with dysphagia. It is the subjective sensation of a lump or mass in the throat. Particularly seen in young women.
- Cancer-induced achalasia occurs with tumours at the gastro-oesophageal junction usually due to adenocarcinoma of the stomach.
- Severe oesophageal reflux predisposes to adenocarcinoma.
- Oesophageal strictures can be benign, usually secondary to chronic reflux oesophagitis, or due to malignancy.
- Be careful of a change of symptoms in the presence of longstanding reflux. Consider stricture or cancer.
- A prominent hard lymph node in the left supraclavicular fossa (Troisier sign) is suggestive of cancer of the stomach.
- Dysphagia can be caused by a tight fundoplication and can be diagnosed by manometry or barium swallow.¹

Dietary adjustments (dysphagia diets)

1. Thin liquids, e.g. fruit juice, coffee, tea
2. Nectar-thick liquids, e.g. creamy soup, tomato juice
3. Honey-consistency diet (honey-thick liquids)
4. Pudding-thick foods, e.g. mashed bananas, cooked cereals, purees
5. Mechanical soft foods, e.g. meat loaf, baked beans, casseroles
6. Chewy foods, e.g. pizza, cheese, bagels
7. Foods that fall apart, e.g. bread, rice, muffins

Consider nutritional methods such as nasogastric feeding and gastrostomy.

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38 Dyspnoea

When man grows old ... there is much gas within his thorax, resulting in panting and troubled breathing.

HUANG TI (2697–2597 BCE), *THE YELLOW EMPEROR'S CLASSIC OF INTERNAL MEDICINE*

Dyspnoea is the subjective sensation of breathlessness that is excessive for any given level of physical activity. It is a cardinal symptom affecting the cardiopulmonary system and can be very difficult to evaluate. Appropriate breathlessness following activities such as running to catch a bus or climbing several flights of stairs is not abnormal but may be excessive due to obesity or lack of fitness.

Key facts and checkpoints

- Determination of the underlying cause of dyspnoea in a given patient is absolutely essential for effective management.
- The main causes of dyspnoea are lung disease, heart disease, obesity and functional hyperventilation.¹
- The most common cause of dyspnoea encountered in family practice is airflow obstruction, which is the basic abnormality seen in chronic asthma and chronic obstructive pulmonary disease (COPD).²
- Wheezing, which is a continuous musical or whistling noise, is an indication of airflow obstruction.
- Some patients with asthma do not wheeze and some patients who wheeze do not have asthma.
- Other important pulmonary causes include restrictive disease, such as fibrosis, collapse and pleural effusion.
- Dyspnoea is not inevitable in lung cancer but occurs in about 60% of cases.³

- Normal respiratory rate is 12–16 breaths/minute.

Terminology

It is important to emphasise that dyspnoea or breathlessness is a subjective sensation of the desire for increased respiratory effort and must be considered in relation to the patient's lifestyle and individual tolerance of discomfort. It also depends on the age, physical fitness and physical expectations of the person. Patients may complain of tightness in the chest and this must be differentiated from angina.

The American Thoracic Society guide for grades of dyspnoea is:

- Grade 0 No breathlessness except with strenuous exercise
- Grade 1 Breathlessness when hurrying on the level or walking up a slight hill
- Grade 2 Walks slower than people of the same age on the level because of breathlessness or has to stop for breath when walking at own pace on level
- Grade 3 Stops for breath after walking about 100 metres or a few minutes on the level
- Grade 4 Too breathless to leave the house or breathless when dressing or undressing

Glossary of terms

Hyperpnoea An increased level of ventilation (e.g. during exertion).

Hyperventilation Overbreathing.

Orthopnoea Breathlessness lying down flat.

Paroxysmal nocturnal dyspnoea Inappropriate breathlessness causing waking from sleep.

Tachypnoea An increased rate of breathing.

Difference between heart and lung causes

The distinguishing features between dyspnoea due to heart disease and to lung disease are presented in TABLE 38.1 .

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Table 38.1 Comparison of distinguishing features between dyspnoea due to heart disease and lung disease

Lung disease	Heart disease
History of respiratory disease	History of hypertension, cardiac ischaemia or valvular heart disease
Slow development	Rapid development
Present at rest	Mainly on exertion
Productive cough common	Cough uncommon and then 'dry'
Aggravated by respiratory infection	Usually unaffected by respiratory infection

Source: Adapted with permission from Stenton C. The MRC breathlessness scale. Occup Med (Lond). 2008; 58(3): 226–227.
doi:10.1093/occmed/kqm162

The history is a good indication and a useful guideline is that dyspnoea at rest is typical of lung disease, especially asthma, while it tends to be present on effort with heart disease as well as with COPD.

Wheezing

Wheezing is any continuous musical expiratory noise heard with the stethoscope or otherwise. Wheeze includes stridor, which is an inspiratory wheeze.

Common causes of wheezing

Localised:

- partial bronchial obstruction:
 - impacted foreign body
 - impacted mucus plugs
 - extrinsic compression
 - tracheomalacia

Generalised:

- asthma
- obstructive bronchitis
- bronchiolitis

'Cardiac asthma' and bronchial asthma

The term 'cardiac asthma' is (somewhat confusingly) used to describe a wheezing sensation such as that experienced with paroxysmal nocturnal dyspnoea. Differentiating features are presented in TABLE 38.2 .

Table 38.2 Comparison of distinguishing features between 'cardiac asthma' and bronchial asthma

	Cardiac	Bronchial
Dyspnoea	Mainly inspiratory	Mainly expiratory
Cough	Follows dyspnoea	Precedes dyspnoea
Sputum	Pink and frothy	Thick and gelatinous
Relief	Standing up (by an open window) Intravenous diuretic/CPAP, morphine	Coughing up sputum Bronchodilator
Lung signs	Mainly crackles	Mainly wheezes

Is it asthma or COPD?

This question is often asked, especially in the middle-aged or elderly person with dyspnoea. Differentiating features are presented in TABLE 38.3 . A mixed picture can occur, particularly as people with decades of asthma get older (esp. smokers).

Table 38.3 Comparison of asthma and COPD

	Asthma	COPD
Symptoms <35 years	Common	Unusual
Smoking history	Possible	Invariable
Chronic cough	Uncommon	Common
Dyspnoea	Diurnal and variable	Constant and progressive
Response to inhaled bronchodilator	Good	Poor
Nocturnal waking with symptoms	Common	Uncommon
Airflow obstruction	Reversible	Non-reversible

A diagnostic approach

A summary of the diagnostic strategy model is presented in [TABLE 38.4](#) .

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Table 38.4 Dyspnoea: diagnostic strategy model

Probability diagnosis

- Bronchial asthma
- Bronchiolitis (children)
- COPD
- Ageing; lack of fitness/physical deconditioning
- Left heart failure (increased left ventricular and diastolic pressure)
- Obesity

Serious disorders not to be missed

Cardiovascular:

- acute heart failure (e.g. AMI)
- arrhythmia
- pulmonary embolism
- fat embolism
- pulmonary hypertension
- dissecting aneurysm
- cardiomyopathy
- pericardial tamponade
- anaphylaxis

Neoplasia:

- bronchial carcinoma, other malignancy

Severe infections:

- coronavirus infection, e.g. SARS, COVID-19
- avian influenza
- pneumonia
- acute epiglottitis (children)

Respiratory disorders:

- inhaled foreign body
- upper airways obstruction
- pneumothorax

- atelectasis
- pleural effusion
- tuberculosis
- acute respiratory distress syndrome (ARDS)

Neuromuscular disease:

- infective polyneuritis
- poliomyelitis

Pitfalls (often missed)

Interstitial lung disease:

- idiopathic pulmonary fibrosis
- hypersensitivity pneumonitis
- sarcoidosis
- others

Chemical pneumonitis

Metabolic acidosis

Radiotherapy

Kidney failure (uraemia)

Multiple small pulmonary emboli

Seven masquerades checklist

Depression

Diabetes (incl. ketoacidosis)

Drugs

Anaemia

Thyroid disorder (thyrotoxicosis)

Spinal dysfunction (ankylosing spondylitis)

Is the patient trying to tell me something?

Consider functional hyperventilation (anxiety and panic attacks).

Probability diagnosis

The common causes of dyspnoea are lung disease, heart disease, obesity, anaemia (tissue hypoxia) and functional hyperventilation. More specifically, bronchial asthma, COPD, acute pulmonary infections and left heart failure (often insidious) are common individual causes.

Serious disorders not to be missed

Severe cardiovascular events such as acute heart failure, which may be precipitated by

myocardial infarction (may be silent, especially in diabetics), a life-threatening arrhythmia, pulmonary embolism, dissecting aneurysm or a cardiomyopathy (such as viral myocarditis) require early diagnosis and corrective action. Recurrent pulmonary embolism may present a diagnostic problem. There may be a history of deep venous thrombosis, pregnancy, malignancy or taking the contraceptive pill.⁴

Severe infections such as lobar pneumonia, tuberculosis and myocarditis must be considered. In children, acute epiglottitis, croup, bronchiolitis, pneumonia and bronchitis are serious infections responsible for respiratory distress.

Primary carcinoma is an important consideration, especially in dyspnoea of gradual onset. Other malignant conditions to consider are metastases, lymphangitis carcinomatosis, lymphomas and pleural mesothelioma. Pleural effusion may be the mode of presentation of some of these serious disorders.

Pitfalls

Interstitial pulmonary disease can be a diagnostic dilemma because the physical signs and X-ray appearances can be minimal in the early stages despite the presence of significant dyspnoea. Allergic alveolitis, such as that caused by birds (e.g. hypersensitivity to their droppings), can be a pitfall. The diagnosis is easier if a known disease associated with pulmonary infiltration, such as sarcoidosis, is present. Measuring the diffusing capacity will help with diagnosis.

Pericardial tamponade may cause difficulty in diagnosis either with an acute onset, such as malignancy involving pericardium, or insidiously. The patient usually has a weak pulse with pulsus paradoxus, hypotension and a raised jugular venous pressure.

It is important to be careful not to attribute dyspnoea simply to obesity or lack of fitness when it could have a true organic disorder such as heart failure.

Seven masquerades checklist

Most of the masquerades have to be considered as underlying causes. Depression can be associated with dyspnoea, anaemia is an important cause of dyspnoea, thyrotoxicosis can rarely present with dyspnoea and diabetic ketoacidosis can cause rapid deep breathing.

Drugs must also be considered, especially as a cause of interstitial pulmonary fibrosis that presents with dyspnoea, cough and fever. Drugs that cause this disorder include several cytotoxic agents (especially bleomycin, cyclophosphamide, methotrexate), amiodarone, sulfasalazine, penicillamine, nitrofurantoin, gold salts and adrenergic nasal sprays.³ Poisons that may cause hyperventilation are salicylate, methyl alcohol, theophylline overdosage and ethylene glycol. Anaemia must be considered, especially in those at risk. Dyspnoea is unlikely to be caused solely by chronic anaemia unless the haemoglobin level is less than 80 g/L.⁴ It is more likely to occur if another predisposing cause, such as ischaemic heart disease, is present.

Red flag pointers for dyspnoea

History	Examination
Increased age	Pallor/cyanosis
Sudden onset	Dyspnoea at rest
Ischaemic heart disease	Fever
TB risk	Hypotension
Recent travel	Tachycardia
Asthma/allergy	Tachypnoea
Unexplained weight loss	Calf tenderness
Haemoptysis	Chest wall signs
Significant trauma	Altered conscious state
HIV	Elevated JVP
Drugs: social, biologicals	Wheezing

Psychogenic considerations

Functional dyspnoea or hyperventilation is common. However, it is important to exclude organic causation such as asthma, drugs and thyrotoxicosis before settling with the psychogenic label and to reassure the patient strongly if there is no organic cause. Any uncomfortable sensation in the chest may be interpreted as dyspnoea by anxious people. Depression, anxiety and panic attacks may be underlying the problem. Characteristic associated features of hyperventilation with anxiety include dizziness, faintness, palpitations, yawning, paraesthesia of the hands and legs, inability to take a deep breath or a sensation of smothering. These patients may exhibit sighing and irregular breathing on examination. In true psychogenic dyspnoea, chest X-rays and pulmonary function tests are normal but symptoms are often reproduced after 15–30 seconds of voluntary hyperventilation. It is important to remember that it may be present in a patient who has organic disease of a mild degree such as asthma.

The clinical approach

History

Special attention should be paid to evaluating exactly what the patient means by breathlessness or restriction of breathing. The analysis should then include provoking factors and associated symptoms with a view to differentiating between pulmonary causes such as asthma and COPD. Wheeze is often (but not in all people, and not at all times) present in asthma and chronic airflow obstruction. Most respiratory causes of dyspnoea also produce cough. The rate of development of dyspnoea gives an indication of the possible cause (see TABLE 38.5).⁵ The sudden onset of dyspnoea at rest is suggestive of pulmonary embolism or pneumothorax. Severe dyspnoea developing over 1 or 2 hours is most likely due to left heart failure or bronchial asthma. Bronchial asthma is usually easily distinguished from left heart failure by the history of previous

attacks, by the absence of chest pain and the absence of cardiac murmurs. ‘My breathing feels tight’ indicates asthma. A complaint of ‘suffocation or feeling smothered’ or ‘just not getting enough air’ may be a pointer to functional dyspnoea.

Table 38.5 Typical causes of dyspnoea related to time of onset

Sudden

- Lung collapse
- Inhaled foreign body/other choking
- Spontaneous pneumothorax
- Arrhythmia
- Anaphylaxis
- Myocardial infarction
- Pulmonary embolism

Rapid (over a few hours)

- Asthma
- Hyperventilation (can be sudden)
- Acute exacerbations of COPD
- Pneumonia
- Diabetic ketoacidosis
- Extrinsic allergic alveolitis
- High altitude
- Left heart failure (acute pulmonary oedema)
- Pericardial tamponade
- Poisons

Over days or weeks

- Congestive heart failure
- Pleural effusion
- Carcinoma of the bronchus/trachea

Over months or years

- COPD
- Tuberculosis
- Fibrosing alveolitis
- Pneumoconiosis

Non-respiratory causes

- Anaemia

The dyspnoea of asthma tends to occur at rest and at night, while that with chronic airflow obstruction occurs with exertion.

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Examination

The routine findings from inspection, percussion and auscultation will determine whether the underlying lung disease is localised or generalised. The generalised findings for various disorders of the lungs are summarised in TABLE 38.6 .

Table 38.6 Comparison of examination findings for various lung disorders

	Trachea	Chest wall movement	Percussion note	Breath sounds	Vocal fremitus
Normal	Midline	Equal expansion	Resonant	Vesicular	Normal
Asthma	Midline	Decreased (bilateral)	Resonant	Vesicular — prolonged expiration	Normal or decreased
Emphysema	Midline	Decreased (bilateral)	Resonant to hyper-resonant	Vesicular — decreased	Decreased
Consolidation (e.g. lobar pneumonia)	Midline	Decreased on affected side	Dull	Bronchial	Increased
Collapse: major bronchus	Towards affected side	Decreased (unilateral)	Dull	Absent or decreased	Absent or decreased
Collapse: peripheral bronchus	Towards affected side	Decreased (unilateral)	Dull	Bronchial	Increased

Pleural effusion (>500 mL)	Towards opposite side (if massive)	Decreased (unilateral)	Stony dull	Absent or decreased	Absent or decreased
Pneumothorax (large)	Towards opposite side (if tension)	Decreased (unilateral)	Hyper-resonant	Absent or decreased	Absent or decreased
Fibrosis (generalised)	Midline	Decreased (bilateral)	Normal	Vesicular	Increased
Bronchiectasis	Midline	Slight decrease	Resonant to dull	Bronchial	Normal or decreased



Careful inspection is mandatory. The patient should be stripped to the waist and observed for factors such as cyanosis, clubbing, pallor, mental alertness, dyspnoea at rest, use of accessory muscles, rib retraction and any other abnormalities of the chest wall. A coarse tremor or flap of the outstretched hands indicates carbon dioxide intoxication.⁶ To obtain maximum value from auscultation, request the patient to open their mouth and take deep breaths. Adventitious sounds that are not audible during tidal breathing may then be heard. Wheezes are high-pitched continuous sounds heard either in expiration or inspiration, being more pronounced in expiration.

Crackles are short interrupted sounds heard mainly at the end of inspiration, resembling the crackling sound of hair being rubbed between the fingers near the ear. Fine crackles, previously referred to as crepitations, occur typically in lobar pneumonia and diffuse interstitial fibrosis, and are not cleared by coughing. Medium crackles are typical of congestive cardiac failure, while coarse crackles indicate airway mucus and usually clear on coughing.

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Causes of pulmonary crackles

- Left ventricular failure
- Idiopathic pulmonary fibrosis
- Extrinsic allergic alveolitis
- Pneumonia
- Bronchiectasis
- Chronic bronchitis

- Asbestosis
- Pulmonary fibrosis

Investigations

The two most important initial investigations for respiratory disease are chest X-ray and pulmonary function tests.

Pulmonary function tests (PFTs)

These relatively simple tests provide considerable information.

Peak expiratory flow rate

The most practical instrument for office use to detect chronic airway obstruction due to asthma or chronic bronchitis is the mini peak flow meter, which measures peak expiratory flow rate (PEFR). However, it gives considerably less information than spirometry.

The interpretation of the tests, which vary according to sex, age and height, requires charts of predicted normal values. A chart for PEFR in normal adult subjects is presented in Appendix V. The value for a particular patient should be the best of three results.

Spirometry

Spirometry is the gold standard test, and increasingly available in general practice. The measurement of the forced vital capacity (FVC) and the forced expiratory volume in one second (FEV₁) provide a very useful guide to the type of ventilatory deficit. Both the FVC and the FEV₁ are related to sex, age and height.

The FEV₁ expressed as a percentage of the FVC is an excellent measure of airflow limitation. In normal subjects it is approximately 70%. A normal spirometry pattern is shown in [FIGURE 38.1](#) and abnormal patterns in [FIGURE 38.2](#) . [FIGURE 38.3](#) summarises the relative values for these conditions.

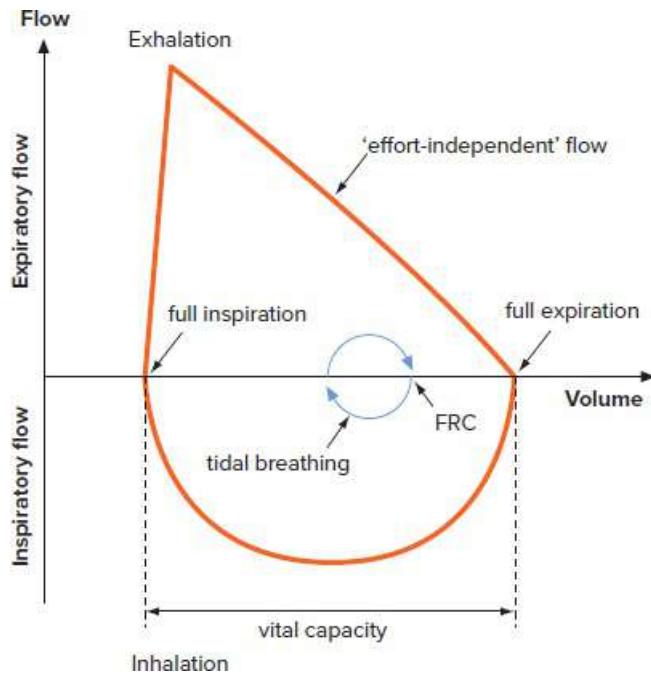


FIGURE 38.1 Spirometry patterns showing normal flow volume loop

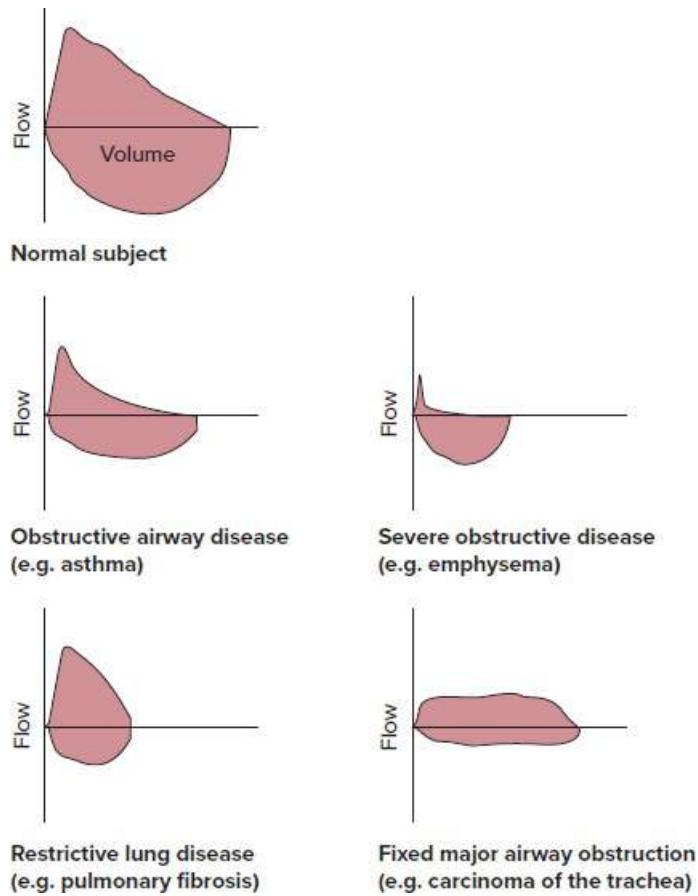


FIGURE 38.2 Maximum expiratory and inspiratory flow volume curves with examples of relative changes of patterns

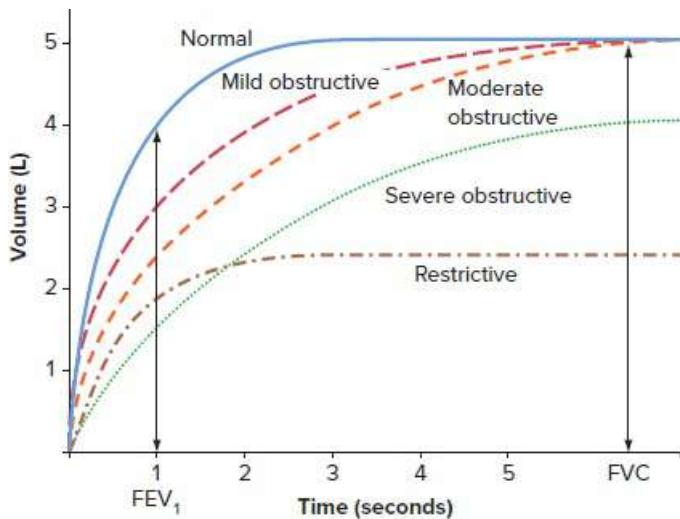


FIGURE 38.3 Spiromograms

Lung volume

Tidal volume (TV) and vital capacity (VC) can be measured by a simple spirometer but the total lung capacity and the residual volume are measured by the helium dilution method in a respiratory laboratory (rarely required).

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Diffusing capacity (gas transfer factor)

This test measures the carbon monoxide uptake by a single breath analysis for whole lungs. In normal lungs the transfer factor is a true measure of the diffusing capacity of the lungs for oxygen and depends on the thickness of the alveolar-capillary membrane.⁵ Gas transfer is usually reduced in patients with severe degrees of emphysema and fibrosis, anaemia and congestive cardiac failure. Useful for COPD and interstitial lung disease. Normal in asthma.

Pulse oximetry

An outstanding monitoring aid is transcutaneous pulse oximetry, which estimates oxygen saturation (SpO_2) of capillary blood. The estimates are generally very accurate and correlate to within 5% of measured arterial O_2 saturation (SaO_2).⁷ The ideal level is 97–100%; median levels —neonates 97%, children 98%, adults 98%. <92% is very serious.

Histamine challenge test

This test indicates the presence of airway or bronchial hyper-reactivity, which is a fundamental feature with asthma. The test should not be performed on those with poor lung function and only

performed by a respiratory technician under medical supervision. The test is potentially dangerous.

Other investigations (to select from)

- Haemoglobin, red cell indices and PCV
- White blood cell count (e.g. eosinophilia of asthma)
- Sputum, culture and cytology
- Tuberculosis tests
- ESR/CRP
- Arterial blood gas analysis
- Cardiological investigations:
 - ECG, including exercise
 - echocardiography (technically difficult in emphysema)
 - nuclear gated blood pool scan to assess heart function
 - cardiac enzymes
- Other medical imaging:
 - high-resolution CT (modality of choice for interstitial lung disease)
 - MRI
 - ventilation and perfusion radionuclide scan (pulmonary embolism)
- Bronchoscopy, especially fibre-optic bronchoscopy
- Thoracocentesis and pleural biopsy
- Open lung biopsy
- Alpha₁-antitrypsin measurement (normal range 1.1–2.2 g/L)

Pleural effusion

Key points

- Normal pleural space has 10–20 mL fluid

- Can be detected on X-ray if >300 mL fluid in pleural space
- Can be detected clinically if >500 mL fluid
- Can be subpulmonary—simulates a raised diaphragm
- May be asymptomatic
- Dyspnoea common with large effusion
- Chest pain in setting of pleuritis, infection or trauma
- Signs: refer [TABLE 38.6](#)
- The fluid may be transudate or exudate (diagnosed by aspirate)
- If bloodstained—malignancy, pulmonary infarction, TB

Transudate

Protein content <25 g/L; lactic dehydrogenase <200 IU/L.

Causes

- Heart failure (90% of cases)
- Hypoproteinaemia, e.g. nephrotic syndrome
- Liver failure with ascites
- Constrictive pericarditis
- Hypothyroidism
- Ovarian tumour—right-sided effusion (Meigs syndrome)

Exudate

Protein content >35 g/L; lactic dehydrogenase >200 IU/L

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Causes

- Infection—bacterial pneumonia, pleurisy, empyema, TB, viral
- Malignancy—bronchial carcinoma, mesothelioma, metastatic
- Pulmonary infarction

- Connective tissue diseases (e.g. SLE, RA)
- Acute pancreatitis
- Lymphoma
- Sarcoidosis
- HIV with parasitic pneumonia

Management

Aspiration if symptomatic: may require repeats and pleurodesis. Treat the underlying cause.

Dyspnoea in children

There are numerous causes of dyspnoea in children but the common causes are asthma, bronchiolitis and pulmonary infections. The important infections that can be fatal—croup, epiglottitis and myocarditis—must be kept in mind and intensively managed.

Bronchiolitis is an important cause of respiratory distress in infants under 6–12 months. It should not be confused with asthma (refer to CHAPTER 89) and does not respond to salbutamol or corticosteroids.

Sudden breathlessness or stridor may be due to an inhaled foreign body. Signs of lobar collapse may be present but physical examination may be of little help and a chest X-ray is essential.

Cardiovascular disorders, including congenital heart disease, can cause dyspnoea. Extra respiratory causes include anaemia, acidosis, aspiration, poisoning and hyperventilation.

Dyspnoea in the elderly

Dyspnoea in the elderly is common and is caused usually by heart failure and COPD. Other associations with ageing include lung cancer, pulmonary fibrosis and drugs. The classic problem of the aged is acute heart failure that develops typically in the early morning hours. The acute brain syndrome is a common presentation of all these disorders.

Respiratory disease in the elderly

The respiratory system, like most other bodily systems, matures until about the age of 25 years and subsequently slowly loses efficiency due to a variety of factors such as disease, smoking, pollution and ageing. There is a decline in lung function and gas exchange, and decreased ventilatory responses to hypoxia and hypercapnia.

Heart failure

Heart failure occurs when the heart is unable to maintain sufficient cardiac output to meet the demands of the body. Dyspnoea is a common early symptom as pulmonary congestion causes hypoxia (increased ventilation) and decreased compliance (increased work). The incidence of congestive cardiac failure (CCF) has been increasing steeply, partly due to the ageing population.

Symptoms

- Increasing dyspnoea progressing to (in order):

fatigue, especially exertional fatigue

paroxysmal nocturnal dyspnoea

weight change: gain or loss

It is convenient to divide heart failure into left and right heart failure but they rarely occur in isolation and often occur simultaneously. Right failure is invariably secondary to left failure. The distinction between systolic and diastolic dysfunction is increasingly being replaced by the related concept of heart failure with reduced or preserved ejection fraction (HFrEF and HFpEF). Both present in the same way clinically; diagnosis requires echocardiography and sometimes other tests such as BNP. This permits an accurate diagnosis and prognosis, and helps guide treatment.

Refer to Chronic heart failure ([CHAPTER 76](#)).

Chronic obstructive pulmonary disease

Chronic bronchitis and emphysema should be considered together as both these conditions usually coexist to some degree in each patient. An alternative, and preferable, term—chronic obstructive pulmonary disease (COPD)—is used to cover chronic bronchitis and emphysema with chronic airflow limitation.⁶

For more detail on the management of COPD refer to [CHAPTER 74](#).

Interstitial lung diseases (ILD)

Interstitial lung diseases comprise a group of disorders that have the common features of inflammation (pneumonitis) and fibrosis of the interalveolar septum, representing a non-specific reaction of the lung to injury of various causes.^{8,9}

In many there is a hypersensitivity reaction to various unusual antigens. The fibrosis may be localised as following unresolved pneumonia, bilateral as with tuberculosis or widespread.

Consider the possibility of fibrosis of the lungs in chronic dyspnoea and a dry cough

with normal resonance. If ILD is suspected, referral to a specialist physician for diagnosis is advisable.

Causes of widespread interstitial pulmonary fibrosis include:

- idiopathic pulmonary fibrosis
- hypersensitivity pneumonitis (extrinsic allergic alveolitis)
- drug-induced
- lymphangitis carcinomatosis
- various occupational lung disorders (pneumoconiosis)
- sarcoidosis
- acute pulmonary oedema
- immunological/multisystemic disease (e.g. connective tissue disorders, rheumatoid arthritis, vasculitis, inflammatory bowel disease)

Common clinical features:

- dyspnoea and dry cough (insidious onset)
- fine inspiratory crackles at lung base with faint breath sounds
- cyanosis and finger clubbing may be present
- PFTs:
 - restrictive ventilatory deficit
 - decrease in gas transfer factor
- characteristic X-ray changes

High-resolution CT scanning has been a major advance in diagnosis. May show 'honeycomb lung'.

§ **Idiopathic pulmonary fibrosis**

Idiopathic pulmonary fibrosis, also known as idiopathic fibrosing interstitial pneumonia and cryptogenic fibrosing alveolitis, is the most common diagnosis among those presenting with interstitial lung disease.

People usually present in the fifth to seventh decade with the clinical features as outlined under interstitial lung diseases, such as slowly progressive dyspnoea over months to years. Chest X-ray

abnormalities are variable but include bilateral diffuse nodular or reticulonodular shadowing favouring the lung bases. High-resolution CT scans are effective for diagnosis. Open lung biopsy may be needed for diagnosis and staging. The usual prognosis is poor, with death occurring about 3.5–5 years after diagnosis. The usual treatment is high doses of oral corticosteroids with azathioprine and no smoking.^{9,10}

If refractory, refer to a palliative care service.¹¹

Pulmonary sarcoidosis

Sarcoidosis is a multisystemic disorder of unknown aetiology, which is characterised by non-caseating granulomatous inflammation that involves the lung in about 90% of affected patients. A characteristic feature is bilateral hilar lymphadenopathy, which is often symptomless and detected on routine chest X-ray (CXR). Radiological lung involvement can be associated with or occur independently of hilar lymphadenopathy.

Clinical features^{8,9}

- May be asymptomatic (one-third)
- Onset usually third or fourth decade (but any age)
- Bilateral hilar lymphadenopathy (on CXR)
- Cough
- Fever, malaise, arthralgia
- Skin lesions: erythema nodosum, lupus pernio
- Ocular lesions (e.g. anterior uveitis)
- Other multiple organ lesions (uncommon)
- Overall mortality 2–5%

Erythema nodosum with an acute swinging fever, malaise and arthralgia in a young adult female is diagnostic of sarcoidosis.

Diagnosis

Histological evidence from biopsy specimen, usually transbronchial biopsy (essential if an alternative diagnosis, e.g. lymphoma, cannot be excluded) or skin biopsy in cases of erythema nodosum. A better modern diagnostic method is biopsy via video-assisted thoracoscopy.

Supporting evidence:

- elevated serum ACE (non-specific)
- PFTs: restrictive pattern; impaired gas transfer in advanced cases
- +tive Kveim test (not recommended these days)
- serum calcium

Treatment

Sarcoidosis may resolve spontaneously (hilar lymphadenopathy without lung involvement does not require treatment).

Indications for treatment with corticosteroids:

- no spontaneous improvement or worsening after 3–6 months
- symptomatic pulmonary lesions
- eye, CNS and other systems involvement
- hypercalcaemia, hypercalciuria
- erythema nodosum with arthralgia
- persistent cough

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Glossary of terms

Chronic airflow limitation A physiological process measured as impairment of forced expiratory flow, which is the major cause of dyspnoea in these patients.

Chronic bronchitis A clinical condition characterised by a productive cough on most days for at least 3 months of the year for at least 2 consecutive years in the absence of any other respiratory disease that could be responsible for such excessive sputum production (such as tuberculosis or bronchiectasis).

COPD A chronic, slowly progressive disorder characterised by the presence of airway obstruction, which may (or may not) be partially reversible by bronchodilator therapy.⁸

Emphysema This is defined in pathological rather than clinical terms as permanent dilatation and destruction of lung tissue distal to the terminal bronchioles.

Corticosteroid treatment⁹

- Prednisolone 0.5 mg/kg (up to 50 mg) (o) daily for 4–6 weeks, then reduce to lowest dose that maintains improvement.⁹ If there is a response, taper the dose to 10–15 mg (o) daily as a maintenance dose for 6–12 months.⁹
- Prednisolone 20–30 mg for 2 weeks for erythema nodosum of sarcoidosis.

Hypersensitivity pneumonitis

Hypersensitivity pneumonitis (extrinsic allergic alveolitis) is characterised by a widespread diffuse inflammatory reaction in both the small airways of the lung and the alveoli, due to the inhalation of allergens, which are usually spores of micro-organisms such as thermophilic actinomycetes in ‘farmer’s lung’ or (more commonly) avian protein from droppings or feathers in ‘bird fancier’s lung’. Occupational causes of extrinsic alveolitis have been described by Molina¹² (see TABLE 38.7).

Table 38.7 Various causes of hypersensitivity pneumonitis (extrinsic allergic alveolitis)

Occupation/disease	Source of antigen
Farmer’s lung	Mouldy hay, grain and straw
Bagassosis	Mouldy sugar cane fibre (bagasse)
Bird fancier’s lung	Avian proteins: dropping dust (e.g. from pigeons); ‘bloom’ on budgerigar feathers
Mushroom worker’s lung	Mushroom compost
Cheese washer’s lung	Moulds or mites on cheese
Wheat weevil lung	Infested wheat flour (insect)
Ventilator pneumonitis	Humidified hot air system Air-conditioning system
Wood pulp worker’s disorder	Contaminated wood dust
Detergent worker’s disorder	Proteolytic enzymes
Suberosis	Mouldy cork bark
Rat handler’s lung	Rat urine and serum
Malt worker’s lung	Mouldy barley
Coffee worker’s lung	Coffee dust

Sisal worker's lung	Sisal dust
Sericultural workers	Silkworms
Furrier's lung	Fur dust
Sausage workers	Dust
Prawn workers	Prawn fumes

Illness may present as acute or subacute episodes of pyrexia, chills and malaise with dyspnoea and a peripheral neutrophil several hours after exposure.¹² Management is based on prevention, namely avoiding exposure to allergens or wearing protective, fine-mesh masks. Prednisolone can be used (with caution) to control acute symptoms. Note that this allergic disorder is different from the infection psittacosis.

⌚ Drug-induced interstitial lung disease⁹

Drugs are an important cause of this disorder and have three main effects:

- 1. *Alveolitis with or without pulmonary fibrosis.* This is mainly due to cytotoxic drugs, nitrofurantoin and amiodarone. The drug should be removed and consideration given to prescribing prednisolone 50 mg (o) daily for several weeks, depending on response.
- 2. *Eosinophilic reactions.* This is presumably an immunological reaction, which may present as wheezing, dyspnoea, a maculopapular rash and pyrexia. The many implicated drugs include various antibiotics, NSAIDs, cytotoxic agents, major tranquillisers and antidepressants, and anti-epileptics. Treatment is drug removal and a short course of prednisolone 20–40 mg (o) daily for 2 weeks.
- 3. *Non-cardiogenic acute pulmonary oedema.* This is rare and has been reported to occur with opioids, aspirin, hydrochlorothiazide, β2-adrenoceptor agonists (given IV to suppress premature labour), cytotoxics, interleukin-2, heroin.

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Occupational pulmonary disease

Various types of acute and chronic pulmonary diseases are related to exposure to noxious substances such as dusts, gases and vapours in the workplace. Common chemical causes include formaldehyde used in processed woods, e.g. chipboard and medium-density fibre. GPs have a crucial role in the identification of the possible work-relatedness of lung disease.

Disorders due to chemical agents include:

- obstructive airways disorders, such as occupational asthma, acute bronchitis, (chronic) industrial bronchitis, byssinosis (asthma-like condition due to cotton dust)
- hypersensitivity pneumonitis

- pulmonary fibrosis (pneumoconiosis) due to mineral dust
- lung cancer due to industrial agents such as asbestos, various hydrocarbons
- pleural disorders, usually associated with asbestosis

Pneumoconiosis

The term ‘pneumoconiosis’ refers to the accumulation of dust in the lungs and the reaction of tissue to its presence, namely chronic fibrosis. The main cause worldwide is inhalation of coal dust, a specific severe variety being progressive massive fibrosis (complicated coal worker’s pneumoconiosis) in which the patient suffers severe dyspnoea of effort and a cough often productive of black sputum. TABLE 38.8 summarises the important causes.

Table 38.8 Selected pneumoconioses

Fibrotic lung disease	Agent	Typical occupations
Coal dust		
Coal worker’s pneumoconiosis	Coal dust	Coal mining
Metal dust		
Siderosis	Metallic iron or iron oxide	Mining Welding Foundry work
Inorganic dusts		
Silicosis	Silica (silicon dioxide)	Quarrying Demolition Rock mining Stone masons Sandblasting
Silicate dusts		
Asbestosis	Asbestos	Mining Shipbuilding Insulation Power stations Wharf labouring

Of particular concern are diseases caused by inhalation of fibres of asbestos, which is a mixture of silicates of iron, magnesium, cadmium, nickel and aluminium. These diseases include asbestosis, diffuse pleural thickening, pleural plaques, mesothelioma and increased bronchial carcinoma in smokers. Pulmonary asbestosis has classic X-ray changes but high-resolution CT scans may be required to confirm the presence of calcified pleural plaques. It usually takes 10–20 years from exposure for asbestosis to develop and 20–40 years for mesothelioma to develop,⁸ while bronchial carcinoma is caused by the synergistic effects of asbestosis and cigarette smoking.

Silicosis

Silicosis, caused by the inhalation of very fibrogenic silica particles, is a constant concern for workers. Mild cases cause no or minimal symptoms, but those affected experience progressive dyspnoea, intense dry cough and weakness.

Investigations include FBE, chest X-ray, CT scan, pulse oximetry and spirometry (restrictive ventilatory defect).

Management: avoid further exposure, wear special protective masks and check for associated tuberculosis.

Acute respiratory distress syndrome (ARDS)

ARDS, also known as acute lung injury and formerly called ‘adult respiratory distress syndrome’, refers to acute hypoxaemic respiratory failure following a pulmonary or systemic insult with no apparent cardiogenic cause of pulmonary oedema. This occurs about 12–48 hours after the event.¹³ The most common cause is sepsis, which accounts for about one-third of ARDS patients. The mortality rate is 30–40%, increasing if accompanied by sepsis. Management is based on early diagnosis, early referral, identification and treatment of the underlying condition and then optimal intensive care.¹⁴

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Clinical features

- Sudden onset of respiratory distress
- Stiff lungs—reduced lung compliance
- Refractory hypoxaemia
- Bilateral pulmonary infiltrates on X-ray
- No apparent evidence of congestive heart failure
- Absence of elevated left atrial pressure
- Specific gas exchange abnormalities

- Signs: tachypnoea, laboured breathing, rib retraction, central cyanosis, fine crackles on auscultation

The differential diagnoses are pneumonia and acute heart failure. Common risk factors/associations for ARDS include (indirectly—systemic)—sepsis, shock, trauma, burns, drug overdose (e.g. heroin), multiple transfusions, obstetric complications (e.g. eclampsia, amniotic fluid embolism), and many direct causes such as pulmonary aspiration, toxic gas inhalation, blast injury and pneumonia (e.g. COVID-19, SARS). Admit to an intensive care unit.

Coronavirus infection and COVID-19¹⁵

The disease caused by the SARS-CoV-2 coronavirus was declared by WHO to be a pandemic in March 2020 (see [CHAPTER 18](#)). It has since proven to be the most deadly respiratory pandemic in a century.¹⁶ Widespread vaccination commenced around a year after the virus was first identified. The mortality rate is around 1–2% (around 10 times the seasonal influenza rate), with most fatalities in those aged over 50 years; deaths in children are very rare.

Effective primary preventive measures at an individual level include: regular handwashing, social/physical distancing, avoiding large gatherings (particularly indoors), wearing face masks and coughing/sneezing etiquette. The majority of transmission is via asymptomatic (usually pre-symptomatic) individuals—hence the need for universal precautions (see [CHAPTER 18](#)).

Key features

- Most get mild symptoms similar to URTIs: mild fever, dry cough, sore throat, rhinorrhoea, malaise, headache, muscle pain.
- Diarrhoea and vomiting are common, and loss of taste/smell is notable.
- Dyspnoea (respiratory distress caused by pneumonia) increases with the severity of the illness, and is a cardinal feature in those requiring ICU admission.
- Extrapulmonary complications include septic shock, acute kidney injury (proteinuria is common), altered mental state and multiorgan failure.
- There is no effective, specific treatment for the virus; treatment is supportive.
- *Long COVID:* of those hospitalised, 70% report fatigue and half remain breathless 1–2 months post-discharge.¹⁷
- Dyspnoea is usually due to resolving infection and deconditioning—but bear in mind the increased risk of lung fibrosis, pulmonary embolus, myocarditis, heart failure and rhythm disturbance.¹⁸
- GPs should critically review ongoing symptoms, offer supportive treatment and investigate where necessary.

Practice tips

- Remember to order a chest X-ray and pulmonary function tests in all doubtful cases of dyspnoea.
- All heart diseases have dyspnoea as a common early symptom.
- Increasing dyspnoea on exertion may be the earliest symptom of incipient heart failure.
- Several drugs can produce a wide variety of respiratory disorders, particularly pulmonary fibrosis and pulmonary eosinophilia. Amiodarone and cytotoxic drugs, especially bleomycin, are the main causes.
- Dyspnoea in the presence of lung cancer may be caused by many factors, such as pleural effusion, lobar collapse, upper airway obstruction and lymphangitis carcinomatosis.
- The abrupt onset of severe dyspnoea suggests pneumothorax or pulmonary embolism.
- If a patient develops a relapse of dyspnoea while on digoxin therapy, consider the real possibility of digoxin toxicity and/or electrolyte abnormalities leading to left heart failure.
- Recurrent attacks of sudden dyspnoea, especially waking the patient at night, are suggestive of asthma or left heart failure.
- Causes of hyperventilation include drugs, asthma, thyrotoxicosis and panic attacks/anxiety.

Bronchial carcinoma

Dyspnoea is associated with about 60% of cases of lung cancer³ (see [CHAPTER 32](#)). It is not a common early symptom unless bronchial occlusion causes extrinsic collapse. In advanced cancer, whether primary or secondary, direct spread or metastases may cause dyspnoea. Other factors include pleural effusion, lobar collapse, metastatic infiltration, upper airway obstruction due to superior vena cava (SVC) obstruction and lymphangitis carcinomatosis. A special problem arises with coexisting chronic bronchitis and emphysema.

When to refer

- Patients with acute onset of severe dyspnoea
- All patients with heart failure resistant to initial therapy or where the diagnosis is in doubt
- Patients with pulmonary disease of uncertain aetiology, especially those requiring respiratory function tests
- Those in whom lung cancer is suspected

Patient education resources

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

- Acute respiratory distress syndrome
- Asthma
- Asthma in children
- Chronic obstructive pulmonary disease
- Asthma: dangerous asthma
- Heart failure
- Silicosis

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39 The painful ear

The ears should be kept perfectly clean; but it must never be done in company. It should never be done with a pin, and still less with the fingers, but always with an ear picker.

ST JEAN BAPTISTE DE LA SALLE (1651–1719)

Pain in the ear (otalgia) is a common symptom in general practice. It affects all ages, but is most prevalent in children, where otitis media is the commonest cause. Ear pain may be caused by disorders of the ear or may arise from other structures, and in many instances the precise diagnosis is difficult to make. Important causes of ear pain are summarised in TABLE 39.1 ¹.

Table 39.1 Causes of ear pain

1 Ear

External ear:

- Perichondritis
- Otitis externa:
 - *Candida albicans*
 - *Aspergillus nigra*
 - *Pseudomonas* spp.
 - *Staphylococcus aureus*
- Furunculosis
- Trauma
- Neoplasia
- Herpes zoster (Ramsay–Hunt syndrome)
- Viral myringitis
- Wax-impacted

Middle ear:

- Eustachian insufficiency
- Eustachian tube dysfunction

- Barotrauma
 - Acute otitis media
 - Chronic otitis media and cholesteatoma
 - Acute mastoiditis
-

2 Periotic cause

Dental disorders, e.g. dental abscess; malocclusion

Upper cervical spinal dysfunction

TMJ arthralgia

Parotitis

Temporal arteritis

Lymph node inflammation

Other referred causes

Pharyngeal disorders

Tonsillitis

Glossopharyngeal neuralgia

A patient with a painful ear often requests urgent attention, and calls in the middle of the night from anxious parents of a screaming child are commonplace. Infants may present with nothing except malaise, vomiting or screaming attacks.

Key facts and checkpoints

- Of patients presenting with earache, 77% can be expected to have acute otitis media and 12% otitis externa.
- Approximately 1 in every 25 patients in general practice will present with an earache.
- Two-thirds of children will sustain at least one episode of otitis media by their second birthday; 1 in 7 children will have had more than 6 episodes by this age. Peak prevalence is 9–15 months.²
- Otitis media is unlikely to be present if the tympanic membrane (TM) is mobile. Pneumatic otoscopy greatly assists diagnosis since the most valuable sign of otitis media is absent or diminished motility of the TM.
- Bullous myringitis, which causes haemorrhagic blistering of the eardrum or external ear canal, is an uncommon cause of severe pain. It is caused by a virus, probably influenza.³ Consider herpes zoster.

- The role of antibiotics (usually amoxicillin) is limited.
- Otitis externa can be distinguished from otitis media by pain on movement of the pinna.

A diagnostic approach

The five self-posed questions can be answered using the diagnostic strategy model (see [Page 469](#) [TABLE 39.2](#)).

Table 39.2 The painful ear: diagnostic strategy model

Probability diagnosis

Otitis media (viral or bacterial)
Otitis externa (fungal, viral or bacterial)
TMJ arthralgia
Eustachian tube dysfunction

Serious disorders not to be missed

Neoplasia of external ear
Cancer of other sites (e.g. tongue, nasopharynx)
Herpes zoster (Ramsay–Hunt syndrome)
Acute mastoiditis
Cholesteatoma
Necrotising otitis externa

Pitfalls (often missed)

Foreign bodies in ear
Hard ear wax
Barotrauma
Dental causes (e.g. abscess)
Referred pain: neck, throat
Unerupted wisdom tooth and other dental causes
TMJ arthralgia
Facial neuralgias, esp. glossopharyngeal
Chondrodermatitis nodularis helicis
Furuncles of canal or pinna
Post tonsillectomy:

- from the wound
- from TMJ due to mouth gag

Seven masquerades checklist

Depression

Spinal dysfunction (cervical)

Is the patient trying to tell me something?

Unlikely, but always possible with pain. More likely in children. Consider factitious pain.

Probability diagnosis

The commonest cause of ear pain is acute otitis media. Chronic otitis media (often painless) and otitis externa are also common. In the tropics, ‘tropical ear’ due to acute bacterial otitis is a particular problem. Temporomandibular joint (TMJ) arthralgia, which may be acute or chronic, is also common and must be considered, especially when otitis media and otitis externa are excluded.

Serious disorders not to be missed

As always, it is important not to overlook malignant diseases, especially the obscure ones, such as cancer of the tongue, palate or tonsils, which cause referred pain.

Locally destructive cholesteatoma associated with chronic otitis media must be searched for. It signifies the ‘unsafe’ ear (see FIG. 39.1) that must be distinguished from the so-called ‘safe’ ear (see FIG. 39.2).

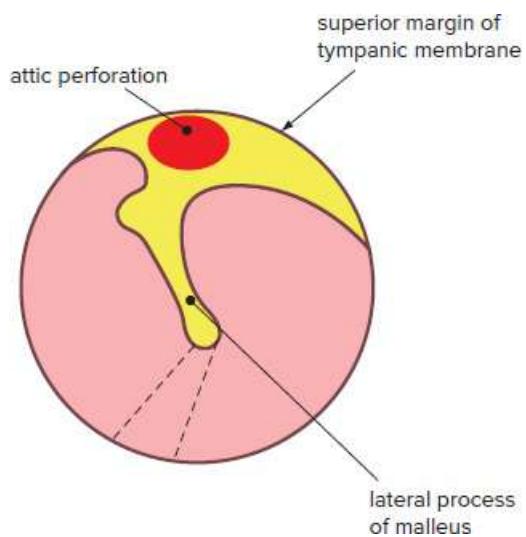


FIGURE 39.1 Infected ear: unsafe perforation

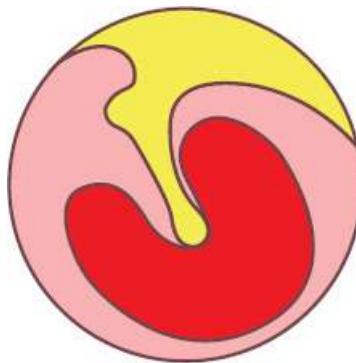


FIGURE 39.2 Infected ear: safe perforation

Herpes zoster should be considered; it is easily missed if it does not erupt on the pinna and is confined to the ear canal (usually the posterior wall), and especially in the older person.

Pitfalls

The medical aphorism ‘more things are missed by not looking than by not knowing’ applies particularly to the painful ear—good illumination and focusing of the auroscope are mandatory. Particular attention should be paid to the external canal—look for hard wax, otitis externa, furuncles and foreign objects such as insects.

It may not be possible to visualise the TMs so consider cleaning the canal to permit this (if possible, on the first visit), particularly if there are any atypical presenting features. Otitis media may coexist with otitis externa. Barotrauma should be considered, especially if pain follows air travel or diving.

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General pitfalls

- Failing to visualise the TM before diagnosis and treatment
- Not checking out possible referral sites such as the oropharynx and teeth
- Overlooking musculoskeletal causes such as TMJ arthralgia and cervical spondylosis
- Failing to recognise the unsafe ear

Red flag pointers for painful ear

- Offensive discharge >9 days

- Downward displacement of pinna
- Swelling behind ear
- Neurological symptoms (e.g. headaches, drowsiness)
- Older person: unexplained, intractable ear pain
- Persistent fever

Seven masquerades checklist

Of the conditions in the checklist, depression and dysfunction of the upper cervical spine have to be considered. Depressive illnesses should be considered in any patient complaining of chronic pain.

Disorders of the upper cervical spine are an occasional overlooked cause of periorbital pain. Pain from the C2 and C3 levels is referred to the posterior region of the ear.

Psychogenic considerations

Such factors are unlikely, although pain in the periorbital region can be magnified by a depressive state.

The clinical approach

History

In assessing the painful ear the relevant features are:

- site of pain and radiation
- details of the onset of pain
- nature of the pain
- aggravating or relieving factors, especially swimming
- associated features such as deafness, discharge, vertigo, tinnitus and irritation of the external ear, sore throat

Agonising pain may be caused by perichondritis or furunculosis of the external ear and by the rare problem of herpes zoster (Ramsay–Hunt syndrome).³ Movement of the pinna markedly increases the pain of acute otitis externa and perichondritis, and movement of the jaw usually causes an exacerbation of TMJ arthralgia or severe otitis externa.

Key questions (especially children)

- Where is the pain?
- Is it in the ear, behind or below it?
- Is it in one ear or both ears?
- Have you noticed any other symptoms such as sore throat, fever or vomiting?
- Has anyone hit you over the ear?
- Has there been a discharge from the ear?
- Have you noticed any deafness?
- Are you allergic to penicillin?
- Have you been swimming in a spa, and where?
- Have you been in an aeroplane?

Examination

The person's general state and behaviour is observed during the history taking. Sudden, jabbing pain may indicate neuralgia, particularly glossopharyngeal neuralgia or a severe infection. The external ear is carefully inspected and the pinna manipulated to determine any tenderness.

Palpate the face and neck and include the parotid glands, the regional lymph nodes, the mastoid process and the skin. Inspect the TMJs—tenderness from dysfunction typically lies immediately in front of the external auditory meatus. Palpate the TMJ over the lateral aspect at the joint disc. Ask the patient to open the mouth fully; tenderness is maximal. The TMJ can be palpated posteriorly by inserting the little finger into the external canal.

Inspect both ear canals and TMs with the auroscope, using the largest earpiece that comfortably fits into the canal. Better visualisation of the TM can be achieved by pulling the pinna back and downwards in young children and up and back in older children—see [FIGURE 39.3](#) for normal appearance. The diagnostic examination features of acute otitis media are illustrated in [FIGS. 39.8](#) and [39.9](#). Impacted wax does not often explain the otalgia. If herpes zoster involves the facial nerve, vesicles may be noted in and around the external auditory meatus (notably the posterior wall).

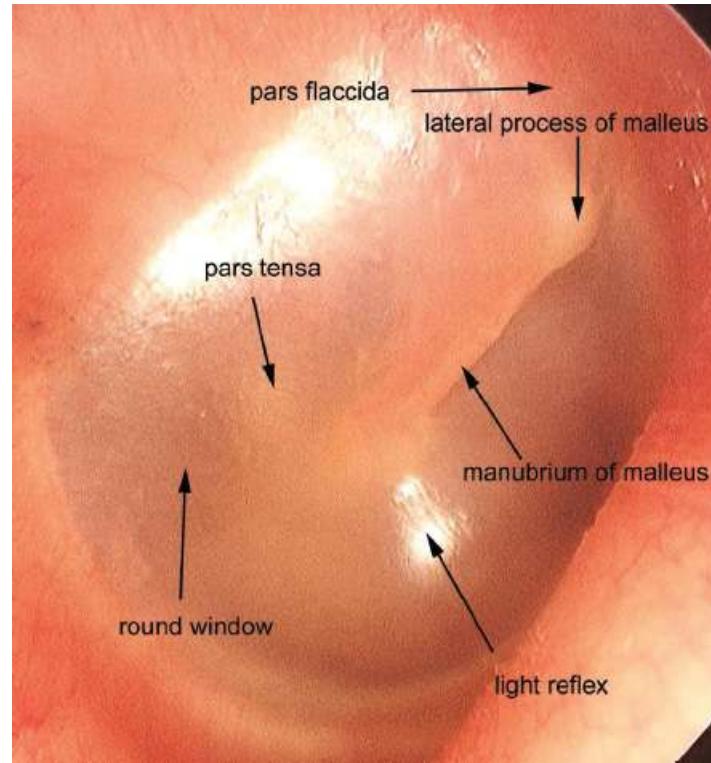


FIGURE 39.3 Normal right tympanic membrane

If the diagnosis is still doubtful, look for causes of referred pain; inspect the cervical spine, the nose and postnasal space and the mouth, including the teeth (percuss molars with a tongue depressor), pharynx and larynx.

Pharyngeal and mandibular causes of periorbital pain are summarised in [FIGURES 39.4](#) and [39.5](#).

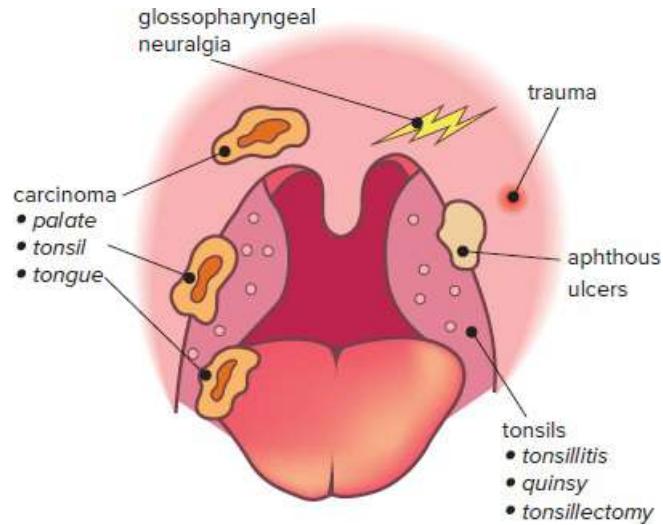


FIGURE 39.4 Pharyngeal causes of otalgia

Source: Courtesy of Bruce Black

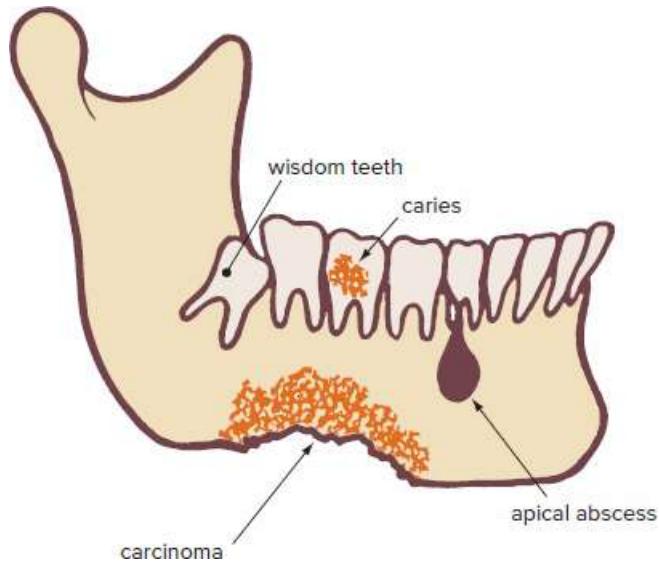


FIGURE 39.5 Mandibular causes of otalgia

Source: Courtesy of Bruce Black

Inspect sites supplied by the nerves V2, IX, X, XI, C1, C2 and C3 to exclude other causes of referred pain.

Investigations

Investigations are seldom necessary. Office-based hearing tests are useful, especially for children; use speech discrimination, hair rubbing and/or tuning fork tests. For potentially ongoing conditions such as chronic otitis media, refer for audiology. Audiology combined with tympanometry and physical measurement of the volume of the ear canal can be performed in children, irrespective of age.

Swabs from discharge, especially to determine bacterial causes, such as *Staphylococcus aureus* or *Pseudomonas* spp. infection, may be necessary. However, swabs are of no value if the TM is intact.

Radiology and CT/MRI scanning may be indicated for special conditions such as a suspected extraotic malignancy.

Ear pain in children

Important causes of primary otalgia in children include otitis media (particularly acute), otitis

externa, external canal furuncle or abscess, chronic eczema with fissuring of the auricle, impacted wax, foreign body, barotrauma, perichondritis, mastoiditis and bullous myringitis. Secondary otalgia includes pharyngeal lesions, dental problems, gingivostomatitis, mumps and postauricular lymphadenopathy. Peritonsillar abscess (quinsy) may cause ear pain.

Foreign bodies

Foreign bodies (FBs) are frequently inserted into the ear canal (see FIG. 39.6). They can usually be syringed out or lifted with thin forceps. Various improvised methods can be used to remove FBs in cooperative children. These include a probe to roll out the FB, a hooked needle or a rubber catheter used as a form of suction, or otherwise a fine sucker.⁴

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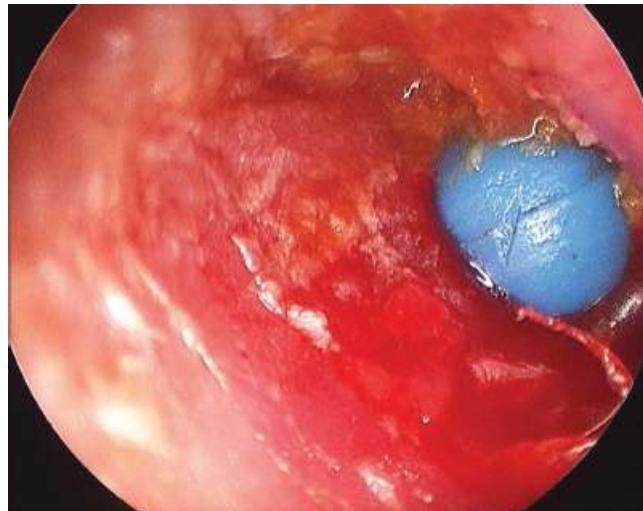


FIGURE 39.6 Foreign body (bead) in ear canal of a 3-year-old child, showing reactive tissue in ear canal

Probe method

This requires good vision using a head mirror or head light and a thin probe. The probe is inserted under and just beyond the FB. Lever it in such a way that the tip of the probe ‘rolls’ the foreign body out of the obstructed passage. With practice, this can be done with the probe inserted through the middle of an auriscope whose lens is slid half-way open.

Rubber catheter suction method

The only equipment required for this relatively simple and painless method is a straight rubber catheter (large type) and perhaps a suction pump. The end of the catheter is cut at right angles, a thin smear of petroleum jelly is applied to the rim and this end is applied to the FB. Suction is applied either orally or by a pump. Gentle pump suction is preferred but it is advisable to pinch closed the suction catheter until close to the FB as the hissing noise may frighten the child.

Insects in the ear

Live insects should be immobilised by first instilling drops of vinegar, methylated spirits or olive oil, and then syringing the ear with warm water.

Dead flies that have originally been attracted to pus are best removed by suction or gentle syringing.

Note: If simple methods such as syringing fail to dislodge the FB, it is important to refer for examination and removal under microscopic vision. Syringing should not be performed if there is a possibility of the FB perforating the TM.

Otitis media in children

Otitis media is very common in children and is the most common reason a child is brought in for medical attention. Persistent middle-ear effusions may follow and affect the language and cognitive development of young children. An abrupt onset is a feature.

Clinical features

- It is a clinical diagnosis
- Two peaks of incidence: 9–15 months of age, and school entry
- Seasonal incidence coincides with URTIs
- Bacteria cause two-thirds of cases⁵
- The commonest organisms are *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis*
- Fever, irritability, otalgia and otorrhoea may be present
- The main symptoms in older children are increasing earache and hearing loss
- Pulling at the ears is a common sign in infants
- Removal of wax may be necessary to visualise the TM (about 30% have occluded views), although with the decreasing role of antibiotics this visualisation becomes less crucial

Visualisation of the tympanic membrane

Use the largest ear speculum that will comfortably fit in the child's ear. A good technique to enable the examination of the ears (also nose and throat) in a reluctant child is where the child is held against the parent's chest while the parent's arm embraces the child's arm and trunk. Use of the pneumatic otoscope may reveal absent or limited movement.

Note the following features of the TM: translucency, colour, position and motility.

Treatment

Provide adequate pain relief with paracetamol or a NSAID. Short-term use of topical 2% lignocaine drops is effective for severe cases.

Many children with viral URTIs have mild reddening or dullness of the eardrum and antibiotics are not warranted, particularly in the absence of systemic features (fever and vomiting).⁵ Best practice in healthy, non-Indigenous children over 6 months of age is shared decision making with parents about symptomatic relief with analgesics and watchful waiting.⁶

The role of antibiotics in acute otitis media has diminished in response to emerging evidence from large double-blinded RCTs. The number needed to treat (NNT) with an antibiotic to prevent pain in one child at day 2–3 is 20, and no reduction in pain is found compared to placebo at day 1 or day 7.^{6,7}

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The antibiotic of choice for children who fit into those categories is a 5-day course of:⁵

amoxicillin 15 mg/kg 8 or 12 hourly up to 500 mg (o)

or

amoxicillin 30 mg/kg 12 hourly (o)

If β-lactamase-producing bacteria are suspected or documented, or initial treatment fails, use:

amoxicillin/clavulanate 22.5+3.2 mg/kg 12 hourly

Possible clinical indications for antibiotics in children with painful otitis media^{5,6}

Consider immediate and aggressive treatment:

- infants <6 months
- children <2 years with bilateral OM
- acute OM in the only hearing ear
- risk of complications in vulnerable groups, e.g. Aboriginal and Torres Strait Islander children, immunocompromised, cochlear implant (usually IV antibiotics)

Other considerations:

- neurological symptoms, e.g. facial palsy, vertigo

- sick child (fever and other systemic features)
- persistent fever and pain after 48–72 hours' conservative approach

For children with penicillin allergy, use:

trimethoprim/sulfamethoxazole 4+20 mg/kg 12 hourly

or

cefuroxime 15 mg/kg 12 hourly

With appropriate treatment most children with acute otitis media are significantly improved within 48 hours. Parents should be encouraged to contact their doctor if no improvement occurs within 72 hours. This problem is usually due to a resistant organism or suppuration. With a view to hospital admission, the patient should be re-evaluated at 10 days.⁸

Antibiotic drops

A randomised trial has found that antibiotic eardrops are superior to oral antibiotics for the treatment of acute otorrhoea in babies with grommets.⁹ This method has advocates among specialists who recommend ciprofloxacin drops following aural toilet.

Symptomatic treatment

Rest the child in a warm room with adequate humidity. Use analgesics such as paracetamol or ibuprofen in high dosage. Local anaesthetic ear drops are a reasonable option.

Follow-up: at review, check that the otitis media symptoms and signs have resolved, and perform an office-based hearing screen. If there are doubts about hearing, or a middle-ear effusion persists, refer for audiological screening.

Complications^{5,6}

- *Middle-ear effusion.* 70% of children will have an effusion present 2 weeks from the time of diagnosis, 40% at 4 weeks, with 10% having persistent effusions for 3 months or more. If the effusion is still present at 6–8 weeks, a course of antibiotics should be prescribed.² If the effusion persists beyond 3 months, refer for an ENT opinion or earlier if an associated speech delay or educational difficulty (especially a 20 dB hearing loss).
- *Acute mastoiditis.* This is a rare, major complication that presents with pain, swelling and tenderness developing behind the ear associated with a general deterioration in the condition of the child (see FIG. 39.7). Such a complication requires immediate referral.
- *Chronic suppurative otitis media.* Discharge through a perforation of the TM >6 weeks. Consider ciprofloxacin 0.3% ear drops, 5 drops 12 hourly until discharge free for at least 3

days.

- *Rare complications.* These include labyrinthitis, petrositis, facial paresis and intracranial abscess.

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Glue ear (serous otitis media)

This represents incomplete resolution of suppurative otitis media. Signs include loss of drum mobility, hearing loss and abnormal impedance confirmed by pneumatic otoscopy or tympanometry. Most resolve spontaneously but a Cochrane review⁷ found that resolution at 2–3 months was more likely if oral antibiotics were used (NNT=5, NNH [harms of side effects]=20). Consider recommending the safer and cheaper option of autoinflation, using Otovent-assisted nasal inflation.¹⁰ There is no evidence to support the use of nasal steroid sprays,¹¹ antihistamines or decongestants.¹²



FIGURE 39.7 Mastoiditis in a child with recurrent otitis media showing erythema and swelling behind the ear. Surgical drainage was performed.

If an effusion lasts for >3 months, arrange for a hearing assessment and consider referral to an ENT surgeon for possible tympanostomy tubes (grommets).

However, bear in mind that the evidence of benefit for grommets is also modest—a minor improvement in hearing (around 10 dB) at 3–6 months that subsequently disappears as natural resolution catches up. Grommets have not been demonstrated to benefit speech, language or behaviour.¹³

A positive outcome from arranging hearing assessment may be altering classroom seating position and the use of sound amplifiers; these are frequently used in remote area Aboriginal and Torres Strait Islander schools, where rates of glue ear are very high.

Recurrent acute bacterial otitis media

Antibiotic prevention of acute otitis media is indicated (arguably) if it occurs more often than every other month or for three or more episodes in 6 months or >4 in 12 months.¹⁴ Treat as for acute otitis media.

Consider:

chemoprophylaxis (for about 4 months)

amoxicillin twice daily (first choice)

or

cefaclor twice daily

Consider *Pneumococcus* vaccine in children over 18 months of age (if not already given) in combination with the antibiotic. Avoid smoke exposure (cigarettes and wood fires) and group child care.

Consider review by ENT consultant.

Viral infections

Most children with viral URTIs have mild–moderate reddening or dullness of the eardrum and antibiotics are not warranted. If painful bullous otitis media is present, either prick the bulla with a sterile needle for pain relief or instil dehydrating eardrops such as anhydrous glycerol.

Ear pain in adults and the elderly

Causes of otalgia that mainly afflict the elderly include herpes zoster (Ramsay–Hunt syndrome), TMJ arthralgia, temporal arteritis and neoplasia. It is especially important to search for evidence of malignancy.

Acute otitis media

Acute otitis media causes deep-seated ear pain, deafness and often systemic illness (see FIG. 39.8). The sequence of symptoms is a blocked ear feeling, pain and fever. Discharge may follow if the TM perforates, with relief of pain and fever.



FIGURE 39.8 Acute otitis media causing true otalgia. The ear drum bulges laterally due to pus in the middle ear. Perforation and otorrhoea imminent.

The commonest organisms are viruses (adenovirus and enterovirus), and the bacteria *H. influenzae*, *S. pneumoniae*, *Moraxella catarrhalis* and β-haemolytic streptococci.

The two cardinal features of diagnosis are inflammation and middle-ear effusion.

Appearance of the tympanic membrane (all ages)

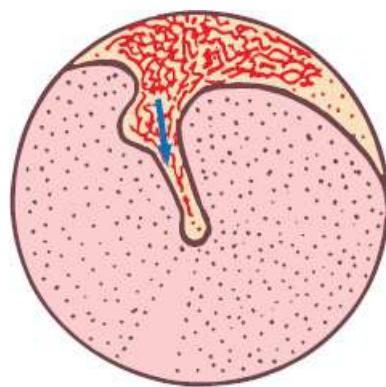
Translucency. If the middle-ear structures are clearly visible through the drum, otitis media is unlikely.

Colour. The normal TM is a shiny pale-grey to brown: a yellow colour is suggestive of an effusion.

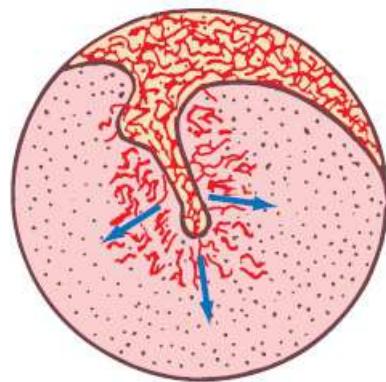
Diagnosis

The main diagnostic feature is the redness of the TM. The inflammatory process usually begins in the upper posterior quadrant and spreads peripherally and down the handle of the malleus (see FIG. 39.9). The TM will be seen to be reddened and inflamed with engorgement of the vessels, particularly along the handle of the malleus. The loss of light reflex follows and anatomical features then become difficult to recognise as the TM becomes oedematous. Bulging of the drum is a late sign. Blisters are occasionally seen on the TM and this is thought to be due to a viral

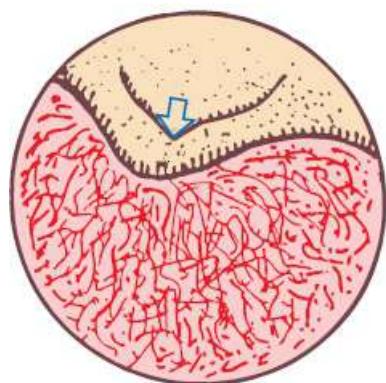
infection in the epidermal layers of the drum.



- erythema of prominent blood vessels progressing down handle of malleus
- normal drum



- progressive erythema
- loss of light reflex



- bulging pars flaccida
- red pars tensa
- anatomical structures unidentifiable

FIGURE 39.9 The appearances of the left tympanic membrane in the progressive development of acute otitis media

Treatment of acute otitis media (adults)

- Analgesics to relieve pain
- Adequate rest in a warm room
- Antibiotics for 5 days, repeated if necessary
- Treat associated conditions (e.g. adenoid hypertrophy)
- Follow-up: review and test hearing audiometrically

Antibiotic treatment⁵

First choice:

amoxicillin 750 mg (o) bd for 5 days⁵

or

500 mg (o) tds for 5 days

A longer course (up to 10 days) may be required depending on severity and response to 5-day course.

Alternatives:

doxycycline 100 mg (o) bd for 5–7 days (daily for milder infections)

or

cefaclor 250 mg (o) tds for 5–7 days

or

(if resistance to amoxicillin is suspected or proven) amoxicillin/potassium clavulanate 500/125 mg (o) tds for 5 days (the most effective antibiotic)

Consider surgical intervention for failed therapy.

Chronic otitis media

There are two types of chronic suppurative otitis media and they both present with deafness and

discharge without pain. The discharge occurs through a perforation in the TM: one is safe (see FIG. 39.10A), the other unsafe (see FIG. 39.10B).

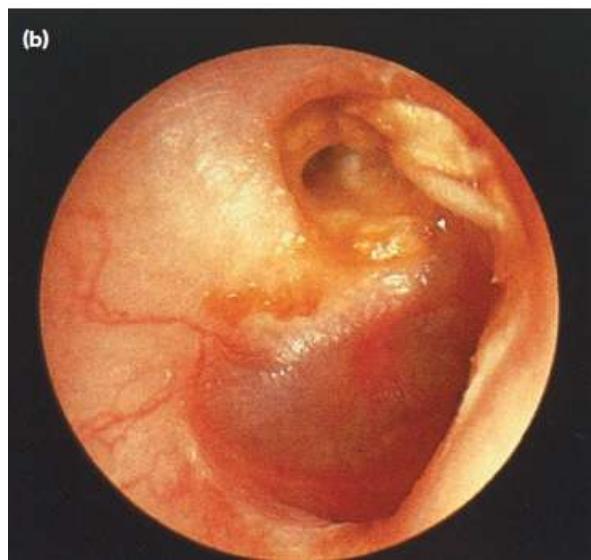


FIGURE 39.10 (a) Chronic otitis media with loss of the tympanic membrane; this is ‘safe ear’, (b) unsafe ear: chronic otitis media with attic cholesteatoma

⌚ Chronic discharging otitis media (safe)^{5,15}

If aural discharge persists for >6 weeks after a course of antibiotics, treatment can be with topical

steroid and antibiotic combination drops, following ear toilet. The toileting can be done at home by dry mopping with a rolled tissue spear. If persistent, referral to exclude cholesteatoma or chronic osteitis is advisable.

Recognising the unsafe ear

Examination of an infected ear should include inspection of the attic region, the small area of drum between the lateral process of the malleus, and the roof of the external auditory canal immediately above it. A perforation here renders the ear ‘unsafe’ (see FIG. 39.1); other perforations, not involving the drum margin (see FIG. 39.2), are regarded as ‘safe’.¹⁵

Cholesteatoma¹⁶

Refer CHAPTER 33 .

The status of a perforation depends on the presence of accumulated squamous epithelium (termed cholesteatoma) in the middle ear because this erodes bone. An attic perforation contains such material; safe perforations do not.

Red flags for cholesteatoma include meningitis-type features, cranial nerve deficits, sensorineural hearing loss and persistent deep ear pain.

Cholesteatoma is visible through the hole as white flakes, unless it is obscured by discharge or a persistent overlying scab (or wax). Either type of perforation can lead to chronic infective discharge, the nature of which varies with its origin. Mucus admixture is recognised by its stretch and recoil when this discharge is being cleaned from the external auditory canal. The types of discharge are compared in TABLE 39.3 .

Table 39.3 Comparison of types of discharge

	Unsafe	Safe
Source	Cholesteatoma	Mucosa
Odour	Foul	Inoffensive
Amount	Usually scant, never profuse	Can be profuse
Nature	Purulent	Mucopurulent

Table 39.4 Ear discharge: diagnostic strategy model

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Probability diagnosis

Acute otitis media with perforation

Chronic suppurative otitis media
Furuncle (boil) of ear canal
Infected otitis externa
Reactive skin conditions (e.g. eczema)
Liquefied wax

Serious disorders not to be missed

Infections:

- *Pseudomonas pyocyanea*
- cholesteatoma
- herpes zoster oticus
- mastoiditis

Cancer:

- malignancy with discharge (e.g. SCC)

Other:

- cerebrospinal fluid otorrhoea (fractured temporal bone)
- necrotising otitis media

Pitfalls (often missed)

Foreign body with infection/liquidisation (e.g. insects)

Trauma ± blood

Rarities:

- keratitis obliterans
- branchial or salivary fistula
- Wegener granulomatosis

Is the patient trying to tell me something?

Factitious

?Excessive manipulation of ear canal

Diagnostic tip

Acute ear discharge is most likely due to otitis externa or perforated ear drum with otitis media

Management

If an attic perforation is recognised or suspected, specialist referral is essential. Cholesteatoma cannot be eradicated by medical means: surgical removal is necessary to prevent a serious infratemporal or intracranial complication. Adjunct suction with care may be necessary to decompress the mass.

Diagnostic strategy for ear discharge (otorrhoea) mass

⌚ Otitis externa¹⁵

Otitis externa (see FIG. 39.11), also known as ‘swimmer’s ear’, ‘surfer’s ear’ and ‘tropical ear’, is common in a country whose climate and coastal living leads to extensive water sports. It is more prevalent in hot humid conditions and therefore in the tropics.

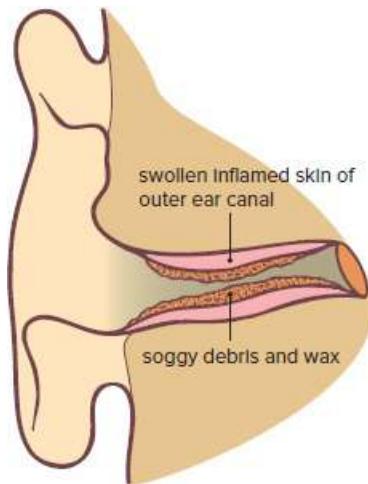


FIGURE 39.11 Otitis externa

Predisposing factors are allergic skin conditions, ear canal trauma, water penetration (swimming, humidity, showering), water and debris retention (wax, dermatitis, exostoses), foreign bodies, contamination from swimming water including spas, and use of Q tips and hearing aids.

Common responsible organisms

- Bacteria:

Pseudomonas sp.

Escherichia coli

S. aureus

Proteus sp.

Klebsiella sp.

- Fungi:

Candida albicans

Aspergillus sp.

Clinical features

- Itching at first
- Otalgia/pain (mild to intense) in 70%
- Fullness in ear canal
- Scant discharge
- Hearing loss

Signs

- Oedema (mild to extensive)
- Tenderness on moving auricle or jaw
- Erythema
- Discharge (offensive if coliform)
- Pale cream ‘wet blotting paper’ debris—*C. albicans* (see FIG. 39.12)
- Black spores of *Aspergillus nigra*
- TM granular or dull red



FIGURE 39.12 Acute otitis externa showing purulent discharge and narrowing of the ear canal

Obtain culture, especially if resistant *Pseudomonas* sp. suspected, by using small ear swab.

Note: ‘Malignant’ otitis externa occurs in diabetics due to *Pseudomonas* infection at base of skull.

Management

Aural toilet

Meticulous aural toilet by gentle suction and dry mopping with a wisp of cotton wool on a fine brooch under good lighting is the keystone of management. This enables topical medication to be applied directly to the skin.

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Syringing

This is appropriate in some cases but the canal must be dried meticulously afterwards. For most cases it is not recommended.

Topical antimicrobials for acute diffuse otitis externa^{5,17}

Most effective, especially when the canal is open, is an antibacterial, antifungal and corticosteroid preparation, e.g.:

- Kenacomb, Otodex or Sofradex drops (2–3 drops tds)

or

- Locacorten-Vioform drops (2–3 drops bd)

or

- Ciproxin HC (3 drops bd)

Use all for 7 days. Be cautious of ear drops with neomycin (hypersensitivity). The tragus should be pumped for 30 seconds after instillation by pressing on it repeatedly, within the limitation of any pain.

Other measures¹⁵

- Strong analgesics are essential
- Antibiotics have a minimal place in treatment unless a spreading cellulitis has developed (refer if in doubt)

- Prevent scratching and entry of water
- Use a wick soaked in combination steroid and antibiotic ointment for more severe cases
- Follow up ENT opinion for ‘red flags’

Dressings

Dressings are recommended in moderate and severe otitis media. After cleaning and drying, insert a cotton ear wick (an alternative is 10–20 cm of 4 mm Nufold gauze—see FIG. 39.13) impregnated with a steroid and antibiotic cream.

For severe oedematous otitis externa, a wick (e.g. Pope ear wick) is important and will reduce the oedema and pain in 12–24 hours (see FIG. 39.13). The wick can be soaked in an astringent (e.g. aluminium acetate 4% solution or glycerin and 10% ichthammol). The wick needs replacement daily until the swelling has subsided.

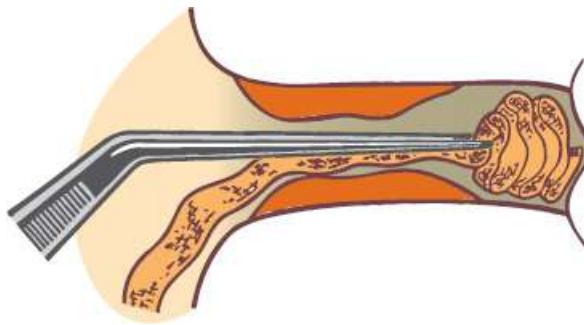


FIGURE 39.13 Insertion of a wick; it is packed gradually by short back-and-forth movements of the forceps

Source: Courtesy of Bruce Black

Practice tip for severe ‘tropical ear’

Prednisolone (o) 15 mg statim then 10 mg 8-hourly for six doses followed by:

- Merocel ear wick
- topical Kenacomb or Sofradex drops

Prevention

- Keep the ear dry, especially those involved in water sports
- Protect the ear with various waterproofing methods:

- cotton wool coated with petroleum jelly
 - an antiseptic drying agent (e.g. ethanol) after swimming and showering
 - tailor-made ear plugs (e.g. EAR foam plugs)
 - silicone putty or Blu-Tack
 - a bathing cap pulled well forward allows these plugs to stay *in situ*
- Avoid poking objects such as hairpins and cotton buds in the ear to clean the canal
 - If water enters, shake it out or use Aquaear drops (acetic acid/isopropyl alcohol), 4–5 drops to help dry the canal

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Necrotising otitis externa

This severe complication, usually due to *Pseudomonas aeruginosa*, can occur in the person who is elderly, immunocompromised or has diabetes. It involves cartilage and bone, and should be considered where there is treatment failure, severe persistent pain or fever and visible granulation tissue. Urgent referral is advisable.

Ear exostoses ('surfer's ear')

These periosteal bony overgrowths are usually caused by water retention in the ear. They are often multiple. They tend to trap keratin, wax and water, leading to infection.

Prevention

- Use plugs or Blu-Tack to waterproof ear.
- Dry thoroughly with hair dryer after swimming.

They may require surgical removal.

Acute localised otitis externa (furunculosis)

Furunculosis is a staphylococcal infection of the hair follicle in the outer cartilaginous part of the ear canal. It is usually intensely painful. Fever occurs only when the infection spreads in front of the ear as cellulitis. The pinna is tender on movement—a sign that is not a feature of acute otitis media. The furuncle (boil) may be seen in the external auditory meatus (see FIG. 39.14).

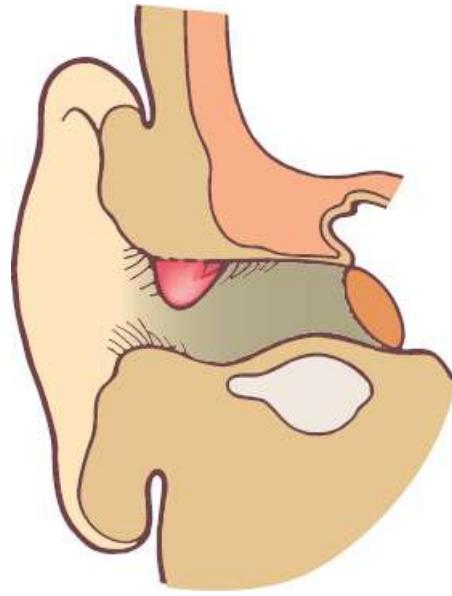


FIGURE 39.14 Furuncle (boil) in hair-bearing area at opening of the ear canal

Management

- If pointing, it can be incised after a local anaesthetic or freezing spray
- Warmth (e.g. use hot washcloth, hot-water bottle)
- If fever with cellulitis—flu/dicloxacillin or cephalexin

⌚ Perichondritis

Perichondritis is infection of the cartilage of the ear characterised by severe pain of the pinna, which is red, swollen and exquisitely tender. It is rare and follows trauma or surgery to the ear. As the organism is frequently *P. pyocyanus*, the appropriate antibiotics must be carefully chosen (e.g. ciprofloxacin).

⌚ Infected ear lobe

In a pierced ear, the cause is most likely a contact allergy to nickel in an earring, complicated by a *S. aureus* infection.

Management

- Discard the earrings
- Clean the site to eliminate residual traces of nickel

- Swab the site and then commence antibiotics (e.g. flucloxacillin or erythromycin)
- Instruct the person to clean the site daily, and then apply the appropriate ointment
- Use a ‘noble metal’ stud to keep the tract patent
- Advise the use of only gold, silver or platinum studs in future

Eustachian tube dysfunction

This is a common cause of discomfort.¹⁷ Symptoms include fullness in the ear, pain of various levels and impairment of hearing. The most common causes of dysfunction are disorders causing oedema of the tubal lining, such as viral URTI and allergy when the tube is only partially blocked; swallowing and yawning may elicit a crackling or popping sound. Examination reveals retraction of the TM and decreased mobility on pneumatic otoscopy. The problem is usually transient after a viral URTI.

Treatment

- Systemic and intranasal decongestants (e.g. pseudoephedrine or corticosteroids in allergic patients)
- Autoinflation by forced exhalation against closed nostrils (avoid in active intranasal infection)
- Avoid air travel, rapid altitude change and underwater diving

Otic barotrauma

Barotrauma is damage caused by undergoing rapid changes in atmospheric pressure in the presence of an occluded Eustachian tube (see FIG. 39.15). It affects scuba divers and aircraft travellers.

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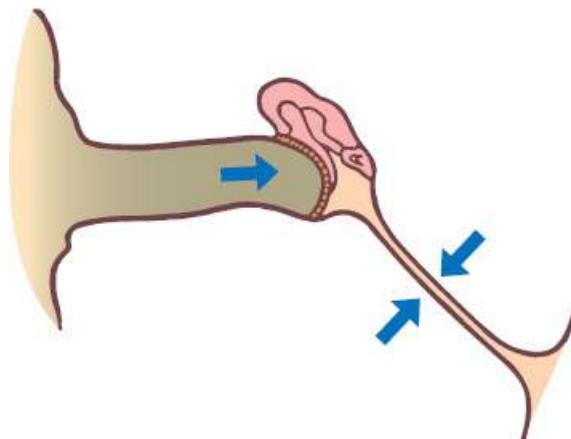


FIGURE 39.15 Mechanism of barotrauma, with blocking of the Eustachian

tube due to increased pressure at the sites indicated

Source: Courtesy of Bruce Black

The symptoms include temporary or persistent pain or pressure in both ears, deafness, vertigo, tinnitus and perhaps discharge.

Inspection of the TM may reveal (in order of seriousness): retraction; erythema; haemorrhage (due to extravasation of blood into the layers of the TM); fluid or blood in the middle ear; perforation. Perform conductive hearing loss tests with tuning fork.

Treatment

Most cases are mild and resolve spontaneously in a few days, so treat with analgesics and reassurance. Menthol inhalations are soothing and effective. Refer if any persistent problems for consideration of the Politzer bag inflation or myringotomy.

Prevention

Flying. Perform repeated Valsalva manoeuvres during descent. Use decongestant drops or sprays before boarding the aircraft, and then 2 hours before descent.

Diving. Those with nasal problems, otitis media or chronic tubal dysfunction should not dive.

Penetrating injury to tympanic membrane

A penetrating injury to the TM can occur in children and adults from various causes such as pencils and slivers of wood or glass. Bleeding invariably follows and infection is the danger.

Management

- Remove blood clot by suction toilet or gentle dry mopping
- Ensure no FB is present
- Check hearing
- Prescribe a course of broad-spectrum antibiotics (e.g. cotrimoxazole)
- Prescribe analgesics
- Instruct the person not to let water enter ear
- Review in 2 days and then regularly
- At review in 1 month, the drum should be virtually healed
- Check hearing 2 months after injury

Complete healing can be expected within 8 weeks in 90–95% of such cases.¹⁸

Temporomandibular joint arthralgia

If rheumatoid arthritis is excluded, a set of special exercises, which may include ‘chewing’ a piece of soft wood over the molars, invariably solves this problem (see CHAPTER 41). If an obvious dental malocclusion is present, referral is necessary.

When to refer

Otitis media

- Incomplete resolution of acute otitis media
- Persistent middle-ear effusion for 3 months after an attack of acute otitis media
- Persistent apparent or proved deafness
- Evidence or suspicion of acute mastoiditis or other severe complications
- Frequent recurrences (e.g. four attacks a year)
- Presence of craniofacial abnormalities

Other ear problems

- Attic perforation/cholesteatoma
- FBs in ear not removed by simple measures such as syringing
- No response to treatment after 2 weeks for otitis externa
- Suspicion of carcinoma of the ear canal
- Acute TM perforation that has not healed in 6 weeks
- Chronic TM perforation (involving lower two-thirds of TM)

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Practice tips

- The pain of acute otitis media may be masked by fever in babies and young children.
- A red TM is not always caused by otitis media. The blood vessels of the drum

head may be engorged from crying, sneezing or nose blowing. In crying babies, the TM as well as the face may be red.

- In otitis externa, most cases will resolve rapidly if the ear canal is expanded and then cleaned meticulously.
- If an adult presents with ear pain but normal auroscopy, examine possible referral sites, namely TMJ, mouth, throat, teeth and cervical spine.
- Consider mastoiditis if foul-smelling discharge is present over 7+ days.
- Antibiotics have no place in the treatment of otic barotrauma.
- It is good practice to make relief of distressing ear pain a priority. Adequate analgesics must be given. There is a tendency to give too low a dose of paracetamol in children. The installation of nasal drops in infants with a snuffy nose and acute otitis media can indirectly provide amazing pain relief.
- Spirit ear drops APF are a cheap and simple agent to use for recurrent otitis externa where wetness of the ear canal is a persistent problem.

Patient education resources

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

- Earache in children
- Ear infection (otitis media)
- Ear: otitis externa
- Ear: wax in your ear

Resources

National Health and Medical Research Council (NHMRC). Australian Clinical Practice Guidelines: Available from: <https://www.clinicalguidelines.gov.au>.

National Institute for Health and Care Excellence (NICE). Respiratory tract infection: Antibiotic prescribing. London: NICE, 2008. Available from:
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- 14 Buckley N (Chair). *Australian Medicines Handbook*. Adelaide: Australian Medicines Handbook Pty Ltd, 2013: 386–7.
- 15 Kaushik V, Malik T, Saeed SR. Intervention for acute otitis externa (Cochrane Review). *Cochrane Database of Syst Rev*, 2010; Issue 1: Art No. CD004740.
- 16 Black B. Otitis media: how to treat. *Australian Doctor*, 29 November 2002: I–VIII.

- 17** Wall GM et al. Ciprofloxacin 0.3% dexamethasone 0.1% sterile otic suspension for topical treatment ear infections: a review of the literature. *Pediatr Infect Dis J*, 2009; 28(2): 141–4.
- 18** Kruger R, Black B. Penetrating injury eardrum. *Aust Fam Physician*, 1986; 15: 735.

40 The red and tender eye

Those with sore eyes ... find the light painful, while the darkness, which permits them to see nothing, is restful and agreeable.

DIO CHRYSOSTOM (40–115 CE)

A red eye accounts for at least 80% of eye problems encountered in general practice.¹ An accurate history combined with a thorough examination will permit the diagnosis to be made in most cases without recourse to specialist ophthalmic equipment. A summary of the diagnostic strategy model is presented in TABLE 40.1 .

Table 40.1 The red and tender eye: diagnostic strategy model

Probability diagnosis

Conjunctivitis:

- bacterial
- adenovirus
- allergic

Irritants:

- stye
- pterygium/pinguecula

Serious disorders not to be missed

Acute glaucoma

Uveitis:

- acute iritis
- choroiditis

Corneal ulcer

Herpes simplex keratitis

Microbial keratitis (e.g. fungal, amoebic, bacterial)

Herpes zoster ophthalmicus
Penetrating injury
Endophthalmitis
Orbital cellulitis
Trachoma

Pitfalls (often missed)

Scleritis/episcleritis
Foreign body
Trauma—contusion, penetrating injury
Ultraviolet light ‘keratitis’
Blepharitis
Cavernous sinus arteriovenous fistula

Seven masquerades checklist

Drugs (hypersensitivity)
Thyroid disorder (hyperthyroidism)

Is the patient trying to tell me something?

Unlikely.

Key facts and checkpoints

- Acute conjunctivitis accounts for over 25% of all eye complaints seen in general practice.²
- Viral conjunctivitis (compared to bacterial) is more common in adults, is usually bilateral and discharge is more watery than purulent.³
- Viral conjunctivitis can be slow to resolve and may last for weeks.
- Pain and visual loss suggest a serious condition such as glaucoma, uveitis (including acute iritis) or corneal ulceration.
- Beware of the unilateral red eye—think beyond bacterial or allergic conjunctivitis. It is rarely conjunctivitis and may be a corneal ulcer, keratitis, foreign body, trauma, uveitis or acute glaucoma.⁴
- Keratitis (inflammation of the cornea) is one of the most common causes of an uncomfortable red eye. Apart from the well-known viral causes (herpes simplex, herpes zoster, adenovirus and measles), it can be caused by fungal infection (usually on a damaged cornea), bacterial infection, protozoal infection or inflammatory disorder such as ankylosing spondylitis.⁵
- Herpes simplex keratitis (dendritic ulcer) often presents painlessly, as the

neurotrophic effect grossly diminishes sensation.

The clinical approach

The five essentials of the history are:

- history of trauma (including wood/metalwork—foreign body)
- vision
- the degree and type of discomfort
- presence of discharge
- presence of photophobia

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The social and occupational history is also very important. This includes a history of exposure to a ‘red eye’ at school, work or home; incidents at work such as injury, welding, foreign bodies or chemicals; and genitourinary symptoms.

When examining the unilateral red eye, keep the following diagnoses in mind:

- trauma
- foreign body, including intra-ocular (IOFB)
- corneal ulcer
- iritis (uveitis)
- viral conjunctivitis (commonest type)
- acute glaucoma

The manner of onset of the irritation often gives an indication of the cause. Conjunctivitis or uveitis generally has a gradual onset of redness, while a small foreign body will produce a very rapid hyperaemia. Photophobia occurs usually with uveitis and keratitis. It is vital to elicit careful information about visual acuity. The wearing of contact lenses is important as these are prone to cause infection or the ‘overwear syndrome’, which resembles an acute ultraviolet (UV) burn.

The key eye symptoms

The key eye symptoms are:

- itch

- irritation
- pain (with pus or watering)
- loss of vision (red or white eye)

red = front of eye

white = back of eye

Key questions

- Have you noticed blurring of your vision?
- Have you been in close contact with others with the same condition?
- Have you had a cold or running nose recently?
- Do you wear contact lenses?
- Can you recall scratching or injuring your eye?
- What were you doing at the time you noticed trouble?
- Have you been putting any drops, ointments or cosmetics in or around your eye?
- Do you suffer from hay fever?
- Do you have any problems with your eyelids?
- Had your eyes been watering for some time beforehand?
- Have you had any other problems?
- Have you been exposed to arc welding?

Loss of vision in the red eye

Consider:

- iritis (uveitis)
- scleritis
- acute glaucoma (pain; nausea and vomiting)
- chemical burns

Red eye red flags (urgent ophthalmic referral)^{6,7}

- Severe ocular pain
- Severe orbital pain
- Reduction ophthalmic or loss of vision
- Diplopia
- Dilated pupil
- Abnormal corneal signs
- Globe displacement
- Endophthalmitis
- Microbial keratitis ± contact lens use

The painful red eye

Causes to consider:

- foreign body
- keratitis
- uveitis (iritis)
- episcleritis
- scleritis
- acute glaucoma
- hypopyon (pus in the anterior chamber)
- endophthalmitis (inflammation of internal structures—may follow surgery)
- corneal abrasion/ulceration

Pain with discharge:

- keratitis

Pain with photophobia:

- uveitis
- episcleritis

Examination

The basic equipment:

- eye testing charts at 45 cm (18 in) and 300 cm (10 ft)
- multiple pinholes
- torch (and Cobalt blue or ultraviolet light)
- magnifying aid (e.g. binocular loupe)
- glass rod or cotton bud to aid eyelid eversion
- fluorescein sterile paper strips
- anaesthetic drops
- ophthalmoscope
- Ishihara colour vision test
- tonometer (if available, e.g. Schiotz)

The four essentials of the examination are:

- testing and recording vision
- meticulous inspection under magnification
- testing the pupils
- testing ocular tension⁴

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Also:

- local anaesthetic test
- fluorescein staining
- subtarsal examination

Inspection

A thorough inspection is essential, noting the nature of the inflammatory injection, whether it is localised (episcleritis) or diffuse, viewing the iris for any irregularity, observing the cornea, and searching for foreign bodies, especially under the eyelids, and for any evidence of penetrating injury. No ocular examination is complete until the upper eyelid is everted and closely inspected. Both eyes must be examined since many patients presenting with conjunctivitis in one eye will have early signs of conjunctivitis in the other. Use fluorescein to help identify corneal ulceration. Local anaesthetic drops instilled prior to the examination of a painful lesion are recommended. The local anaesthetic test is a sensitive measure of a surface problem—if the pain is unrelieved a deeper problem must be suspected.

Palpate for enlarged pre-auricular lymph nodes, which are characteristic of viral conjunctivitis.

The nature of the injection is important. In conjunctivitis the vessels are clearly delineated and branch from the corners of the eye towards the cornea, since it involves mainly the tarsal plate. Episcleral and scleral vessels are larger than conjunctival vessels and are concentrated towards the cornea (see FIG. 40.1).

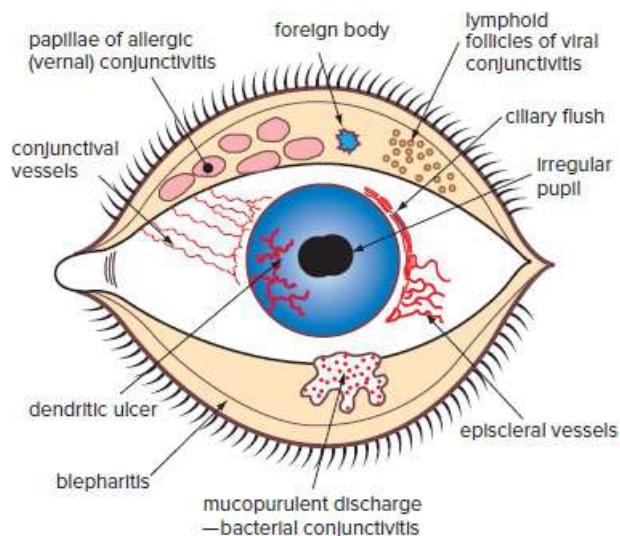


FIGURE 40.1 Physical signs to search for in a patient with a red eye (eyelids everted)

Ciliary injection appears as a red ring around the limbus of the cornea (the ciliary flush), and the individual vessels, which form a parallel arrangement, are not clearly visible. Ciliary injection may indicate a more serious deep-seated inflammatory condition such as anterior uveitis or a deep corneal infection. The presence of fine follicles on the tarsal conjunctivae indicates viral infection while a cobblestone appearance indicates allergic conjunctivitis.

Note: Slit lamp examination is ideal for the examination of the eye.

Investigations

These include:

- swab of discharge for micro and culture or for viral studies
- ESR/CRP
- imaging

Red eye in children

Children can suffer from the various types of conjunctivitis (commonly), uveitis and trauma. Of particular concern is orbital cellulitis, which may present as a unilateral swollen lid and can rapidly lead to blindness if untreated. Bacterial, viral and allergic conjunctivitis are common in all children. Conjunctivitis in infants is a serious disorder because of the immaturity of tissues and defence mechanisms. Serious corneal damage and blindness can result.

Neonatal conjunctivitis (ophthalmia neonatorum)

This is conjunctivitis in an infant less than 1 month old and is a notifiable disease. Chlamydial and gonococcal infections are uncommon but must be considered if a purulent discharge is found in the first few days of life.³ In both conditions the parents must be investigated for associated venereal infection and treated accordingly (this includes contact tracing) (see [CHAPTER 109](#)).

Chlamydia trachomatis accounts for 50% or more of cases. Its presentation in neonates is acute, usually 1–2 weeks after delivery, with moderate mucopurulent discharge. It is a systemic disease and may be associated with pneumonia. The diagnosis is confirmed by PCR tests on the conjunctival secretions.

Treatment is with azithromycin 20 mg/kg orally, daily for 3 days. Regular face washing and the treatment of all household contacts is recommended.³

Neisseria gonorrhoeae conjunctivitis, which usually occurs within 1–2 days of delivery, requires vigorous treatment with intravenous cephalosporins or penicillin and local sulfacetamide drops. The discharge is highly infectious and the organism has the potential for severe corneal infection with perforation and blindness⁷ or septicaemia.³

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Other common bacterial organisms can cause neonatal conjunctivitis, and herpes simplex virus type II can cause conjunctivitis and/or eyelid vesicles or keratitis.²

Trachoma

More than 6 million people worldwide have trachoma-caused blindness.

Trachoma is a chronic chlamydial conjunctivitis that is prevalent in the Aboriginal and Torres Strait Islander population, particularly in dry, remote regions. *C. trachomatis* is usually transmitted by human contact between children and parents and also by flies, especially where hygiene is inadequate. It is the most common cause of blindness in the world. Recurrent and untreated disease leads to lid scarring and inturned lashes (entropion) with corneal ulceration and

visual loss. It is important to commence control of the infection in childhood.

Treatment

- Prevention/community education
- Antibiotics—azithromycin
- Surgical correction (where relevant)

Blocked nasolacral duct

Delayed development of the nasolacral duct occurs in about 6% of infants,³ resulting in blocked lacrimal drainage; the lacrimal sac becomes infected, causing a persistent discharge from one or both eyes. In the majority of infants, spontaneous resolution of the problem occurs by the age of 6 months.

Management

- Bathing with normal saline
- Frequent massage over the lacrimal sac
- Referral for probing of the lacrimal passage before 6 months if the discharge is profuse and irritating or between 6 and 12 months if the problem has not self-corrected (refer [CHAPTER 84](#))
- Reserve topical antibiotics for true secondary infection (uncommon)

Red eye in the elderly

Elderly people have an increased risk of acute glaucoma, uveitis and herpes zoster. Acute angle closure glaucoma should be considered in anyone over the age of 50 presenting with an acutely painful red eye.

Eyelid conditions such as blepharitis, trichiasis, entropion and ectropion are more common in the elderly.

Acute conjunctivitis

Acute conjunctivitis is defined as an episode of conjunctival inflammation lasting less than 3 weeks.² The two major causes are infection (either bacterial or viral) and acute allergic or toxic reactions of the conjunctiva (see [TABLE 40.2](#)).

Major causes of a red eye

Table 40.2

	Site of inflammation	Pain	Discharge	Vision	Photo-phobia
Bacterial conjunctivitis	Conjunctiva, including lining of lids (usually bilateral)	Irritation—gritty	Purulent, lids stuck in the morning	Normal	No
Viral conjunctivitis	Conjunctiva, lining of lids often follicular (uni or bilateral)	Gritty	Watery	Normal	No
Allergic (vernal) conjunctivitis	Conjunctiva, papillary swellings on lid linings (bilateral)	Gritty—itching	Watery	Normal	No
Contact hypersensitivity (dermato-conjunctivitis)	Conjunctiva and eyelids Oedema	Itching	Watery	Normal—may be blurred	No
Subconjunctival haemorrhage	Beefy red area fading at edge (unilateral)	No	No	Normal	No
Herpes simplex keratitis	Unilateral—circumcorneal Dendritic ulcer	Yes—gritty	No, reflex lacrimation	Blurred, but variable, depends on site	Yes
Corneal ulcer	Unilateral—circumcorneal (exclude foreign body)	Yes	No, reflex lacrimation	Blurred, but variable, depends on site	Yes
Scleritis/episcleritis	Localised deep redness Tender area	Yes	No	Normal	Yes
Acute uveitis/iritis	Maximum around cornea	Yes—radiates to brow,	No, reflex lacrimation	Blurred	Yes

		temple, nose			
Acute glaucoma	Diffuse but maximum circumcorneal	Yes, severe with nausea and vomiting	No, reflex lacrimation	Haloes around lights	Yes



Practice tip

Be cautious of loss of vision, pain or photophobia—refer if appropriate.

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Clinical features

- Diffuse hyperaemia of tarsal or bulbar conjunctivae
- Absence of ocular pain, good vision, clear cornea
- Infectious conjunctivitis is usually bilateral (especially after the first day) with a discharge, and a gritty or sandy sensation

⌚ Bacterial conjunctivitis

Bacterial infection may be primary, secondary to a viral infection or secondary to blepharitis.

History

Purulent discharge with sticking together of eyelashes in the morning is typical. It usually starts in one eye and spreads to the other. There may be a history of contact with a person with similar symptoms. The organisms are usually picked up from contaminated fingers, face cloths or towels.

Clinical features

- Gritty red eye
- Purulent discharge, usually without lymphadenopathy
- Clear cornea

Examination

There is usually a bilateral mucopurulent discharge with uniform engorgement of all the conjunctival blood vessels and a non-specific papillary response (see FIG. 40.2). Fluorescein staining is negative.



FIGURE 40.2 Acute bacterial conjunctivitis with mucopurulent discharge, no corneal stain

Causative organisms

These include:

- *Streptococcus pneumoniae*
- *Haemophilus influenzae*
- *Staphylococcus aureus*
- *Streptococcus pyogenes*
- *N. gonorrhoeae* (a hyperacute onset)
- *Pseudomonas aeruginosa*

Diagnosis is usually clinical, but a swab should be taken for smear and culture with:²

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- hyperacute or severe purulent conjunctivitis
- prolonged infection
- neonates

Management³

Limit the spread by avoiding close contact with others, use of separate towels and good ocular hygiene. Clear away debris and mucus with saline solution before topical treatment. Exclude serious causes and a foreign body.

Mild cases

Mild cases may resolve with saline irrigation of the eyelids and conjunctiva but may last up to 14 days if untreated.⁸ An antiseptic eye drop such as propamidine isethionate 0.1% (Brolene) 1–2 drops, 6–8 hourly for 5–7 days can be used. Cooled black tea is reportedly widely used in Middle Eastern countries with good effect.

More severe cases

Chloramphenicol 0.5% eye drops, 1–2 drops 2 hourly for 24 hours,¹ decrease to 4 times a day for another 7 days (max. 10 days—cases of aplastic anaemia have been reported with long-term use).

Also use chloramphenicol 1% eye ointment each night or, alternatively, framycetin 0.5% eye drops, 1–2 drops every 1–2 hours for the first 24 hours, decreasing to 8 hourly until discharge resolves for up to 7 days.

Note: Never pad a discharging eye.

Practice tip

Brick-red eye—think of chlamydia.

Specific organisms

- *Pseudomonas* and other coliforms: use topical gentamicin and tobramycin. Chloromycetin ineffective.
- *N. gonorrhoeae*: use appropriate systemic antibiotics depending on sensitivity (use Gram stain culture and PCR). Use ceftriaxone or cefotaxime 1 g IM or IV as a single dose (adults).³
- *Chlamydia trachomatis*—may be sexually transmitted (a full STI screen is advisable). Differs from trachoma-causing strains. Shows a brick-red follicular conjunctivitis with a stringy mucus discharge. Treatment is with azithromycin.

⌚ Viral conjunctivitis

The most common cause of this very contagious condition is adenovirus.

History

It is commonly associated with URTIs and is the type of conjunctivitis that occurs in epidemics (pink eye).¹ The conjunctivitis usually has a 2–3 week course; it is initially one-sided but with cross-infection occurring days later in the other eye. It can be a severe problem with a very irritable, watering eye.

Examination

The examination should be conducted with gloves. It is usually bilateral with diffuse conjunctival infection and productive of a scant watery discharge. Viral infections typically but not always produce a follicular response in the conjunctivae (tiny, pale lymphoid follicles) and an associated pre-auricular lymph node (see FIG. 40.3). Subconjunctival haemorrhages may occur with adenovirus infection. High magnification, ideally a slit lamp, may be necessary to visualise some of the changes, such as small corneal opacities, follicles and keratitis.



FIGURE 40.3 Viral conjunctivitis: watery eye, lid swelling, typical eyelid follicles, associated local lymphadenopathy

Diagnosis is based on clinical grounds and a history of infected contacts. Viral culture and serology can be performed to identify epidemics.

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Treatment

- Limit cross-infection by appropriate rules of hygiene and patient education.
- Treatment is symptomatic—cool compress or saline bathing, possibly with topical lubricants (artificial tear preparations) or vasoconstrictors (e.g. phenylephrine).
- Do not pad; avoid bright light.
- Watch for secondary bacterial infection. Avoid corticosteroids, which reduce viral shedding and prolong the problem.

Primary herpes simplex infection

This viral infection produces follicular conjunctivitis. About 50% of patients have associated lid or corneal ulcers/vesicles, which are diagnostic.²

Dendritic ulceration highlighted by fluorescein staining is diagnostic (see FIG. 40.4). Antigen detection or culture may allow confirmation.

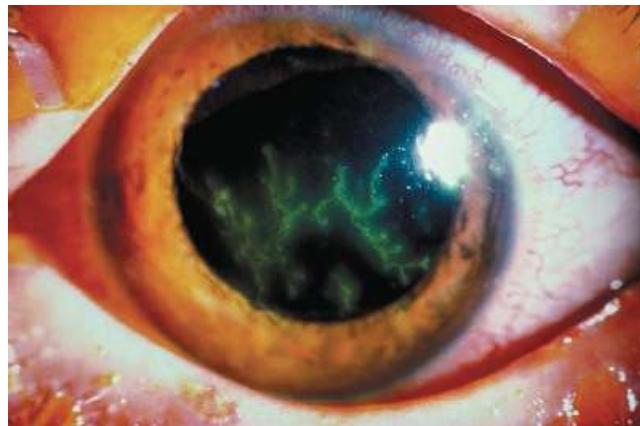


FIGURE 40.4 Herpes simplex keratitis—gritty, watery eye with typical dendritic ulcer, stained with fluorescein

Treatment (herpes simplex keratitis)

- Attend to eye hygiene
- Aciclovir 3% ointment, five times a day for 14 days or for at least 3 days after healing³
- Atropine 1% 1 drop, 12 hourly, for the duration of treatment will prevent reflex spasm of the pupil (specialist supervision)
- Debridement by an ophthalmologist

Never use corticosteroids, and refer all new cases early to an ophthalmologist.

⌚ Chlamydial conjunctivitis

Chlamydial conjunctivitis is encountered in three common situations:

- neonatal infection (first 1–2 weeks)
- young patient with associated venereal infection
- isolated Aboriginal and Torres Strait Islander people with trachoma

Take swabs for culture and PCR testing.

Systemic antibiotic treatment:⁸

- neonates: azithromycin 20 mg/kg orally, daily for 3 days³
- children over 6 kg and adults: azithromycin 1 g (o) as single dose

Note: Treat contacts in cases of STI.

Allergic conjunctivitis

Allergic conjunctivitis results from a local response to an allergen. It includes:

- vernal (hay fever) conjunctivitis
- contact hypersensitivity reactions, e.g. reaction to preservatives in drops

⌚ Vernal (hay fever) conjunctivitis

This is usually seasonal and related to pollen exposure, particularly in younger people. There is usually associated rhinitis (see [CHAPTER 72](#)).

Treatment³

Tailor treatment to the degree of symptoms. Artificial tear preparations may give adequate symptomatic relief. Oral antihistamines may be required but topical measures usually suffice.

Eye drop options:

1. Medications with both antihistamine and mast cell stabilising properties
 - ketotifen or olopatadine twice daily, or azelastine 2–4 times daily
2. Mast cell stabilisers
 - cromoglycate or lodoxamide 4 times daily
3. Antihistamines
 - levocabastine 2–3 times daily

I. Topical corticosteroids (severe cases, should refer)

Avoid vasoconstrictors (e.g. naphazoline, tetrahydrozoline).

⌚ Contact hypersensitivity

Common topical allergens and toxins include topical ophthalmic medications, especially

antibiotics, contact lens solutions (often the contained preservative) and a wide range of cosmetics, soaps, detergents and chemicals. Clinical features include burning, itching and watering with hyperaemia and oedema of the conjunctiva and eyelids. A skin reaction of the lids usually occurs.

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Treatment

- Withdraw the causative agent.
- Apply normal saline compresses.
- Treat with naphazoline or phenylephrine.
- If not responding, refer for possible corticosteroid therapy.

⌚ Subconjunctival haemorrhage

Subconjunctival haemorrhage, which appears spontaneously, is a beefy red localised haemorrhage with a definite posterior margin (see FIG. 40.5). If it follows trauma and extends backwards, it may indicate an orbital fracture. It is usually caused by a sudden increase in intrathoracic pressure such as coughing and sneezing. It is not related to hypertension but it is worthwhile measuring the blood pressure to help reassure the patient.



FIGURE 40.5 Subconjunctival haemorrhage: usually a localised haemorrhage that appears spontaneously. It is pain free. If traumatic and extends posteriorly, it may indicate an orbital fracture.

Management

No local therapy is necessary. The haemorrhage absorbs over 2 weeks. Patient explanation and reassurance is necessary ('a highly visible minor bruise'). If haemorrhages are recurrent, a bleeding tendency should be excluded.

Episcleritis and scleritis

Episcleritis and scleritis present as a localised area of inflammation (see FIGS 40.1 and 40.6). The episclera is a vascular layer that lies just beneath the conjunctiva and adjacent to the sclera. Both may become inflamed, but episcleritis (which is more localised) is essentially self-limiting, while scleritis (which is rare) is more serious as the eye may perforate.⁶ Both conditions may be confused with inflammation associated with a foreign body, pterygium or pinguecula. There are no significant associations with episcleritis, which is usually idiopathic, but scleritis may be associated with connective tissue disease, especially rheumatoid arthritis and herpes zoster and rarely sarcoidosis and tuberculosis.

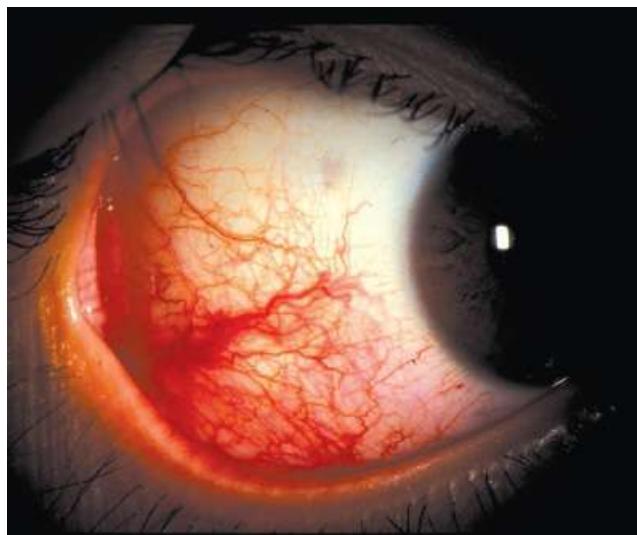


FIGURE 40.6 Episcleritis showing inflammation of the conjunctival and episcleral tissue only. Note the absence of violaceous colour that is seen in scleritis.

Clinical features

Episcleritis:

- no discharge
- no watering
- vision normal (usually)
- often sectorial
- usually self-limiting

Treat with topical or oral steroids.

Scleritis:

- painful loss of vision
- urgent referral

History

A red and sore eye is the presenting complaint. There is usually no discharge but there may be reflex lacrimation. Scleritis is much more painful than episcleritis⁶ and the eye becomes intensely red.

Examination

With scleritis, there is a localised area of inflammation that is tender to touch (FIG. 40.6), and which is more extensive than with episcleritis, being uniform across the eye. The inflamed vessels are larger than the conjunctival vessels. Application of topical phenylephrine 2.5% produces blanching of superficial episcleral vessels but no blanching of deep scleral vessels.

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Management

An underlying cause such as an autoimmune condition should be identified. Refer the patient, especially for scleritis. Corticosteroids or NSAIDs may be prescribed.

⌚ Uveitis (iritis)

The iris, ciliary body and the choroid form the uveal tract, which is the vascular coat of the eyeball.³

Anterior uveitis (acute iritis or iridocyclitis) is inflammation of the iris and ciliary body and this is usually referred to as acute iritis (see FIG. 40.7). The iris is sticky and sticks to the lens. The pupil may become small because of adhesions, and the vision is blurred.

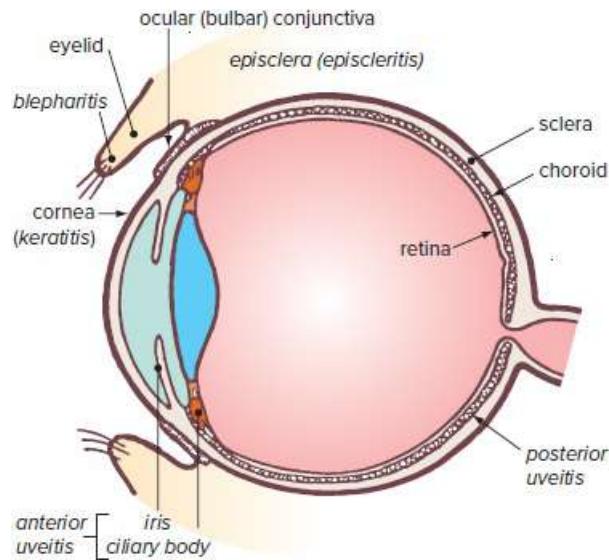


FIGURE 40.7 Diagrammatic representation of eye structures involved in inflammatory disorders

Causes include autoimmune-related diseases such as the seronegative arthropathies (e.g. ankylosing spondylitis), SLE, IBD, sarcoidosis and some infections (e.g. toxoplasmosis and syphilis).

Clinical features

- Eye redness, esp. around the edge of the iris
- Eye discomfort or pain
- Increased tearing
- Blurred vision
- Sensitivity to light
- Floaters in the field of vision
- Small pupil

The examination findings are summarised in TABLE 40.2 . The affected eye is red, with the infection being particularly pronounced over the area covering the inflamed ciliary body (ciliary flush). However, the whole bulbar conjunctivae can be infected. The patient should be referred to a consultant. Slit lamp examination aids diagnosis.

Management involves finding the underlying cause. Treatment includes pupil dilatation with atropine drops and topical steroids to suppress inflammation. Systemic corticosteroids may be

necessary. The prognosis of anterior uveitis is good if treatment and follow-up are maintained, but recurrence is likely.

Posterior uveitis (choroiditis) may involve the retina and vitreous membrane. Blurred vision and floating opacities in the visual field may be the only symptoms. Pain is not a feature. Referral to detect the causation and for treatment is essential.

Acute glaucoma

Acute glaucoma should always be considered in a patient over 50 years presenting with an acutely painful red eye. Permanent damage will result from misdiagnosis. The attack characteristically strikes in the evening or early morning when the pupil becomes semidilated.⁶

Clinical features

- Patient >50 years
- Pain in one eye
- ± Nausea and vomiting
- Impaired vision
- Haloes around lights
- Hazy cornea
- Fixed semidilated pupil
- Eye feels hard

Management

Urgent ophthalmic referral is essential since emergency treatment is necessary to preserve the eyesight. If immediate specialist attention is unavailable, treatment can be initiated with acetazolamide (Diamox) 500 mg IV and pilocarpine 4% drops to constrict the pupil or pressure-lowering drops.

Keratoconjunctivitis sicca

Dry eyes are a common problem, especially in elderly women. Lack of lacrimal secretion can be functional (e.g. ageing), or due to systemic disease (e.g. rheumatoid arthritis, SLE, Sjögren syndrome), drugs (e.g. β-blockers) or other factors, including menopause. Up to 50% of patients with severe dry eye have Sjögren syndrome.

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Clinical features

- A variety of symptoms
- Dryness, grittiness, stinging and redness
- Sensation of foreign body (e.g. sand)
- Photophobia if severe
- Slit light examination diagnostic with special stains

Treatment

- Treat the cause.
- Bathe eyes with clean water.
- Use artificial tears: hypromellose (e.g. Tears Naturale), polyvinyl alcohol (e.g. Tears Plus).
- Be cautious of adverse topical reactions.
- Refer severe cases.

Eyelid and lacrimal disorders

There are several inflammatory disorders of the eyelid and lacrimal system that present as a ‘red and tender’ eye without involving the conjunctiva. Any suspicious lesion should be referred.

⌚ Stye (external hordeolum)

A stye is an acute abscess of a lash follicle or associated glands of the anterior lid margin, caused usually by *S. aureus*. The patient complains of a red tender swelling of the lid margin, usually on the medial side (see FIG. 40.8). A stye may be confused with a chalazion, orbital cellulitis or dacryocystitis.



FIGURE 40.8 Hordeolum: a stye. This is a focal staphylococcal infection of the root of an eyelash.

Management

- Use heat to help it ‘point’ and discharge by using direct steam from a thermos (see FIG. 40.9) onto the enclosed eye or by hot-water compresses.
- If fluctuant and pointing, perform lash epilation to allow drainage of pus (incise with a size 11 blade if epilation does not work). Squeezing is discouraged.
- Do not use antibiotics (topical or oral) unless secondary spread (cellulitis).⁶



FIGURE 40.9 Steaming the painful eye: allow steam to rise from a thermos onto the closed eye for 10–15 minutes

⌚ Chalazion (meibomian cyst)

Also known as an internal hordeolum, this granuloma of the meibomian gland in the eyelid may become inflamed and present as a tender irritating lump in the lid. Look for evidence of blepharitis. Differential diagnoses include sebaceous gland carcinoma and basal cell carcinoma.

Management

Conservative treatment may result in resolution. This involves heat either as steam from a thermos or by applying a hot compress (a hand towel soaked in hot water) followed by light massage. If the chalazion is very large, persistent or uncomfortable, or is affecting vision, it can be incised and curetted under local anaesthesia. This is best performed through the inner conjunctival surface using a chalazion clamp (blepharostat) (see FIG. 40.10).

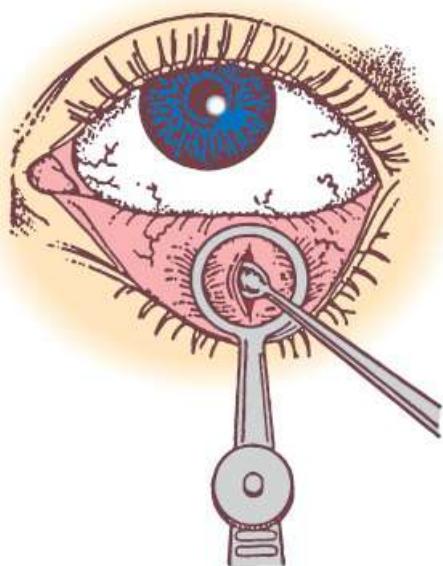


FIGURE 40.10 Excision of a meibomian cyst, using a chalazion clamp and curette

Meibomianitis is usually a staphylococcal micro-abscess of the gland, and oral antistaphylococcal antibiotics (not topical) are recommended (e.g. di/flucloxacillin 500 mg (o) 6 hourly for adults). Surgical incision and curettage may also be necessary.

฿ Blepharitis

This common chronic condition of the eyelids may involve inflammation of the lid margins (anterior blepharitis) which is commonly associated with secondary ocular effects such as styes, chalazia and conjunctival or corneal ulceration (see FIG. 40.11). Posterior blepharitis, which involves abnormalities of the submucus meibomian glands at the rim of the eyelids, is frequently associated with seborrhoeic dermatitis (especially) and atopic dermatitis, and less so with rosacea.⁸ There is a tendency to colonisation of the lid margin with *S. aureus*, which causes an ulcerative infection. May have lash loss and trichiasis if chronic.

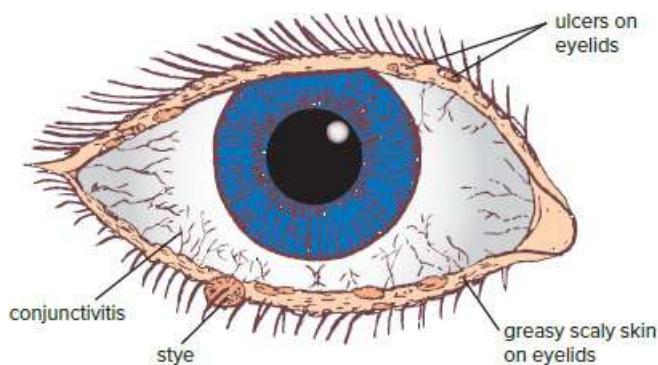


FIGURE 40.11 Blepharitis: common complicating features

The two types are:

- anterior blepharitis—staphylococcal
- posterior blepharitis—seborrhoeic (mainly) and rosacea

Clinical features⁹

- Persistent sore eyes or eyelids
- Irritation, grittiness, burning, dryness and ‘something in the eye’ sensation, worse in mornings
- Lid or conjunctival swelling and redness
- Crusts or scales around the base of the eyelids
- Discharge or stickiness, especially in morning
- Inflammation and crusting of the lid margins

Management

Anterior blepharitis

- Eyelid hygiene is the mainstay of therapy. The crusts and other debris should be gently cleaned with a cotton wool bud dipped in a 1:10 dilution of baby shampoo or a solution of sodium bicarbonate, once or twice daily. Application of a warm water compress or saline soak with gauze for 5 minutes is also effective. Proprietary lid solutions or wipes can also be used. If not controlled, apply chloromycetin 1% ointment once or twice daily for up to 4 weeks and review. Refer resistant cases.

Posterior blepharitis

- Follow the same hygiene methods as above but with firm eye massage in a circular motion towards the lid margins to closed eyes, for 5–10 minutes twice a day.
- Ocular lubricants such as artificial tear preparations may greatly relieve symptoms of keratoconjunctivitis sicca (dry eyes).
- Control scalp seborrhoea with regular medicated shampoos.
- If persistent, short-term use of a mild topical corticosteroid ointment (e.g. hydrocortisone 0.5%) can be effective.
- Treat infection with an antibiotic ointment smeared on the lid margin (this may be necessary

for several months) (e.g. tetracycline hydrochloride 1% or framycetin 0.5% or chloramphenicol 1% ointment to lid margins 3–6 hourly).⁸

- If not controlled by topical measures, use systemic antibiotics such as doxycycline 50 mg daily for at least 8 weeks (erythromycin for children <8 years), or flucloxacillin may be required for lid abscess.
- Avoid wearing make-up and contact lenses if inflammation is present.

Dacryocystitis

Acute dacryocystitis is infection of the lacrimal drainage system secondary to obstruction of the nasolacrimal duct at the junction of the lacrimal sac (see FIG. 40.12). Inflammation is localised over the medial canthus. There is usually a history of a watery eye for months beforehand. The problem may vary from being mild (as in infants) to severe with abscess formation. Page 493



FIGURE 40.12 Acute dacryocystitis with abscess formation. Associated obstruction of the nasolacrimal duct at the junction of the lacrimal sac, which has become infected.

Management

- Use local heat: steam or a hot moist compress.
- Use analgesics.
- In mild cases, massage the sac and duct with warm compresses, and instil astringent drops (e.g. zinc sulphate + phenylephrine) or chloramphenicol 0.5% eye drops if inflammation.
- For acute cases, systemic antibiotics are best guided by results of Gram stain and culture but initially use di/flucloxacillin or cephalexin.
- Measures to establish drainage are required eventually. Recurrent attacks or symptomatic

watering of the eye are indications for surgery such as dacryocystorhinostomy.

Dacryoadenitis

Dacryoadenitis is infection of the lacrimal gland presenting as a tender swelling on the outer upper margin of the eyelid. It may be acute or chronic and has many causes. It is usually caused by a viral infection (e.g. mumps), which is treated conservatively with warm compresses. Bacterial infection is treated with appropriate antibiotics.

§ Orbital cellulitis

Orbital cellulitis includes two basic types—peri-orbital (or preseptal), which is soft tissue infection of the eyelids, and orbital (or postseptal) cellulitis. The latter, which arises from infection of the paranasal sinus, dental abscess or orbital trauma, is a potentially blinding and life-threatening condition. It is especially important in children in whom blindness may develop in hours. The patient, often a child, presents with unilateral swollen eyelids that may be red. Ask about a history of sinusitis, peri-ocular trauma, surgery, bites and immunocompromise issues.

Features to look for in orbital cellulitis include:⁶

- a systemically unwell patient
- proptosis
- peri-ocular swelling and erythema
- tenderness over the sinuses
- ocular nerve compromise (reduced vision, impaired colour vision or abnormal pupils)
- restricted and painful eye movements (see FIG. 40.13)

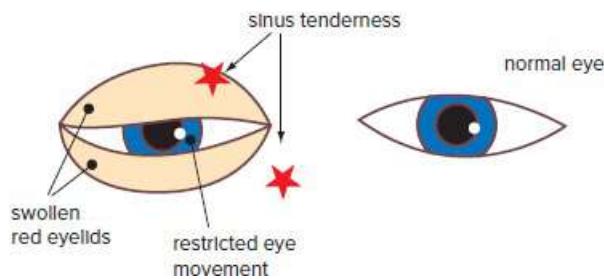


FIGURE 40.13 Important signs in the patient presenting with orbital cellulitis

In peri-orbital cellulitis, which usually follows an abrasion, there is no pain or restriction of eye movement (see FIG. 40.14).



FIGURE 40.14 Peri-orbital cellulitis following an abrasion to the eye. Treat this as an urgent condition.

Immediate referral to hospital for specialist treatment is essential for both types. Treatment is usually with IV cefotaxime until afebrile, then amoxicillin/clavulanate for 7–10 days for peri-orbital cellulitis and for orbital cellulitis, IV cefotaxime + flucloxacillin together followed by amoxicillin/clavulanate (o) 10 days.³

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⌚ Herpes zoster ophthalmicus

Herpes zoster ophthalmicus (shingles) affects the skin supplied by the ophthalmic division of the trigeminal nerve. The eye may be affected if the nasociliary branch is involved. The rash usually appears on the tip of the nose. Ocular problems include conjunctivitis, uveitis, keratitis and glaucoma.

Immediate referral is necessary if the eye is red, vision is blurred or the cornea cannot be examined. Apart from general eye hygiene, treatment usually includes one of the oral antiherpes virus agents such as oral aciclovir 800 mg, five times daily for 10 days or (if sight is threatened) aciclovir 10 mg/kg IV slowly 8 hourly for 10 days (provided this is commenced within 3 days of the rash appearing)^{5,8} and topical aciclovir ointment 4 hourly (see CHAPTER 114).

⌚ Pinguecula and pterygium¹⁰

Pinguecula is a yellowish elevated nodular growth on either side of the cornea in the area of the palpebral fissure. It is common in people over 35 years. The growth tends to remain static but

can become inflamed—pingueculitis. Usually no treatment is necessary unless they are large, craggy and uncomfortable, when excision is indicated. If irritating, topical astringent drops such as naphazoline compound drops (e.g. Albalon) can give relief.

Pterygium is a fleshy overgrowth of the conjunctiva onto the nasal side of the cornea and usually occurs in adults living in dry, dusty, windy areas. Excision of a pterygium by a specialist is indicated if it is likely to interfere with vision by encroaching on the visual axis, or if it becomes red and uncomfortable or disfiguring.

Corneal disorders¹¹

Patients with corneal conditions typically suffer from ocular pain or discomfort and reduced vision. The common condition of dry eye may involve the cornea while contact lens disorders, abrasions/ulcers and infection are common serious problems that threaten eyesight. Inflammation of the cornea—keratitis—is caused by factors such as UV light, e.g. ‘arc eye’, herpes simplex, herpes zoster ophthalmicus and the dangerous microbial keratitis. Bacterial keratitis is an ophthalmological emergency that should be considered in the contact lens wearer presenting with pain and reduced vision.

Topical corticosteroids should be avoided in the undiagnosed red eye.

Corneal abrasion and ulceration

There are many causes of abrasions, particularly trauma from a foreign body embedded on the corneal surface or ‘cul-de-sac’ FB, contact lenses, fingernails including ‘french nails’, and UV burns. The abrasion may be associated with an ulcer, which is a defect in the epithelial cell layer of the cornea. Common causes of a corneal ulcer are listed in TABLE 40.3 .

Table 40.3 Corneal ulceration: common causes

Trauma

Contact lens wear/injury

Infection—microbial keratitis:

- bacterial (e.g. *Pseudomonas* [contact lens])
- viral (e.g. herpes simplex [dendritic ulcer], herpes zoster ophthalmicus)
- fungal
- protozoal (e.g. *Acanthamoeba*)

Neurotrophic (e.g. trigeminal nerve defect)

Immune-related (e.g. rheumatoid arthritis)

Spontaneous corneal erosion

Chronic blepharitis

Overexposure (e.g. eyelid defects)

Symptoms

- Ocular pain
- Foreign body sensation
- Watering of the eye (epiphora)
- Blepharospasm
- Blurred vision

Diagnosis is best performed using fluorescein staining, ideally with a slit lamp and a cobalt blue filter, or use an ultraviolet light (small LED UV lights can replace the traditional Wood's lamp).

Management (corneal ulcer)

- Stain with fluorescein.
- Check for a foreign body.
- Treat with chloramphenicol 1% ointment qid ± homatropine 2% (if pain due to ciliary spasm).
- Double eye pad (if not infected).
- Review in 24 hours.
- A 6 mm defect heals in 48 hours.
- Consider early specialist referral.

⌚ Superficial punctate keratitis

Punctate keratopathy presents as scattered small lesions on the cornea that stain with fluorescein if they are deep enough. It is a non-specific finding and may be associated with blepharitis, viral conjunctivitis, trachoma, keratitis sicca (dry eyes), UV light exposure (e.g. welding lamps, sunlamps), contact lenses and topical ocular agents. Management involves treating the cause and careful follow-up.

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Practice tips

- Think corneal abrasion if the eye is 'watering' and painful (e.g. caused by a large insect such as a grasshopper or other foreign body).