## Fibrinolytic therapy

If angioplasty is unachievable either through timing or the unavailability of the service (such as in rural locations), thrombolysis is an indication for STEMI and the sooner the better, but preferably within 12 hours of the commencement of chest pain.[5,13] The decision should be made by an experienced consultant, especially as PCI is not usually possible once fibrinolytic therapy has been given.

Second-generation fibrin-specific agents (reteplase, alteplase or tenecteplase) are the agents of choice. Streptokinase can be used but it is inappropriate for use in Aboriginal and Torres Strait Islander people and those who have received it on a previous occasion. There are several other contraindications for the use of fibrinolytic agents.

Further management strategies include:

- Full heparinisation for 24–36 hours (after rt-PA—not after streptokinase), especially for large anterior transmural infarction with risk of embolisation, supplemented by warfarin.
- Use LMW heparin (e.g. enoxaparin 1 mg/kg SC bd or unfractionated heparin 5000–7500 units SC 12 hourly).

Further management of STEMI (?myocardial infarction):

- Antiplatelet therapy: aspirin + clopidogrel
- Beta blocker (if no thrombolytic therapy or contraindications) as soon as possible:

  atenolol 25–100 mg (o) daily

  *or*

  metoprolol 25–100 mg (o) twice daily
- Consider glyceryl trinitrate IV infusion if pain recurs
- Start early introduction of ACE inhibitors (within 24–48 hours) in those with significant left ventricular (LV) dysfunction (and other indications)
- Statin therapy to lower cholesterol
- Treat hypokalaemia
- Consider magnesium sulphate (after thrombolysis)
- Consider frusemide

# Post-AMI drug management

Proven:[1,13,14]

- beta blockers—within 12 hours
- ACE inhibitors—within 24 hours after stabilisation
- aspirin 75–150 mg and clopidogrel 75 mg (o) daily or both (alternatives to clopidogrel: ticagrelor or prasugrel)
- lipid-lowering drugs (e.g. statins)
- anticoagulants (for specific indications, e.g. atrial fibrillation)

Targets:

- BP <140/90 (lower if tolerated); TC <4 mmol/L; LDLC <2 mmol/L; TG <2 mmol/L

## Ongoing management

- Education and counselling
- Bed rest 24–48 hours
- Continuing ECG monitoring
- Check serum potassium and magnesium
- Early mobilisation to full activity over 7–12 days
- Light diet
- Sedation
- Beta blocker (o): atenolol or metoprolol
- Anticoagulation where indicated (certainly if evidence of thrombus with echocardiography)
- ACE inhibitors for left ventricular failure and to prevent remodelling
- Monitor psychological issues (e.g. anxiety)

## On discharge

- Rehabilitation program
- Continued education and counselling
- No smoking

- Reduce weight
- Regular exercise, especially walking
- Exercise test (consider, if result will change management)
- Continue beta blockers for 2 years
- Continue ACE inhibitors
- Aspirin 100–150 mg daily and clopidogrel 75 mg daily
- Anticoagulation where indicated (at least 3 months)
- Statin therapy
- Follow-up studies, e.g. myocardial perfusion

# Special management issues

## Indications for coronary angiography

- Development of angina
- Strongly positive exercise test
- Consider after use of streptokinase

## Management of the extensive infarction

- ACE inhibitors (even if no CCF)
- Radionuclide studies (to assess left ventricular function)
- Beta blockers (proven value in severe infarction) if no contraindications or LV dysfunction
- Anticoagulation



## Treating and recognising complications of STEMI

### Acute left ventricular failure

- Signs: basal crackles, extra (third or fourth) heart sounds, X-ray changes
- Treatment (according to severity) (refer to CHAPTER 76 ):

  oxygen

diuretic (e.g. frusemide)

morphine IV

glyceryl trinitrate: IV, SL (o) or topical

ACE inhibitors

### Cardiogenic shock (a major hospital management procedure)

Requires early specialist intervention which may include:

- adrenaline—titrated to BP
- treat hypotension with inotropes
- intra-aortic balloon pump
- urgent angiography ± angioplasty/surgery

### Pericarditis

This occurs in first few days after AMI (usually anterior AMI), with onset of sharp pain.

- Signs: pericardial friction rub
- Treatment: anti-inflammatory medication (e.g. aspirin, indomethacin or ibuprofen for pain) with caution

*Note:* Avoid anticoagulants.

### Post-AMI syndrome (Dressler syndrome)

This occurs weeks or months later, usually around 6 weeks.

- Features: pericarditis, fever, pericardial effusion (an autoimmune response)
- Treatment: as for pericarditis

### Left ventricular aneurysm

This is a late complication.

- Clinical: cardiac failure
- Features: arrhythmias, embolisation
- Signs: double ventricular impulse, fourth heart sound, visible bulge on X-ray

- Diagnosis: 2D electrocardiography
- Treatment:

    antiarrhythmic drugs

    anticoagulants

    medication for cardiac failure

    possible aneurysmectomy

### Right ventricular infarction

This may accompany inferior MI and is life-threatening.

### Ventricular septal rupture and mitral valve papillary rupture

This presents with severe cardiac failure and a loud pansystolic murmur. Both have a poor prognosis and early surgical intervention may be appropriate.

### Cardiac arrhythmias

All types are common with STEMI and require treatment according to guidelines in CHAPTER 59 . Methods may include defibrillation, cardioversion and pacemaking. Post infarct prophylaxis with IV lignocaine is not indicated.[5,16]

### Anxiety and depression

Patients require anticipatory guidance and support including education, reassurance and counselling. If necessary, anxiolytic agents and antidepressants may help recovery.



# Management of other serious spontaneous causes of chest pain

## Aortic dissection

- Early definitive diagnosis is necessary: best achieved by transoesophageal echocardiography.
- 50% of patients are hypertensive; so need pharmacological control of hypertension with IV nitroprusside and beta blockers.
- Emergency surgery needed for many, especially for type A (ascending aorta involved).

*Note:* Increased incidence during pregnancy.

### Pulmonary embolus

Investigations to diagnose suspected pulmonary embolus (choose from):[6,17,18]

- chest X-ray and ECG
- CT pulmonary angiography (first-line study)
- radionucleide imaging—the ventilation/perfusion (V/Q) study
- digital subtraction angiography
- D-dimer assay—sensitive for 'ruling out' in low risk, but not specific for 'ruling in'
- Doppler sonography of lower limbs
- arterial blood gases
- Wells score: if >3, highly probable; if >6, diagnostic

## Management (specialist care)

- Needs supportive medical care and anticoagulation:

  DOACs

  *or*

  heparin IV: 5000 U as immediate bolus, continuous infusion 30 000 U over 24 hours or 12 500 U (sc) bd

  *or*

  low molecular weight heparin

*Note:* Thrombolytic therapy either IV or into the pulmonary artery can be used for major embolism. Surgical embolectomy is rarely necessary but needed if very extensive.

### Pneumothorax[19]

- Can be spontaneous (more common in COPD or asthma) or traumatic. Most episodes resolve spontaneously without drainage.
- Spontaneous pneumothoraces in a healthy adult should initially be managed conservatively with analgesia (and oxygen if necessary) even if large.

- Recent trials have shown conservative management to be superior to pleural intervention in terms of adverse events, complications and days in hospital.
- Pleural intervention with a catheter is necessary for clinical deterioration: falling BP and oxygen saturation, rising pulse and respiratory rate.
- For recurrent attacks, excision of cysts or pleurodesis may be necessary.
- Statistics indicate a 30–50% recurrence rate of spontaneous pneumothorax (most within 12 months), 35% on the same side, 10–15% on the opposite side. Recurrence should not recur after a pleurodesis where the lung surface has been rendered adherent to the chest wall.

### Acute tension pneumothorax

For urgent cases insert a 12–16 gauge needle into the pleural space through the second intercostal space on the affected side. Replace with a formal intercostal catheter connected to underwater seal drainage.

## Treatment of oesophageal disorders

### Gastro-oesophageal reflux

- Achieve normal weight if overweight.
- Avoid coffee, alcohol and spicy foods.
- Avoid large meals and overeating (keep to small meals).
- Use antacids or alginate compounds (e.g. Gaviscon, Mylanta Plus).
- If persistent:

  acid suppression—$H_2$-receptor blockers (e.g. ranitidine)

  *or*

  proton-pump inhibitors (e.g. omeprazole)
- Reassess PPIs after 4–6 weeks; consider deprescribing long-term PPIs

### Oesophageal spasm[20]

Long-acting nitrates (e.g. isosorbide dinitrate 10 mg tds)

*or*

calcium-channel blockers (e.g. nifedipine CR 20–30 mg once daily)

*Note:* Attend to lifestyle and dietary factors, as for reflux.

# Musculoskeletal causes of chest wall pain

There are many musculoskeletal causes, most of which can be eliminated by the history and physical examination. Some of the causes listed in TABLE 30.9 are very uncommon and often part of a general disorder, such as ankylosing spondylitis. Muscular tears or strains of the chest wall are quite common. A differential diagnosis is a fractured rib including a cough fracture.

**Table 30.9** Musculoskeletal causes/origins of chest wall pain (front and back)

Injury to thoracic spine → dysfunction

Vertebral fracture:

- trauma
- pathological:
    - osteoporosis
    - metastatic disease
    - multiple myeloma

Intercostal muscle strains/tears

Rib disorders:

- fractures
- slipping rib

Costochondritis

Tietze syndrome

Fibromyalgia

Musculoskeletal chest pain is typically aggravated or provoked by movements such as stretching, deep inspiration, sneezing and coughing. The pain tends to be sharp and stabbing in quality but can have a constant aching quality.



Costochondritis is a common cause of anterior pain, which is generally well localised to the costochondral junction and may also be a component of an inflammatory disorder, such as one of the spondyloarthropathies.

Management is generally conservative with analgesics, gentle massage with analgesic creams and NSAIDs if there is an inflammatory component. Other measures that can help for very

painful chest wall problems are localised injections of local anaesthetic with or without corticosteroids (with care not to penetrate the parietal pleura) and a modified support (especially for rib injuries) in the form of a special elasticised rib belt (called a universal rib belt) that gives support and symptom relief while permitting adequate lung expansion.

## Posterior chest (thoracic back) pain

Disorders of the musculoskeletal system represent the most common cause of thoracic (dorsal) back pain, especially dysfunction of the joints of the thoracic spine. Refer to CHAPTER 27 for more detail. Probably the commonest cause is costovertebral dysfunction caused by overstress of rib articulations with vertebrae (the costovertebral joints). This fact is clearly demonstrated with the midline thoracic back pain following cardiac surgery when these joints are compressed during sternotomy and splaying of the chest walls.

The back pain may be associated with simultaneous referred anterior chest pain or abdominal pain.

## Acute thoracic back pain

Although posterior pain is invariably caused by vertebral dysfunction, there are several other important causes, including serious bone disease (leading to compression fractures) and life-threatening visceral and vascular causes. Refer to red flag pointers and TABLE 27.3 and management guidelines in CHAPTER 27 .

*Note:*

- Intervertebral disc protrusions are rare in the thoracic spine.
- Rarely, a penetrating peptic ulcer can present with mid to lower thoracic back pain.

# When to refer

- Obvious or suspected myocardial infarction, especially with extensive infarction
- Transfer to major centre with complications of AMI:

    rupture of septum or papillary muscle

    aneurysm

    refractory arrhythmias

    cardiogenic shock

- Patients with persistent post-infarction angina

- Angina:

  angina not responding to drug treatment

  unstable angina

  angina lasting for longer than 15 minutes (unresponsive to sublingual nitrate) needs urgent hospital admission

- Suspected or proven pulmonary embolus or dissecting aneurysm or other serious life-threatening problem (after initial first-line measures, e.g. decompression of tension pneumothorax)

- Suspected oesophageal or other gastrointestinal disorder (e.g. duodenal ulcer), for endoscopy or appropriate gastroenterological evaluation

## Practice tips

- All sudden acute chest pain is cardiac (and potentially fatal) until proven otherwise.
- A careful history is the basis of the diagnosis.
- Mitral valve prolapse can be an undiagnosed cause of chest pain: keep it in mind if pain is recurrent and intermittent (confirm with echocardiography).
- Calcium antagonists can cause peripheral oedema, so be careful not to attribute this to heart failure.
- The pain of oesophageal spasm can be very severe and mimic myocardial infarction.
- Oesophageal spasm responds to glyceryl trinitrate: do not confuse with angina.
- Intervertebral disc protrusions are a very rare cause of severe sudden thoracic pain (T2–9).
- Infective endocarditis can cause pleuritic posterior chest pain.
- GPs need to carefully monitor patients on anticoagulants. The INR ratio (usual range 2–3, mechanical mitral valve 2.5–3.5) should be tested at least monthly.
- The sudden onset of dyspnoea without chest pain can occur frequently with (painless) myocardial infarction and pulmonary embolism.
- If a person recovering from an AMI suddenly develops shortness of breath, consider ventricular septal rupture, mitral valve papillary rupture (with mitral

regurgitation), pulmonary embolus and other serious complications.

- Treat (indefinitely) all post-MI patients with ACE inhibitors, and consider continuing beta blockers beyond 12 months post-MI if reduced ejection fraction (<40%) or ongoing angina.[21]
- Use antiplatelet agents indefinitely—100–300 mg aspirin daily or, if contraindicated, clopidogrel 75 mg daily.[21]



## Patient education resources

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

- Angina
- Cardiovascular (including coronary) risk factors

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# 31 Constipation

*I have finally kum to the konklusion, that a good reliable set of bowels iz wurth more tu a man, than enny quantity of brains.*

HENRY SHAW (1818–1885), *JOSH BILLINGS*

Constipation is the difficult passage of small hard stools. The Rome III criteria define it has having two or more of the following, for at least 12 weeks:

- infrequent passage of stools <3/week
- passage of lumpy or hard stools at least 25% of time
- straining >25% of time
- sensation of incomplete evacuation >25% of time
- use of manual manoeuvres >25% of time
- sensation of anorectal obstruction/blockage >25% of time

Accordingly it affects more than 1 in 5 in the population.[1]

However, the clinical emphasis should be on the consistency of the stool rather than on the frequency of defecation; for example, a person passing a hard stool with difficulty once or twice a day is regarded as constipated, but the person who passes a soft stool comfortably every two or three days does not require any diagnosis. Various causes of chronic constipation are summarised in FIGURE 31.1 .

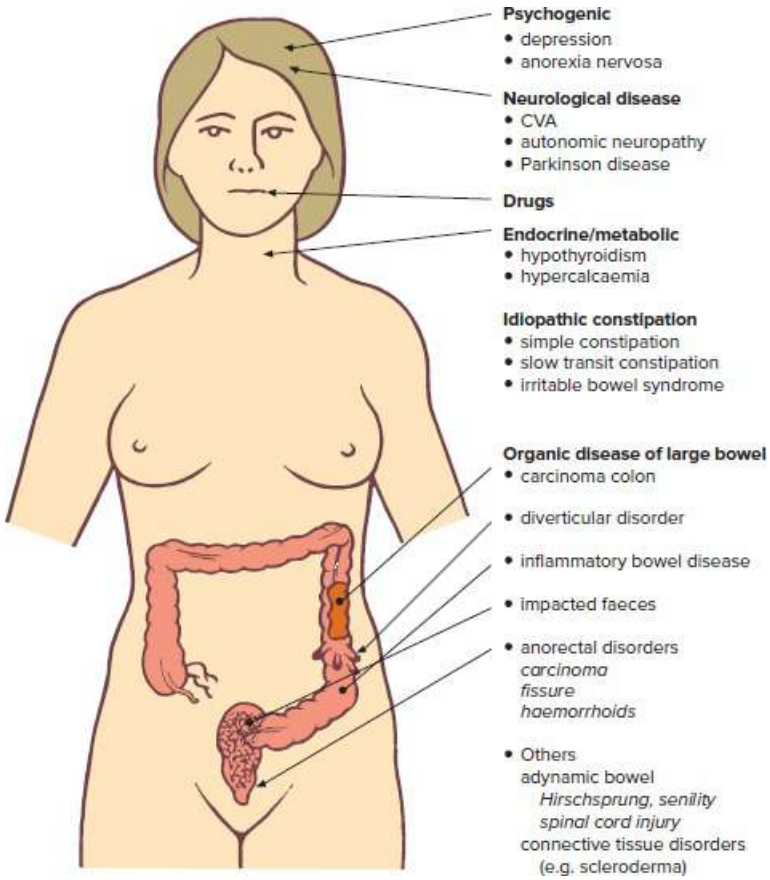


**FIGURE 31.1** Causes of chronic constipation

## Key facts and checkpoints

- The survey showed 10% of adults and 6% of children reported constipation in the preceding 2 weeks.[1]
- Up to 20% of British adults regularly take laxatives.[2]
- Constipation from infancy may be due to Hirschsprung disorder.
- Diet is the single most important factor in preventing constipation.
- Beware of recent-onset constipation in the middle-aged and the elderly.
- Bleeding suggests cancer, haemorrhoids, diverticular disorder and inflammatory bowel disease.
- Always examine the abdomen and rectum.

- Plain abdominal X-rays are generally not useful in the diagnosis of chronic constipation.
- The flexible sigmoidoscope examines the lower bowel in detail.
- Intractable constipation (obstipation) is a challenge at both ends of the age spectrum but improved agents have helped with management.

# A diagnostic approach

Using the diagnostic strategy model (see TABLE 31.1 ), the five self-posed questions can be answered as follows.

**Table 31.1** Chronic constipation: diagnostic strategy model

| |
|---|
| **Probability diagnosis** |
| Functional constipation[3]<br>• primary—slow transit, dyssynergic defecation<br>• lifestyle—diet, low fluids, bad habits |
| **Serious disorders not to be missed** |
| Intrinsic neoplasia: colon, rectum or anus, especially colon cancer<br>Extrinsic malignancy (e.g. lymphoma, ovary)<br>Hirschsprung (children) |
| **Pitfalls (often missed)** |
| Impacted faeces<br>Local anal lesions, e.g. fissure, haemorrhoids<br>Drug/purgative abuse<br>Hypokalaemia<br>Depressive illness<br>Acquired megacolon<br>Diverticular disease<br>Stricture, e.g. Crohn disease<br>*Rarities:*<br>• lead poisoning<br>• hypercalcaemia<br>• hyperparathyroidism<br>• dolichocolon (large colon)/megarectum |

- Chagas disease
- neuromuscular, e.g. systemic sclerosis

**Seven masquerades checklist**

Depression
Diabetes (autonomic neuropathy)
Drugs (TABLE 31.2 )
Thyroid disorder (hypo); hyperparathyroidism
Spinal dysfunction (severe only)

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

May be functional (e.g. depression, anorexia nervosa).

## Probability diagnosis

The commonest is ‘idiopathic’ constipation where there is no structural or systemic disease. This is also referred to as ‘functional’ constipation.

Probably the most frequent single factor causing constipation in Western society is deficiency in dietary fibre, including fruit, green leafy vegetables and wholemeal products. The amount of fibre in our diet is directly related to stool weight and to colonic transit time. The average colonic transit time in the large bowel for Westerners is 60 hours; for a rural African on a very high-fibre diet it is 30 hours. Other compounding factors are dehydration, lack of physical activity and inappropriate bowel habits. Constipation is also a common problem in pregnancy.

## Serious disorders not to be missed

### Neoplasia

It is obvious that colonic or anorectal neoplasms must not be missed, especially in a middle-aged or elderly person presenting with constipation or change in bowel habit. Undetected neoplasias eventually present with bowel obstruction (complete or incomplete).

Extrinsic malignancy, such as lymphoma or ovarian cancer, compressing or invading the rectum also has to be considered. Cancer of the large bowel is prevalent in our society and those aged 50–74 years should be strongly encouraged to participate in the National Bowel Cancer Screening Program.

### Megacolon

In children it is important to detect the presence of megacolon, for example, megacolon secondary to Hirschsprung disorder. Symptoms dating from birth suggest Hirschsprung disorder, which occasionally may present for the first time in adult life.

### Neurological disorders

Constipation, often with faecal impaction, is a common accompaniment to paraplegia, multiple sclerosis, cerebral palsy and autonomic neuropathy.

#### Alarm symptoms

- Recent constipation in >40 years of age
- Rectal bleeding/haematochezia (fresh blood)
- Family history of cancer
- Positive FOBT

## Pitfalls

The pitfalls can be summarised as follows:

- impacted faeces
- depressive illness
- purgative abuse
- local anal lesions
- drugs

Those with impacted faeces often present with spurious (paradoxical) diarrhoea. This is a form of idiopathic constipation and is very commonly encountered in general practice, especially in bedridden elderly people.

Anal pain or stenosis, such as fissure-in-ano, thrombosed haemorrhoids, perianal haematoma or ischiorectal abscess, lead to constipation because the person is hesitant to defecate.

### General pitfalls and tips

- Ensure the person is truly constipated, and not having unrealistic expectations of regularity.
- Ensure that the anthraquinone group of laxatives, including ‘Ford pills’, is never used long term because they cause melanosis coli and associated megacolon.
- Be very wary of alternating constipation and diarrhoea (e.g. colon cancer).

- In a busy practice be careful not to let ‘familiarity breed contempt’ (e.g. onset of hyperparathyroidism, cancer).
- A normal rectal examination does not exclude cancer.

## Seven masquerades checklist

Three of the primary masquerades (see TABLE 31.1 ) are important causes of constipation, namely drugs, depression and hypothyroidism. Many drugs (see TABLE 31.2 ) may be associated with constipation, especially codeine and its derivatives, antidepressants, aluminium and calcium antacids. Cations that constipate include: barium, calcium, aluminium, iron, bismuth. A careful drug history is thus mandatory, because fortunately the constipation usually resolves once the drug is withdrawn. Constipation can be a significant symptom in all types of depressive illness and may be aggravated by treatment with antidepressants.

**Table 31.2** Drugs associated with constipation

| |
|---|
| Analgesics (inhibitors of prostaglandin synthesis) |
| Antacids (containing calcium carbonate or aluminium hydroxide) |
| Anticholinergic agents, antispasmodics |
| Antidiarrhoeal agents |
| Anti-epileptics |
| Antihistamines ($H_1$-receptor blockers)* |
| Antiparkinson drugs* |
| Antipsychotic drugs,* e.g. clozapine, risperidone |
| Barbiturates |
| Barium sulphate |
| Benzodiazepines |
| Calcium-channel blockers (verapamil) |
| Calcium supplements |
| Cholestyramine |
| Clonidine |
| Cough mixtures |
| Cytotoxic drugs |
| Diuretics that cause hypokalaemia |
| Gabapentin |
| Ganglionic blocking agents |
| Heavy metal (especially lead) |
| 5-$HT_3$-receptor antagonists, e.g. ondansetron |
| Iron supplements |

Laxatives (chronic use)
Monoamine oxidase inhibitors
Muscle relaxants
Opioid analgesics (e.g. codeine)
SSRIs
Tricyclic antidepressants*

*Denotes anticholinergic effect.

The metabolic causes of constipation include hypothyroidism, and the rarer hypercalcaemia and porphyria.

Diabetes rarely can be associated with constipation when an autonomic neuropathy can lead to alternating bouts of constipation and diarrhoea.



## Psychogenic considerations

Constipation may be a manifestation of an underlying functional problem and psychiatric disorder, such as depression, anorexia nervosa, schizophrenia or drug misuse. Narcotic misuse must always be considered, and laxatives may cause rebound constipation. More commonly, it may reflect an inactive lifestyle and provide a good opportunity for appropriate counselling.

# The clinical approach

## History

It is important to ask patients to define exactly what they mean by constipation. Some people believe that just as the earth rotates on its axis once a day, so should their bowels open daily to ensure good health. As always, a careful history is appropriate, including stool consistency, frequency, ease of evacuation, pain on defecation and the presence of blood or mucus. A dietary history is very relevant.

### Key questions

- How often do you go to the toilet?
- What are your bowel motions like?
- Are they bulky and hard, like rabbit pellets, or soft?
- Is there pain on opening your bowels?

- Have you noticed any blood?
- Have you noticed any lumps?
- Do you have any soiling on your underwear?
- How do you feel in yourself?
- What medications are you taking?

### Diary

Ask the patient to keep a 10-day diary recording frequency and nature of stools, and whether any difficulty was experienced when passing stool.

## Examination

The important aspects are abdominal palpation and rectal examination. Palpation may reveal the craggy mass of a neoplasm, faecal retention (especially in the thin patient) or a tender spastic colon. The perianal region should be examined for localised disease. The patient should be asked to bear down to demonstrate perianal descent, haemorrhoids or mucosal prolapse. Perianal sensation and the anal reflex should be tested. Digital rectal examination is mandatory, and may reveal a rectal tumour and faecal impaction, as well as testing for rectal size and tone. If there is a history from infancy, a normal or narrow rectum suggests congenital megacolon (Hirschsprung disorder) but, if dilated, acquired megacolon.

General signs that may be significant in the diagnosis of constipation are summarised in FIGURE 31.2 .

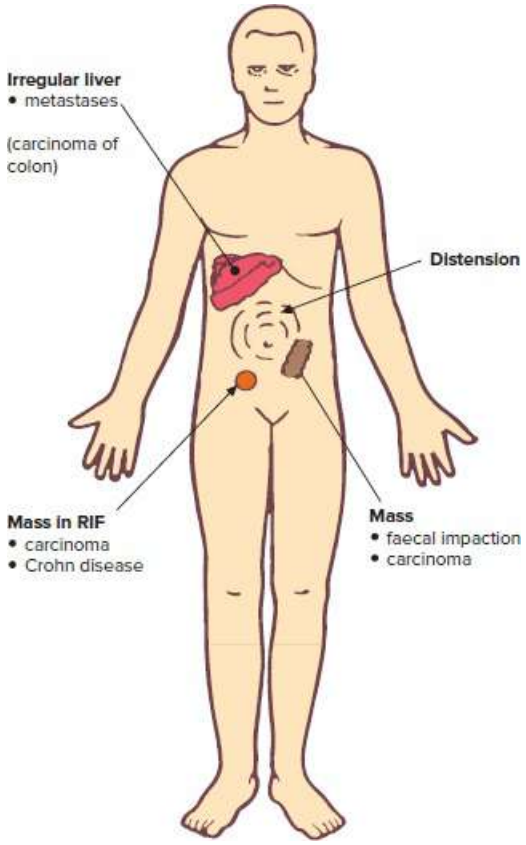


**FIGURE 31.2** Possible significant abdominal signs in the patient with constipation

## Rectal examination

The most important first step is to *do* the examination.

### Method

- Explain to the patient what will happen.
- After inspection with the patient in the left lateral position and with knees drawn up, a lubricated gloved index finger is placed over the anus.
- Part the buttocks.
- Ask the patient to concentrate on slow deep breathing.
- With gentle pressure the finger is then inserted slowly into the anal canal and then into the rectum (it helps patient comfort if they push down or squeeze to accommodate the finger).
- Rotate the finger anteriorly to feel the prostate in males and the cervix in females.

- The finger will reach to about 7–8 cm with gentle thrusting into the perineum.
- Examine the whole circumference of the rectum by sweeping the finger from posterior on both sides.

### Points to note

- Any pain: fissure, proctitis, excoriation from diarrhoea (a rectal examination will not be possible in the presence of a fissure)
- Induration from a chronic fissure or fistula in the anal canal
- The sphincter tone
- The nature of the faeces (?impaction)
- The rectal wall: cancer is usually indurated, elevated and ulcerated; a villous adenoma has a soft velvety feel
- Posteriorly: the sacrum and coccyx
- Laterally: the side walls of the pelvis
- Anteriorly: cervix and pouch of Douglas in the female; prostate and rectovesical pouch in the male

### Prostate examination

- It feels larger if the patient has a full bladder.
- The normal prostate is a firm smooth rubbery bilobed structure (with a central sulcus) about 3 cm in diameter.
- A craggy hard mass suggests cancer.
- An enlarged smooth mass suggests benign hypertrophy.
- A tender, nodular or boggy mass suggests prostatitis.

## Practice tip on treatment

Before resorting to a good old-fashioned ‘3H’ enema (hot water, high and a hell of a lot), use a sorbitol compound (e.g. Microlax 5 mL enema). It can be carried in the doctor’s bag, is very easy to insert and is most effective.

### A common pitfall

In the female, the cervix or a vaginal tampon can be mistaken for a mobile extrarectal tumour.

## Endoscopy

Sigmoidoscopy—in particular, flexible sigmoidoscopy with examination of the rectosigmoid—is important in excluding local disease; search for abnormalities such as blood, mucus or neoplasia. The insufflation of air sometimes reproduces the pain of the irritable bowel syndrome.

It is worth noting that 60% of polyps and cancers will occur in the first 60 cm of the bowel[4] and diverticular disorder should be evident with the flexible sigmoidoscope.

The presence of melanosis coli is an important sign—it may give a pointer to the duration of the constipation and the consequent chronic intake (perhaps denied) of anthraquinone laxatives.

# Investigations

These can be summarised as follows:

- Haematological:

  haemoglobin

  ESR

- Stools for occult blood

- Biochemistry (where suspected):

  thyroid function tests

  serum calcium

  serum potassium

  carcinoembryonic antigen (a targeted tumour marker rather than a screen)

- Radiological:

  CT colonography (virtual colonography)

  double contrast barium enema (especially for primary colonic disease, e.g. megacolon)

  bowel transit studies, using radio-opaque shapes taken orally and checking progress by abdominal X-ray or stool collection

- Physiological tests:

anal manometry—test anal tone

rectal sensation and compliance, using an inflatable rectal balloon

dynamic proctography, to determine disorders of defecation

rectal biopsy, to determine aganglionia

# Idiopathic constipation

It is best to classify idiopathic constipation into three subgroups:

1 simple constipation

2 slow transit constipation

3 normal transit constipation (irritable bowel syndrome)

Of these, the commonest is simple constipation, which is essentially related to a faulty diet and bad habit. Avery Jones,[5] who defined the disorder, originally described it as being due to one or more of the following causes:

- faulty diet—inadequate dietary fibre
- neglect of the call to stool
- unfavourable living and working conditions
- lack of exercise
- travel

*Dyschezia*, or lazy bowel, is the term used to describe a rectum that has become unresponsive to faecal content, and this usually follows repeated ignoring of calls to defecate.



Slow transit constipation occurs primarily in women with an apparently normal colon, despite a high-fibre intake and lack of the other causes described by Avery Jones. Many are young, with a history dating from early childhood or, more commonly, adolescence. Constipation may follow childbirth, uncomplicated abdominal surgery or a period of severe dieting. However, in the majority no precipitating cause is evident.

A *defecatory disorder* is where there is a paradoxical contraction rather than normal relaxation of the anal sphincter and associated muscles responsible for evacuation. Also known as dyssynergic dysfunction.

## Management

Most patients have simple constipation and require reassurance and education once an organic cause has been excluded. Encourage modification of lifestyle. Provide psychological counselling and biofeedback for dyssynergic problems.

### Advice to patients

- Adequate exercise, especially walking, is important.
- Develop good habits: answer the call to defecate as soon as possible. Develop the ‘after breakfast habit’. Allow time for a good relaxed breakfast and then sit on the toilet. Don’t miss meals—food stimulates motility.
- Avoid codeine compounds (tablets or mixture).
- Take plenty of fluids, especially water and fruit juices (e.g. prune juice).
- Eat an optimal bulk diet. Eat foods that provide bulk and roughage, such as vegetables and salads, cereals (especially wheat fibre), fresh and dried fruits, and wholemeal bread. Enough fibre should be taken to convert stools that sink into stools that float.

Examples of food with good bulk properties are presented in TABLE 31.3 .[6] Fruit has good fibre, especially in the skin, and some have natural laxatives (e.g. prunes, figs, rhubarb, apricots).

**Table 31.3** Foods with bulk-forming properties (from least to most)

| |
|---|
| Potato |
| Banana |
| Cauliflower |
| Peas |
| Cabbage |
| Lettuce |
| Apple |
| Carrot |
| Bran |

## Treatment (pharmaceutical preparations)

Some patients may not tolerate unprocessed bran but tolerate pharmaceutical preparations better (see TABLE 31.4 ). An appropriate choice would be one of the hydrophilic bulk-forming agents such as ispaghula or psyllium. Avoid stimulant laxatives except for short sharp treatments.

**Table 31.4** Therapeutic agents (laxatives) to treat constipation (with examples)

| |
|---|
| **Hydrophilic bulk-forming agents** |
| Psyllium mucilloid (Agiofibe, Metamucil) |
| Sterculia (Granocol, Normacol) |
| Ispaghula (Agiolax, Fybogel) |
| Methylcellulose |
| Wheat bran/dextrin (Benefiber) |
| Crude fibre (Fibyrax Extra) |
| **Stimulant (irritant) laxatives** |
| Sodium picosulfate |
| Anthraquinones: senna (Senokot/Sennetabs), senna with dried fruits (Nu-Lax), sennosides A and B; cascara |
| Frangula bark (in Normacol Plus) |
| Castor oil |
| Triphenylmethanes: bisacodyl (e.g. Dulcolax); picosulfate |
| **Osmotic laxatives** |
| Macrogol 3350 with electrolytes (e.g. Movicol) |
| Magnesium sulphate (Epsom salts/Colocap Balance) |
| Magnesium hydroxide (milk of magnesia) |
| Lactulose (several agents) |
| Mannitol |
| Sodium phosphate mixture |
| Sorbitol (Sorbilax) |
| Saline laxatives |
| **Stool-softening/lubricating agents** |
| Liquid paraffin (Agarol) |
| Docusate—poor evidence of efficacy |
| Poloxamer |
| Glycerin suppositories |
| Sorbital/sodium compounds (Microlax) |
| **Laxatives in suppository form** |

Glycerin/glycerol suppository
Sorbitol sodium compounds (e.g. Fleet Enema)
Sodium phosphate enema (e.g. Fleet)
Stimulant microenema or suppository (e.g. Bisa-lax)
Stool-softener microenema (e.g. Enamax)

**Prokinetic agent**

Prucalopride

*Note:* ColonLYTELY used for colonoscopy preparation contains macrogol, sodium sulphate and other mineral salts.

## First-line therapy[7]

Use a general bulking agent, e.g. psyllium or ispaghula granules 1–2 teaspoons (o) once or twice daily, or commercial products as per suggested dose.

## Second-line therapy

Use an osmotic laxative or a fibre-based stimulant preparation, e.g. macrogol 3350 + 1–2 sachets, each dissolved in 125 mL water once daily

*or*

lactulose syrup 15–30 mL (o) daily until response, then 10–20 mL daily

*or*

dried fruits with senna leaf (Nu-Lax) 10 g nocte

*or*

docusate + senna (50–80 mg), 1–2 tabs nocte

## Third-line therapy

(Recheck cause.)

Magnesium sulphate 1–2 teaspoons (15 g) in water once or twice daily (if normal kidney function)

*or*

as capsules (Colocap Balance) 15 caps over 15 minutes

*or*

combined bulking/stimulating agent (e.g. frangula/sterculia [Normacol plus])

*or*

glycerin suppository (retain for 15–20 minutes)

*or*

sodium citrate or phosphate enema (e.g. Fleet Enema)

*or*

Microlax enema



## Constipation in children

Constipation is quite common in children and is idiopathic in 95%. The most common factor is diet. Constipation often begins after weaning or with the introduction of cow's milk. It is rare with breastfeeding. Low fibre intake and a family history of constipation may be associated factors.[8] Most children develop normal bowel control by 4 years of age (excluding any physical abnormality). It is normal to have a bowel movement every 2–3 days, providing it is of not unusual consistency and is not painful.

Constipation usually appears between 2 and 4 years of age, and up to a third of primary school-aged children will report constipation over a 12-month period. In toddlers, the gender distribution is equal, but by age 5, boys are more likely to get constipation than girls, with the frequency of faecal incontinence three times higher in boys. Consider constipation in a child who has recently recommenced bedwetting.

Constipation in children is defined as having two or more of the following over the previous 2 months:

- <3 bowel motions per week
- >1 episode of faecal incontinence per week (previously referred to as encopresis)
- large stools in rectum or palpable on abdominal examination
- retentive posturing (e.g. 'stiff as a board' standing/lying, tip toes, crossed legs, braces against furniture) and withholding behaviour (e.g. refuses, hides, requests nappy, denies need to go)
- painful defecation

Faecal incontinence, which is a consequence of chronic constipation, is the passage of stool in an inappropriate place in children who have been toilet trained. It can present as soiling (encopresis)

due to faecal retention with overflow of liquid faeces (spurious diarrhoea).

Constipation is nearly always functional (>95%),[7] though the GP should check for any red flags for a pathological cause (see below). The key feature in functional constipation is chronic faecal retention leading to rectal dilatation and insensitivity to the normal defecation reflex.

## Red flag pointers for organic causes in children

- Blood in stools
- Perianal disease
- Fever
- Weight loss/delayed growth
- Delayed meconium/thin strip-like stools
- Vomiting
- Urinary symptoms (although bedwetting fairly common)
- Abnormal neurological findings in legs
- Medications used for children with behavioural/developmental issues

Normally the rectum is empty just prior to defecation; with faecal retention, the rectum is stretched, weak and numb and it can leak.

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## Other important conditions

Hirschsprung disorder:

- consider if delay in passing first meconium stool and subsequent constipation

Anal fissure in infants:

- consider if stool hard and associated with pain or bleeding
- the mainstay of treatment is dietary manipulation

## Principles of treatment of functional constipation[7,8]

- Encourage relaxed child–parent interaction with toilet training, such as appropriate encouragement, ‘after breakfast habit’ training, regular toileting (where possible), three

times/day for 3–5 minutes, reinforce desired behaviour with stickers on an age-appropriate chart.

- Introduce psychotherapy or behaviour modification program, especially where ‘fear of the toilet’ exists.
- Establish an empty bowel: remove any severely impacted faeces with microenemas (e.g. Microlax), and even disimpaction under anaesthesia if necessary, particularly if faecal ‘rocks’ are visible on X-ray.
- Advice for parents of children over 18 months:

  Drink ample non-milk fluids each day—several glasses of water, unsweetened fruit juice (be cautious of cow’s milk).

  Use prune juice, which contains sorbitol.

  Get regular exercise—walking, running, outside games or sport.

  Provide high-fibre foods—high-fibre cereals, wholegrain bread, brown rice, wholemeal pasta, fresh fruit with skins left on where possible, dried fruits such as sultanas, apricots or prunes, fresh vegetables.
- Advice on correct posture and position—‘how to do a poo’:[8]

  Feet supported (e.g. with a foot stool)

  Knees higher than bottom and apart

  Leaning forward with elbows on knees

  Encourage child to push out stomach

  Ensure privacy (including at preschool/school)
- Laxatives—if constipation has been brief in duration, treat for 3 months, but for chronic constipation, treat for 6 months minimum.
- Can use macrogol 3350 (Movicol), paraffin oil or lactulose.
- For acute faecal impaction, high-dose laxatives can be used until liquid stools are achieved, and then revert back to maintenance treatment. Enemas are suitable only for children with acute severe rectal pain or distress and are rarely required.
- Use a pharmaceutical preparation as a last resort to achieve regularity.

#### First line[6]

- Paraffin oil (e.g. Parachoc): RCT evidence indicates suitable and better than stimulant laxative

*or*

osmotic laxative (e.g. lactulose): 1–3 mg/kg

1–5 years: 10 mL per day

>5 years: 15 mL per day

*or*

macrogol 3350 with electrolytes:

2–12 years: 1 sachet Movicol-Half in 60 mL water once daily

>12 years: 1 sachet Movicol (or 2 Movicol-Half) daily

Severe constipation/faecal impaction:

- consider admission to hospital
- abdominal X-ray
- macrogol 3350 with electrolytes (double above doses and water)
- Microlax enema

If unsuccessful, add ColonLYTELY via nasogastric tube *or* sodium phosphate enema (Fleet Enema) (not <2 years).

# Congenital megacolon Hirschsprung disorder (aganglionosis)

## Clinical features

- Constipation and abdominal distension from infancy
- Possible anorexia and vomiting
- Male to female ratio = 8:1
- Rectal examination—narrow or normal rectum
- Abdominal X-ray/barium enema—distended colon full of faeces to narrow rectum
- Diagnosis, confirmed by full thickness biopsy, shows absence of ganglion cells
- Absent rectoanal reflex on anal manometry

### Treatment

Resect narrow segment after preliminary colostomy.



## Acquired megacolon

### Clinical features

- In older children and adults
- Mainly due to bad habit
- Can be caused by:

    chronic laxative abuse

    milder form of Hirschsprung disorder

    Chagas disease (Latin America)[2]

    hypothyroidism (‘cretinism’)

    systemic sclerosis
- Marked abdominal distension
- Rectal examination—dilate loaded rectum, lax sphincter
- Abdominal X-ray/barium enema—distended colon full of faeces but no narrowed segment

### Treatment

Re-education of bowel habit is required.

## Constipation in the elderly

Constipation is a common problem in the elderly, with a tendency for idiopathic constipation to increase with age. In addition, the chances of organic disease increase with age, especially colorectal cancer, so this problem requires attention in the older patient. Faecal impaction is a special problem in the aged confined largely to bed. Constipation is often associated with Parkinson disease, and various medications. In the elderly, an osmotic laxative such as sorbitol or lactulose may be required for longstanding refractory constipation, but avoid stimulant and other non-osmotic laxatives.

## Faecal impaction

This is a difficult problem, particularly in the older person who may not be aware of the problem, especially if they have spurious diarrhoea. Symptoms include malaise, anorexia and nausea, confusion, headache, abdominal discomfort ± colic and bloating, a sense of inadequate defecation and frequent amounts of small stool. Complications include spurious diarrhoea, faecal incontinence, bowel obstruction, urinary incontinence or retention. It often follows opioid medication. Confirm with rectal examination ± plain X-ray of abdomen. Treat with oral or osmotic laxatives (e.g. 8 sachets of macrogol 3350 for 3 days with or without rectal suppositories) or enema, e.g. Fleet Enema, Microlax.

### Manual disimpaction

If manual disimpaction should be necessary, the unpleasant procedure can be rendered virtually odourless if the products are ‘milked’ or scooped directly into a container of water. A large plastic cover helps restrict the permeation of the smell.

Discomfort and embarrassment are reduced by this method and by adequate premedication (e.g. IV midazolam and IV fentanyl) if large faecaliths are present.

## Colorectal cancer

### General features

- Commonest GIT malignancy: mainly adenocarcinoma
- Second most common cause of death from cancer in Western society
- Generally men over 50 years (90% of all cases)
- Mortality rate about 30% in the 5 years[9] after diagnosis
- Good prognosis if diagnosed while localised (5-year mortality 10%)
- Two-thirds in descending colon and rectum

Refer to section on genetics of colorectal cancer (see CHAPTER 23 ).

### Predisposing factors

- Ulcerative colitis (longstanding)
- Familial: familial adenomatous polyposis (FAP), hereditary non-polyposis colorectal cancer
- Colonic adenomata
- Decreased dietary fibre
- Age >50 years

### Lifetime risk

This is determined by the family history (see TABLE 31.5 ).

**Table 31.5** Family history and lifetime risk of colorectal cancer[10]

| Family history | Lifetime risk |
|---|---|
| None: population risk | 1:50 |
| One first-degree relative >45 years | 1:17 |
| One first-degree relative and one second-degree relative | 1:12 |
| One first-degree relative <45 years | 1:10 |
| Two first-degree relatives (any age) | 1:6 |
| Hereditary non-polyposis colon cancer | 1:2 |
| Familial adenomatous polyposis | 1:1 |

Consider referral to a familial cancer clinic for assessment.

## Symptoms



- Blood in the stools
- Mucus discharge
- Recent change in bowel habits (constipation more common than diarrhoea)
- Alternating constipation with spurious diarrhoea
- Bowel leakage when flatus passed
- Unsatisfactory defecation (the mass is interpreted as faeces)
- Abdominal pain (colicky) or discomfort (if obstructing)
- Rectal discomfort
- Symptoms of anaemia
- Rectal examination—this is appropriate because many cancers are found in the lowest 12 cm and most can be reached by the examining finger

### Obstruction (distension with ↑ pain)

If obstructing, there is a risk of rupture of the caecum.

Surgery is needed to circumvent the closed loop obstruction.

### Spread

- Lymphatics → epigastric and para-aortic nodes
- Direct → peritoneum
- Blood → portal circulation

Various forms of presentation of large bowel cancers are shown in FIGURE 31.3 .

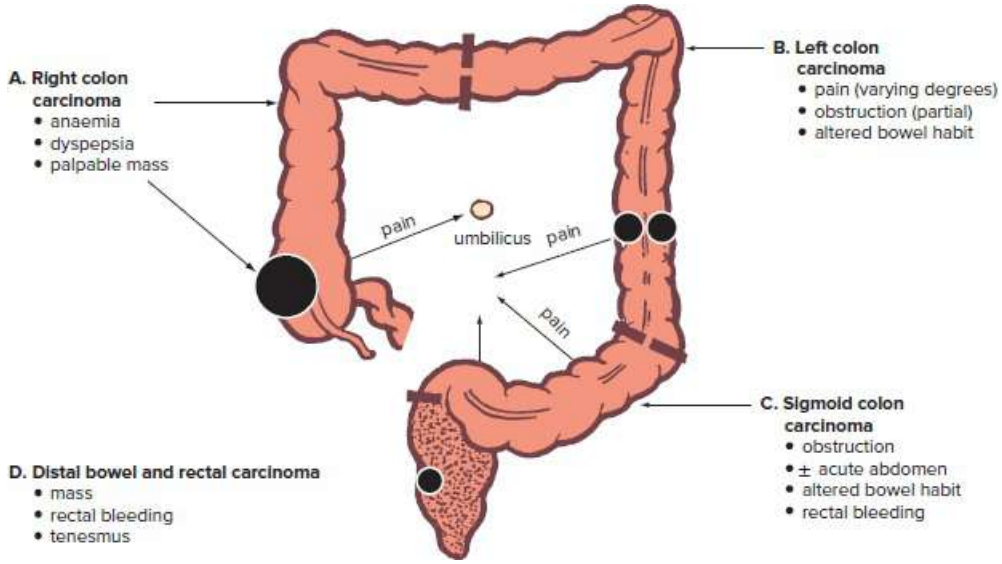


**FIGURE 31.3** Various forms of presentation of large bowel cancer

## Investigations

- FOBT: immunochemical tests (e.g. Inform and InSure) do not require dietary or medication restriction
- Colonoscopy ± biopsy
- CT colonography (investigation of choice)
- Serum CEA level is not useful for diagnosis but is useful for monitoring response to treatment
- Sigmoidoscopy, especially flexible sigmoidoscopy
- Double contrast barium enema may miss tumours and is being superseded by other imaging

- Ultrasonography and CT scanning not useful in primary diagnosis; valuable in detecting spread, especially hepatic metastases
- PET-CT scanning (if available) is useful for follow-up
- Consider defecography

If FOBT is positive—investigate by colonoscopy or by flexible sigmoidoscopy.

## Screening[11]

An FOBT every 2 years is now recommended for all people from 50–74 years (see guidelines in CHAPTER 6 ). FOBT is safer, cheaper and more convenient than colonoscopy. Do not use the CEA blood test as a screening tool.

Colonoscopy as screening is only recommended in 2% of the population, as follows:

- Moderate risk (family history category 2): 2 yearly FOBT from 40–49, then colonoscopy every five years from 50–74 years.
- High risk (family history category 3): 2 yearly FOBT from 35–44, then colonoscopy every 5 years from 45–74 years.[11]

In addition, flexible sigmoidoscopy and rectal biopsy for those with ulcerative colitis. Refer to a bowel cancer specialist to plan appropriate surveillance.



## Management

Early surgical excision is the treatment, with the method depending on the site and extent of the cancer. Dukes classification gives a guide to prognosis (see TABLE 31.6 ). The survival rates for Dukes C cancer have improved with more effective chemotherapy.

**Table 31.6** Modified Dukes classification of colorectal cancer

| Stage | Pathologic description | Approx. treated 5-year survival %* |
|---|---|---|
| A | Cancer limited to mucosa and submucosa | 95 |
| B | Cancer extends into muscularis or serosa | 75–85 |
| C | Cancer involves regional lymph nodes | 26–46 |
| D | Distant metastases (e.g. liver) | 7 |

*Percentage ranges cover several studies

*Note:* Overall survival over 70%[11]

Staging and prognosis are outlined in FIGURE 31.4 . There are other classifications for staging of colorectal cancer, including the 0, I, II, III, IV system.

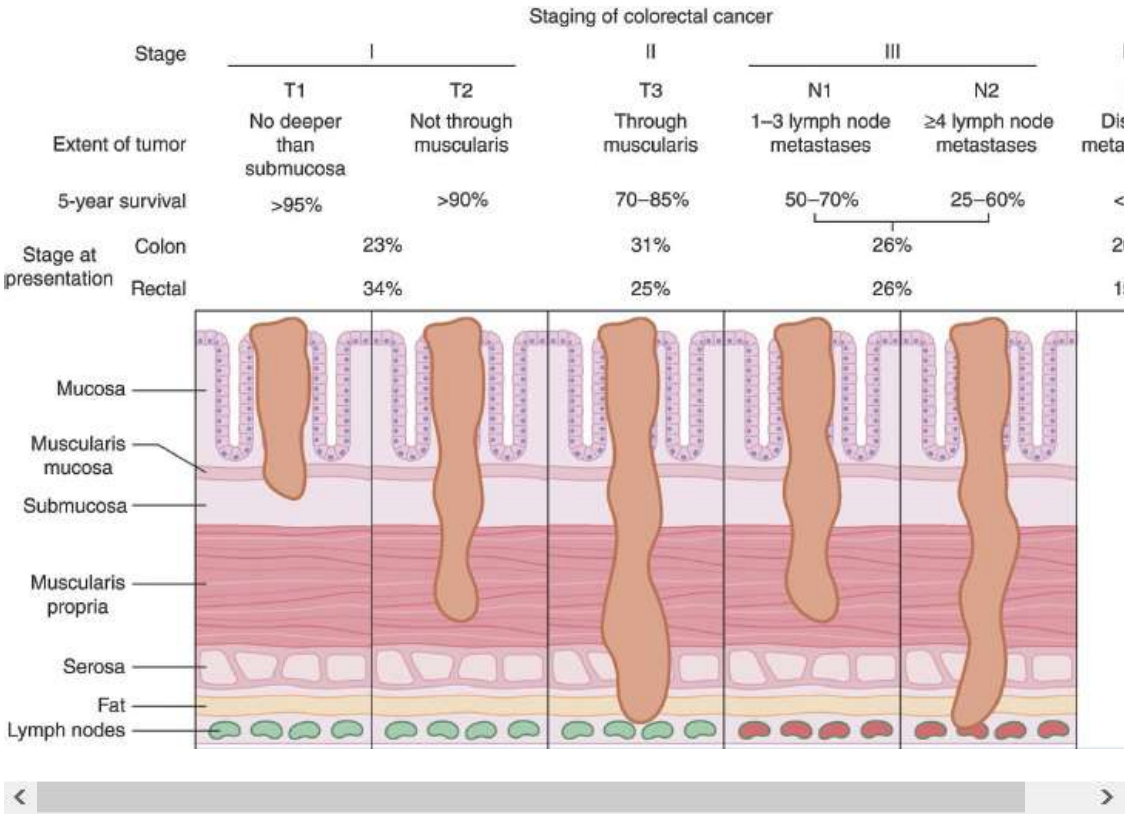


**FIGURE 31.4** Staging (TNM system) and prognosis for patients with colorectal cancer

Follow-up includes:

- CEA antigen
- colonoscopy
- abdominal imaging: ultrasound or CT scan of liver

## When to refer[4]

Patients with constipation or change in bowel habit of recent onset without obvious cause need further investigation.

Those with chronic symptoms who do not respond to simple measures should be referred.

## Practice tips

- The objectives of treatment should be to exclude organic disease and then reassure and re-educate the patient about normal bowel function.
- Discourage long-term use of laxatives, suppositories and microenemas.
- The laxatives to discourage should include anthraquinone derivatives, bisacodyl, phenolphthalein, magnesium salts, castor oil and mineral oils.
- First-line treatment of functional constipation (unresponsive to simple measures) is a bulking agent. An osmotic laxative is good second-line therapy.
- Bleeding with constipation indicates associated organic illness—exclude bowel cancer. Bright red blood usually means haemorrhoids.
- Beware of hypokalaemia causing constipation in the older person on diuretic treatment.
- If cancer can be felt on rectal examination, an abdominal perineal procedure with colostomy usually follows; if not, an anterior resection is generally the rule.

# Patient education resources

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

- Bowel cancer
- Constipation

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# 32 Cough

*I bounded into bed. The bound made me cough—I spat—it tasted strange—it was bright red blood—I don't want to find this is real consumption—I shan't have my work written. That's what matters. How unbearable it would be to die—nothing real finished.*

KATHERINE MANSFIELD (1888–1923), DIARY ENTRY 1918

Cough is one of the five most common symptoms presenting in family practice. There is a wide range of causes (see TABLE 32.1 ) with the great majority being minor and self-limiting, although the possibility of serious causes such as bronchial carcinoma should always be kept in mind. It can be non-productive (dry) or productive (with phlegm or sputum).

**Table 32.1** Significant causes of cough

**Non-productive (dry cough)**

Upper respiratory tract infection

Lower respiratory tract infection:

- viral
- some bacteria (e.g. mycoplasma)

Inhaled irritants:

- smoke, dust, fumes

Drugs

Inhaled foreign body

Bronchial neoplasm

Pleurisy

Interstitial lung disorders:

- fibrosing alveolitis
- extrinsic allergic alveolitis
- pneumoconiosis
- sarcoidosis

| |
|---|
| Tuberculosis |
| Left ventricular failure (esp. nocturnal cough) |
| Whooping cough (pertussis) |
| Gastro-oesophageal reflux and hiatus hernia |
| Chronic rhinosinusitis (and postnasal drip) |
| Obstructive sleep apnoea |

| **Productive cough** |
|---|
| Chronic bronchitis |
| Bronchiectasis |
| Pneumonia (especially bacterial) |
| Asthma |
| Foreign body (later response) |
| Bronchial carcinoma (dry or loose) |
| Lung abscess |
| Tuberculosis (when cavitating) |

Smokers often have a morning cough with little sputum. Coughing can also be initiated by pleural irritation. It is a reflex that provides an essential protective service. It serves to remove substances that may have been accidentally inhaled and removes excess secretions or exudates that may accumulate in the airway.

## Key facts and checkpoints

- Cough is the commonest manifestation of lower respiratory tract infection.
- Cough is the cardinal feature of chronic bronchitis.
- Cough is a feature of asthma with sputum production, especially at night.
- Cough can have a psychogenic basis.
- Cough may persist for many weeks following an acute upper respiratory tract infection (URTI) as a result of persisting bronchial inflammation and increased airway responsiveness.[1]
- Postnasal drip is a common cause of a persistent or chronic cough, especially causing nocturnal cough due to secretions (mainly from chronic sinusitis) tracking down the larynx and trachea during sleep.
- The commonest causes of haemoptysis are URTI (24%), acute or chronic bronchitis (17%), bronchiectasis (13%), TB (10%). Unknown causes totalled 22%

and cancer 4% (figures from a UK study).[2]

# A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 32.2 .



**Table 32.2** Cough: diagnostic strategy model

| |
|---|
| **Probability diagnosis** |
| Upper respiratory infection<br>Postnasal drip/rhinitis/sinusitis<br>Smoking<br>Acute bronchitis<br>Chronic bronchitis/COPD |
| **Serious disorders not to be missed** |
| Cardiovascular:<br>• left ventricular failure<br>Neoplasia:<br>• lung cancer<br>Severe infections:<br>• tuberculosis<br>• pneumonia<br>• influenza<br>• lung abscess<br>• HIV infection<br>• SARS (coronavirus)<br>• COVID-19 (coronavirus)<br>Asthma<br>Cystic fibrosis<br>Inhaled foreign body<br>Pneumothorax |
| **Pitfalls (often missed)** |
| Atypical pneumonias<br>Gastro-oesophageal reflux (nocturnal)<br>Smoking (children/adolescents) |

| |
|---|
| Bronchiectasis |
| Obstructive sleep apnoea |
| Whooping cough (pertussis) |
| Interstitial lung disorders |
| Sarcoidosis |
| **Seven masquerades checklist** |
| Drugs (several, e.g. ACE inhibitors) |
| **Is the patient trying to tell me something?** |
| Anxiety and habit<br>Chronic cough may become self-perpetuating due to larynx irritation |

## Probability diagnosis

The most common cause of cough is an acute respiratory infection, whether a URTI or acute bronchitis.[3] Persistent coughing with a URTI is usually due to the development of sinusitis with a postnasal drip.

Chronic bronchitis is also a common cause of cough.

## Serious disorders not to be missed

Bronchial carcinoma must not be overlooked. A worsening cough is the commonest presenting problem. A bovine cough is suggestive of cancer: the explosive nature of a normal cough is lost when laryngeal paralysis is present, usually resulting from bronchial carcinoma infiltrating the left recurrent laryngeal nerve.

Careful but tactful questioning in relation to IV drug use, sexual practice and previous blood transfusions is important. Chronic cough may be the first presentation of *Pneumocystis jiroveci* pneumonia in an HIV-infected person. Important causes of a chronic cough are summarised in TABLE 32.3 .

**Table 32.3** Some causes of chronic cough[2,4,5]

| |
|---|
| **Normal chest X-ray (includes most causes)** |
| Chronic postnasal drip* |
| Asthma* |
| Asthma + postnasal drip |
| Postinfective bronchial hyper-responsiveness |
| Gastro-oesophageal reflux:* |

- symptomatic
- asymptomatic

Chronic bronchitis
Chronic heart failure
Drugs (e.g. ACE inhibitors, beta blockers, salazopyrin)
Snoring and obstructive sleep apnoea
Irritants: occupational and household
Smoker's cough
Whooping cough (pertussis)
Habit
Functional
Idiopathic

| Abnormal chest X-ray |
| --- |
| Bronchiectasis |
| Cancer: bronchial, larynx |
| Cardiac failure |
| COPD |
| Cystic fibrosis |
| Inhaled foreign body |
| Interstitial lung disorders (e.g. sarcoidosis) |
| Tuberculosis |

*top three

The possibility of a foreign body should always be kept in mind, especially in children, and severe infections such as TB and pulmonary abscess must not be misdiagnosed.

## Red flag pointers for cough

- Age >50 years
- Smoking history
- Asbestos exposure history
- Persistent cough
- Overseas travel

- TB exposure
- Haemoptysis
- Unexplained weight loss
- Dyspnoea
- Fever

It is also important not to overlook asthma in which a nocturnal cough, without wheezing, is a feature in children.



## Pitfalls

Causes that tend to be overlooked, especially in the presence of a normal X-ray, are gastro-oesophageal reflux, postnasal drip and asthma. Gastro-oesophageal reflux is more common as a cause of reflex coughing, especially at night, than appreciated. However, do not use PPIs in the absence of suggestive GORD symptoms.[5] Whooping cough, especially immunisation-modified, can be difficult to diagnose, particularly if the characteristic whoop is absent.

### General pitfalls

- Attributing cough due to bronchial carcinoma in a smoker to ‘smoker’s cough’
- Overlooking TB, especially in the elderly, by attributing symptoms to old age, bronchitis or smoking
- Overlooking the fact that bronchial carcinoma can develop in a person with other pulmonary conditions, such as chronic bronchitis
- Being slow to order a chest X-ray
- Failing to recognise that pertussis presents in adults

## Seven masquerades checklist

The applicable masquerade is drugs, many of which can produce a wide variety of disorders of the respiratory tract that cause a cough. Pulmonary infiltration with fibrosis may result from some cytotoxic drugs, especially bleomycin. Over 20 different drugs are known to produce an SLE-like syndrome, sometimes complicated by pulmonary infiltrates and fibrosis. Cough can be a feature of some of the ACE inhibitors and beta blockers, inhaled steroids and sulfasalazine.

## Psychogenic considerations

A cough can occur for psychosocial reasons. Coughing is under cerebral control and a slight cough before commencing a speech is normal and presumably assists in clearing mucus from around the vocal cords.[6] This can readily become a nervous habit or mannerism. A typical 'psychogenic' cough is barking in quality—the 'Cape Barren goose' cough. It does not occur during sleep.

# The clinical approach

## History

The nature of the cough may provide important diagnostic clues, but it is the associated symptoms, such as the nature of the sputum, breathlessness, wheezing and constitutional symptoms, that provide the most helpful diagnostic value. A diagnosis of asthma should not be made if the predominant symptom is cough without airflow limitation such as wheeze. A history of smoking habits, past and present, is essential and an occupational and hobby history requires investigation. Significant occupations (past or present) include mining (pneumoconiosis), aircraft manufacturing (asbestosis and mesothelioma), farming ('farmer's lung'—allergic pneumonitis from mouldy hay) and bird handling ('bird fancier's lung'—allergic alveolitis or psittacosis from pigeons or budgerigars). A past history of recurrent lung infections from childhood is suggestive of cystic fibrosis and bronchiectasis, a history of hay fever and eczema suggests asthma, while a family history involves asthma, cystic fibrosis, emphysema ($\alpha_1$-antitrypsin deficiency) and tuberculosis.

### Key questions[7]

- How would you describe the cough?
- How long has the cough been present?
- Do you cough up sputum?
- Describe the sputum, especially its colour.
- Is there any blood in the sputum?
- How much sputum do you produce—a teaspoon, an eggcup or more?
- Is there a burning sensation in your throat or chest when you cough?
- Have you noticed any other symptoms?
- What about chest pain, or fever, shivers or sweats?
- Do you have a wheeze?
- Have you had previous attacks of wheezing or hay fever?

- Is there a history of asthma in your family?
- Have you lost weight?
- Has anyone in the family had TB or a persistent cough?
- How much do you smoke?
- Are you exposed to any smoke or fumes?
- What kind of work do you do, now and in the past?
- Is there a chance you have been exposed to asbestos?
- Do you keep birds or pets at home?
- Do you have any birds (e.g. pigeons) nesting outside your bedroom?
- Is there a possibility of a foreign body such as a peanut ‘having gone down the wrong way’?
- Have you had an operation recently or been confined to bed?
- Have you noticed any swelling of your legs?



## Examination

Physical examination includes a general examination with a search for features such as enlarged cervical or axillary glands, which may indicate bronchial carcinoma, as would Horner syndrome (constricted pupil, ptosis). A careful examination of the lungs and cardiovascular system is also appropriate. Fine crackles on auscultation indicate pulmonary oedema or heart failure, interstitial pulmonary fibrosis and early lobar pneumonia, while coarse crackles indicate resolving pneumonia, bronchiectasis and TB. Careful inspection of the sputum forms an important part of the physical examination of the lungs. This should include its colour and consistency, presence of particulate matter and a 24-hour sputum watch.

## Investigations

This applies particularly to those with haemoptysis. Possible investigations include:[8]

- haemoglobin, blood film and white cell count
- sputum cytology and culture
- ESR (elevated with bacterial infection, bronchiectasis, TB, lung abscess and bronchial carcinoma), CRP
- pulmonary function tests/spirometry

- radiology:

    plain chest X-ray (shows many problems)

    helical CT scan: helps more precise localisation of lesion, may show cavitation

    CT pulmonary angiogram

    bronchography: shows bronchiectasis (a very unpleasant procedure)

    ventilation/perfusion isotope scan: for pulmonary infarction

    echocardiogram (pulmonary hypertension)

- skin tests
- lung biopsy
- bronchoscopy (best at time of haemoptysis)

However, all that is needed initially is a plain chest X-ray.

## Diagnostic characteristics

There are important characteristics of cough that may point to the causation. TABLE 32.1 compares typical causes of dry and productive cough.

### Character of the cough

- Brassy → tracheitis and bronchitis (major bronchi); extrinsic pressure on trachea (e.g. tumour)
- Barking → laryngeal disorders (e.g. laryngitis)
- Croupy (with stridor) → laryngeal disorders (e.g. laryngitis, croup)
- Hollow ‘bovine’ (no power) → vocal cord paralysis (left-recurrent laryngeal nerve)
- Weak cough → indicates bronchial carcinoma
- Paroxysmal with whoops → whooping cough
- Painful → tracheitis; left ventricular failure
- Dry chronic → GORD, drugs (e.g. ACEI)

### Timing

- Nocturnal cough →

asthma

left ventricular failure

postnasal drip

chronic bronchitis

whooping cough

- Waking cough →

  bronchiectasis, asthma

  chronic bronchitis

  GORD

  habitual

### Associations

- Changing posture →

  bronchiectasis

  lung abscess

- Meals →

  hiatus hernia (possible)

  oesophageal diverticulum

  tracheo-oesophageal fistula

- Wheezing →

  asthma

- Breathlessness →

  asthma

  left ventricular failure

  COPD

## Sputum

A healthy, non-smoking individual produces approximately 100–150 mL of mucus a day. This normal bronchial secretion is swept up the airways towards the trachea by the mucociliary clearance mechanism and is usually swallowed. The removal from the trachea is assisted also by occasional coughing, although this is carried out almost subconsciously.[6]

Excess mucus is expectorated as sputum. The commonest cause of excess mucus production is cigarette smoking. Mucoid sputum is clear and white.

### Character of sputum

- Clear white (mucoid) → normal or uninfected bronchitis
- Yellow or green (purulent) → due to cellular material (neutrophils or eosinophil granulocytes)

  ± infection (not necessarily bacterial infection)

  asthma due to eosinophils

  bronchiectasis (copious quantities)
- Rusty → lobar pneumonia (*S. pneumoniae*): due to blood
- Thick and sticky → asthma
- Profuse, watery → alveolar cell carcinoma
- Thin, clear mucoid → viral infection
- Redcurrant jelly → bronchial carcinoma
- Profuse and offensive → bronchiectasis; lung abscess
- Thick plugs (cast-like) → allergic bronchopulmonary *Aspergillus;* bronchial carcinoma
- Pink frothy sputum → pulmonary oedema



## Haemoptysis

Bloodstained sputum (haemoptysis), which varies from small flecks of blood to massive bleeding, requires thorough investigation. Always consider malignancy or TB. Often the diagnosis can be made by chest X-ray. Causes are presented in TABLE 32.4 . Haemoptysis must be distinguished from blood-stained saliva caused by nasopharyngeal bleeding or sinusitis and also from haematemesis.[6] Acute bronchitis produces streaky haemoptysis.

**Table 32.4** Haemoptysis (adults): diagnostic strategy model

### Probability diagnosis

Acute chest infection:

- URTI (24%)
- bronchitis

Chronic bronchitis
Trauma: chest contusion, prolonged coughing
Cause often unknown (22%)

### Serious disorders not to be missed

Vascular:

- pulmonary infarction/embolus
- LHF → pulmonary oedema
- mitral stenosis
- pulmonary hypertension
- AV malformation

Infection:

- lobar pneumonia (rusty sputum)
- tuberculosis
- lung abscess

Cancer/tumour (4%):

- bronchogenic carcinoma
- tumour of the larynx or trachea

Other:

- blood disorders including anticoagulants
- cystic fibrosis

### Pitfalls (often missed)

Foreign body
Bronchiectasis (13%)
Iatrogenic (e.g. endotracheal intubation)
Goodpasture syndrome or other vasculitides
Spurious haemoptysis (blood from nose or throat)
Factitious (e.g. Munchausen syndrome)

*Note:* Haemoptysis must be distinguished from bloodstained saliva caused by nasopharyngeal bleeding or sinusitis, also haematemesis.[6] Copious haemoptysis is due to bronchiectasis or TB.

## Impaired cough

Weak or ineffective cough affects ability to clear lower respiratory tract secretions, predisposing to more serious infection, particularly lower lobe pneumonia. Causes include chest wall or abdominal pain/injury, chest wall deformity, decreased cough strength and central respiratory depression.

### Productive cough

- Chronic bronchitis: mucoid or purulent; rarely exceeds 250 mL per day[6]
- Bronchiectasis: purulent sputum; up to 500 mL/day
- Asthma: mucoid or purulent; tenacious sputum
- Lung abscess: purulent and foul-smelling
- Foreign body: can follow impaction

## Cough in children[3]

Cough in children is a very common symptom, but troublesome persistent cough is a great cause of anxiety among parents and probably the commonest symptom for which the family doctor is consulted. Age-related causes of chronic cough (present at least 4 weeks) are presented in TABLE 32.5 . Most children with chronic cough do NOT have asthma. A chest X-ray is advisable for a persistent cough.



**Table 32.5** Age related causes of chronic cough in children (to consider)[9]

**Infants**

- Congenital/structural abnormalities, e.g. tracheao-esophageal fistula
- Milk inhalation/reflux
- Household smoke exposure

**Toddler/preschool**

- Foreign body inhalation
- Asthma
- Viral induced wheeze
- Bronchiolitis/bronchitis
- Whooping cough
- Cystic fibrosis
- Croup

**Older children**

- Asthma
- Acute or chronic bronchitis
- Chronic rhinitis
- Smoke exposure
- Atypical pneumonia

**Adolescents**

- Asthma
- Smoking/other inhalants
- Psychogenic

Common causes of cough generally are:

- asthma
- recurrent viral bronchitis
- acute URTIs
- allergic rhinitis
- croup

Disorders not to be missed are:

- asthma
- cystic fibrosis
- inhaled foreign body
- tracheo-oesophageal fistula
- pneumonia
- whooping cough

Several clinicians describe the catarrhal child syndrome as the commonest cause of chronic cough.[3] This refers to children who develop a postnasal drip following acute respiratory infection and allergic rhinitis. Recurrent cough in children can usually be explained by recurrent viral respiratory infections which commonly occur when first exposed to other children. Their airways tend to be overactive. There is a slight predisposition to asthma.

If asthma is suspected, a therapeutic trial of salbutamol 200 mcg 4 hourly via a spacer may be

worthwhile.

### Psychogenic causes

Habit cough can occur in children, especially those with a history of school phobia. The cough does not occur during sleep and remains unchanged with exertion or infection.

## Croup (laryngotracheobronchitis)

### Clinical features

- Characteristic harsh barking inspiratory cough with stridor
- Prodrome of URTI for 2 days
- Sounds like a dog barking or a seal
- Children 6 months to 6 years
- Fever variable (rarely >39°)
- Usually 11 pm to 2 am
- Auscultation confirms inspiratory stridor
- Occurs in small local epidemics

Management—refer to CHAPTER 89 .

# Pneumonia in children

### Clinical features

- Tachypnoea, expiratory grunt
- Possible focal chest signs
- Diagnosis often only made by chest X-ray

### Pathogens

- Viruses are the most common cause in infants.
- *Mycoplasma* is common in children over 5 years.
- *S. pneumoniae* is a cause in all age groups.

- Pathogens are difficult to isolate—may need blood culture.

## Treatment

Depends on age and tropical vs non-tropical. Almost all those under 48 months should be admitted to hospital. Indicators for hospital admission are shown in the box.

- Minimal handling
- Careful observations including pulse oximetry
- Attend to hydration
- Antibiotics indicated in all cases, even though many are viral. Refer to Therapeutic Guidelines for various age groups, tropical regions and specific confirmed bacterial species. A simplified overview follows:

  Mild to moderate:

  amoxicillin 25 mg/kg up to 1 g orally, 8 hourly for 3 days (mild) or 5–7 days (moderate)

  *plus (if atypical bacteria suspected)*

  azithromycin or clarithromycin or doxycycline

  Severe:[10]

  cefotaxime IV *or* ceftriaxone IV (if *staphylococcus aureus* suspected, add clindamycin or lincomycin)

### Pneumonia in children: guidelines for hospitalisation[11]

Infants:

- RR > 70
- Intermittent apnoea
- Not feeding

Older children:

- RR > 50
- Grunting
- Signs of dehydration

Both groups:

- $S_aO_2$ ≤92%
- Cyanosis
- Difficulty breathing
- Family/social issues



# Cough in the elderly

Important causes of cough to consider in the elderly include chronic bronchitis, lung cancer, pulmonary infarct (check calves), bronchiectasis and left ventricular failure, in addition to the acute upper and lower respiratory infections to which they are prone. It is important to be surveillant for bronchial carcinoma in an older person presenting with cough, bearing in mind that the incidence rises with age. One study found the causes of chronic cough in the elderly to be postnasal drip syndrome 48%, gastro-oesophageal reflux 20% and asthma 17%.[12]

# Common respiratory infections

Respiratory infections, especially those of the upper respiratory tract, are usually regarded as trivial, but they account for an estimated one-fifth of all time lost from work and three-fifths of time lost from school, and are thus of great importance to the community.[13] The majority of respiratory infections are viral in origin and antibiotics are therefore not indicated.

URTIs are those involving the nasal airways to the larynx, while lower respiratory tract infections (LRTIs) affect the trachea downwards.

Combined URTIs and LRTIs include influenza, measles, whooping cough and laryngotracheobronchitis.

## The common cold (acute coryza)

This highly infectious URTI, which is often mistakenly referred to as ‘the flu’, produces a mild systemic upset and prominent nasal symptoms (see FIG. 32.1 ).

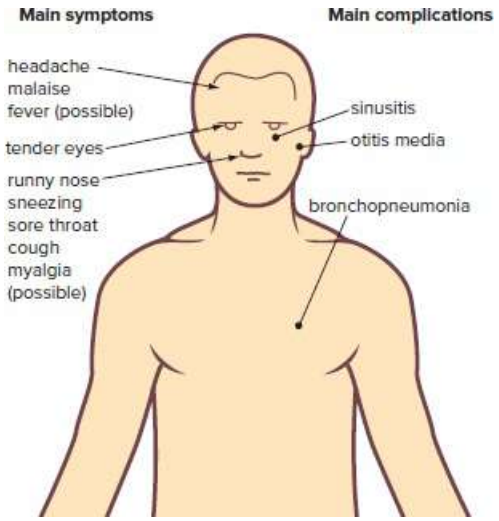


**FIGURE 32.1** The main symptoms and complications of the common cold

## Clinical features

- 24–48 hours of weakness
- Malaise and tiredness
- Sore, runny nose
- Sneezing
- Sore throat
- Slight fever

Other possible symptoms:

- headache
- hoarseness
- cough

The watery nasal discharge becomes thick and purulent in about 24 hours and persists for up to a week. Secondary bacterial infection is uncommon.

## Management

Advice to the patient includes:

- rest—adequate sleep and rest, especially if weak

- drink adequate fluids
- stop smoking (if applicable)
- analgesics—paracetamol (acetaminophen) or aspirin (max. 8 tablets a day in adults)
- steam inhalations for a blocked nose
- cough drops or syrup for a dry cough
- gargle aspirin in water or lemon juice for a sore throat (avoid aspirin in children <16 years)
- vitamin C powder or tablets (e.g. 2 g daily) does not reduce the risk of catching an URTI, but regular usage may possibly reduce duration—but only if used prophylactically before the URTI[14]
- clinical trials of zinc lozenges and echinacea have been unpromising[15,16]

## Influenza

Commonly due to influenza A or influenza B viruses, influenza causes a relatively debilitating illness and should not be confused with the common cold. The differences are presented in TABLE 32.6 . The incubation period is usually 1–3 days and the illness commences abruptly with a fever, headache, shivering and generalised muscle aching (see FIG. 32.2 ). 

**Table 32.6** Comparison of common cold and influenza

| | Common cold | Influenza |
|---|---|---|
| **Incubation period** | 12 hours to 5 days | 1–3 days |
| **Fever** | ± | + + |
| **Cough** | (later) | + |
| **Sore throat** | + + | ± |
| **Rhinitis**<br>**sneezing**<br>**rhinorrhoea** | + | ± late symptom |
| **Muscle aches** | – | + |
| **Toxaemia** | – | ± |
| **Causes** | Rhinoviruses<br>Parainfluenza<br>Influenza B, C<br>Coronavirus | Influenza A<br>Influenza B<br>Novel strains influenza A e.g. H5N1 |

*Diagnosis:* Nasal/throat swabs for viral PCR (rapid and best); viral antigen detection or culture.

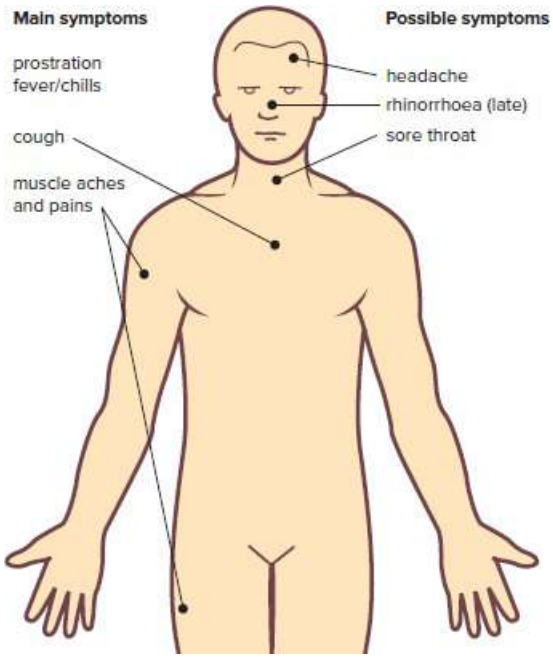


**FIGURE 32.2** The main features of influenza

## Clinical criteria[5]

During an influenza epidemic:

- fever >38°C plus at least one respiratory symptom and one systemic symptom
- cough (dry)
- sore throat
- coryza
- prostration or weakness
- myalgia
- headache
- rigors or chills

## Complications

- Tracheitis, bronchitis, bronchiolitis
- Secondary bacterial infection
- Pneumonia due to *Staphylococcus aureus* (mortality up to 20%)[1]
- Toxic cardiomyopathy with sudden death (rare)
- Encephalomyelitis (rare)
- Depression (a common sequela)

## Diagnosis

- Nasopharyingeal swabs for PCR or other rapid specific tests

## Management

Advice to the patient includes:

- rest in bed until the fever subsides and patient feels better
- analgesics: paracetamol and aspirin or ibuprofen are effective, especially for fever
- fluids: maintain high fluid intake (water and fruit juice)
- freshly squeezed lemon juice and honey preparation

### Antiviral agents[5]

- Neuraminidase inhibitors (cover influenza A and B):

  zanamivir (Relenza) 10 mg by inhalation bd for 5 days

  oseltamivir (Tamiflu) 75 mg (o) bd (child 2 mg/kg) for 5 days

Both should be commenced within 36 hours of onset and given for 5 days.

*Note:* These antiviral agents have questionable benefit in a low-risk population, but treatment for vulnerable patients during an epidemic may be appropriate.

## Prevention

Influenza vaccination for influenza A and B (recommended annually) offers some protection for up to 70% of the population for about 12 months.[1] (See CHAPTER 6 .)



# Coronavirus respiratory infections

Known for the past severe influenza syndromes MERS-CoV and SARS, it has emerged as COVID-19. This respiratory illness has had more worldwide impact than any other in the 21st century, in terms of both individual morbidity and mortality, and the subsequent socioeconomic effects. Prevention of the spread of this coronavirus is via public health measures (handwashing, social distancing, personal protective equipment) and a number of novel vaccines. At the time of writing there is no effective treatment specific to SARS-CoV-2, although various supportive interventions including medications can reduce the severity for hospitalised patients. Prevention through population vaccination is being addressed.

# Bronchitis

## Acute bronchitis

This is acute inflammation of the tracheobronchial tree that usually follows an upper respiratory infection. Although generally mild and self-limiting, it may be serious in debilitated patients.

### Clinical features

Features of acute infectious bronchitis are:

- cough and sputum (main symptoms)
- wheeze and dyspnoea
- usually viral infection
- can complicate chronic bronchitis—often due to *Haemophilus influenzae* and *Streptococcus pneumoniae*
- scattered wheeze on auscultation
- fever or haemoptysis (uncommon)

#### Outcome

- It improves spontaneously in 4–8 days in healthy patients.

### Treatment[5]

- Symptomatic treatment
- Inhaled bronchodilators for airflow limitation
- Distinguish acute bronchitis from bacterial pneumonia and bacterial infective exacerbations of COPD, where antibiotics are useful

- Antibiotics are not indicated for acute bronchitis

## Chronic bronchitis

This is a chronic productive cough for at least 3 successive months in 2 successive years:

- wheeze, progressive dyspnoea
- recurrent exacerbations with acute bronchitis
- occurs mainly in smokers

Refer to COPD (in CHAPTER 74 ).

# Pneumonia[17]

This is inflammation of lung tissue. It usually presents as an acute illness with cough, fever and purulent sputum plus physical signs and X-ray changes of consolidation. It can be broadly classified as typical or atypical, which are caused by different bacteria, viruses or other organisms.

The initial presentation of pneumonia can be misleading, especially when the patient presents with constitutional symptoms (fever, malaise and headache) rather than respiratory symptoms. A cough, although usually present, can be relatively insignificant in the total clinical picture. This diagnostic problem applies particularly to atypical pneumonia but can occur with bacterial pneumonia, especially lobar pneumonia.

## Community-acquired pneumonia[5,11]

CAP occurs in people who are not or have not been in hospital recently, and who are not institutionalised or immunocompromised, i.e. the majority of people in general practice. The choice of antibiotic is initially empirical. CAP is usually caused by a single organism, especially *Streptococcus pneumoniae*, which is demonstrating increasing antibiotic resistance.[10] Treatment is usually for 5–10 days for most bacterial causes, 2 weeks for *Mycoplasma* or *Chlamydia* infection and 2–3 weeks for *Legionella.* Viruses are often present in CAP (25–44%), whether as a sole cause or as a predecessor to bacteria.[18]

### Typical pneumonia

The commonest community-acquired infections are *Streptococcus pneumoniae* (majority), *Haemophilus influenzae*[10] (mainly in COPD), *M. pneumoniae* (young adults) and *Klebsiella pneumoniae*.

#### Clinical features

- Rapidly ill with high temperature, dry cough, pleuritic pain, rigors or night sweats
- 1–2 days later may be rusty-coloured sputum
- Rapid and shallow breathing follows
- Examination: focal chest signs, consolidation
- Investigations: CXR, sputum M&C, oxygen saturation, specific tests/serology, PCR
- Complications: pleural effusion, empyema, lung abscess, respiratory failure
- A particular complication of influenza is a *streptococcus pneumoniae* infection 2–4 weeks later[18]



## The atypical pneumonias

Refer to CHAPTER 19 .

### Clinical features

- Fever without chills, malaise
- Headache
- Minimal respiratory symptoms, non-productive cough
- Signs of consolidation absent
- Chest X-ray (diffuse infiltration) worse than chest signs

### Causes

- Virus, e.g. influenza
- *Mycoplasma pneumoniae*—the commonest:

  adolescents and young adults

  treat with:

  roxithromycin 300 mg (o) daily

  *or*

  doxycycline 100 mg bd for 14 days
- *Legionella pneumophila* (legionnaire disease):

related to cooling systems in large buildings

incubation 2–10 days

Diagnostic criteria include:

- prodromal influenza-like illness
- a dry cough, confusion or diarrhoea
- very high fever (may be relative bradycardia)
- lymphopaenia with moderate leucocytosis
- hyponatraemia

Patients can become prostrate with complications. Treat with:

azithromycin IV (first line) or erythromycin (IV or oral)

*plus* (if very severe)

ciprofloxacin or rifampicin

- *Chlamydia pneumoniae:*

  similar to *Mycoplasma*

- *Chlamydia psittaci* (psittacosis):

  treat with doxycycline, roxithromycin or erythromycin

- *Coxiella burnetti* (Q fever):

  treat with doxycycline 200 mg (o) statim, then 100 mg daily for 14 days

## Antibiotic treatment according to severity[5,13]

This is usually empirical ± 'tools' such as CORB.

### Mild pneumonia

This does not require hospitalisation.

Amoxicillin 1 g 8 hourly for 5–7 days

*plus* (if atypical pneumonia suspected, or suboptimal improvement in 2 days)

doxycycline 100 mg bd for 5–7 days

## Moderately severe pneumonia

This requires hospitalisation (see Guidelines box for severe pneumonia and hospital admission). Monitor with CXR; oximeter (keep $O_2$ saturation ≥94%).

- Neonates
- Age over 65 years
- Coexisting illness
- High temperature: >38°C
- Clinical features of severe pneumonia
- Involvement of more than one lobe
- Inability to tolerate oral therapy

  benzylpenicillin 1.2 g IV 4–6 hourly for 7 days (switch to oral amoxicillin when improved)

  *or*

  procaine penicillin 1.5 g IM daily (drugs of choice for *S. pneumoniae*) plus doxycycline

  *or*

  ceftriaxone 1 g IV daily for 7 days (in penicillin-allergic patient)

- If not so severe and oral medication tolerated, can use amoxicillin/clavulanate or cefaclor or doxycycline
- In tropical regions use a different regimen (refer to Therapeutic Guidelines)
- If atypical pneumonia, use doxycycline, erythromycin, roxithromycin or clarithromycin

## Severe pneumonia

The criteria for severity (with increased risk of death) are presented in the box on Guidelines for severe pneumonia and hospital admission.[11,17] The CORB score indicates severity (Confusion, Hypoxia $pO_2$<90%, Respiratory rate ≥30/min, BP <90/60 mmHg, Age ≥65).[19]

cefotaxime 1 g IV 8 hourly

*or*

ceftriaxone 1 g IV daily

*plus*

azithromycin 500 mg IV daily (covers *Mycoplasma, Chlamydia* and *Legionella*)

*add*

flucloxacillin for *Staphylococcus aureus*



## Guidelines for severe pneumonia and hospital admission in adults: the red flags

- Altered mental state/acute onset confusion
- Rapidly deteriorating course
- Respiratory rate >30 per minute
- Pulse rate >100 per minute
- BP <90/60 mmHg
- CORB ≥2
- Hypoxia $P_aO_2$ <60 mmHg or $O_2$ saturation <92%
- Leucocytes <4 × $10^8$L or >20 × $10^9$/L
- Multilobular involvement on CXR

# Chronic persistent cough

A cough associated with a viral respiratory infection should last no more than 2 weeks. If it does, it is termed *persistent.* A cough lasting 2 months or more is defined as a chronic cough. A cough that lasts longer than 3–4 weeks requires scrutiny. TABLE 32.3 includes some causes of chronic cough.

A chronic cough can be divided into productive and non-productive. The presence of purulent sputum increases the probability of a bacterial infection in the bronchi and/or sinuses.[4] The main organisms are *Haemophilus influenzae* (the most common), *S. pneumoniae* and *Moraxella.* Such infections are most susceptible to amoxicillin or amoxicillin/clavulanate or parenteral cephalosporins.

## Non-productive cough

Some of the many causes of a non-productive cough are included in TABLE 32.1 and more than one may be operative simultaneously; for example, an allergic snorer with oesophageal reflux taking an ACE inhibitor for hypertension may have a viral respiratory infection.[4] It has been shown that a non-productive or irritating cough is usually caused by persistent stimulation of irritant receptors in the trachea and major bronchi, and may result in the production of small amounts of mucoid sputum.

Investigations to be considered in intractable chronic cough include a chest X-ray, spirometry, CT scan of the thorax (searching in particular for a tumour) and ambulatory oesophageal pH monitoring.

If symptoms suggest possible asthma, a trial of inhaled corticosteroids can be used for 2–4 weeks.[5]

### Gastro-oesophageal reflux

This common condition can cause a persistent, non-productive cough in an apparently well person with a history of reflux. In the absence of evidence of aspiration, the cough is considered to be due to stimulation of a distal oesophageal-tracheobronchial reflex. Other studies have established a relationship between bronchial asthma and reflux or swallowing disorders whereby microaspiration can initiate an inflammatory response in the airways.

If reflux is proven or suspected, there is good evidence for diet and weight loss (if achieved) improving the cough, but unfortunately no trial evidence supporting PPIs when used in isolation for GORD-related cough.[20] However, it is reasonable to trial a PPI for 8–12 weeks, along with dietary advice.

For those with idiopathic chronic cough who remain well with no other health concerns, do not dismiss them as ‘just a cough’. Encourage them to avoid cold air, smoke and other environmental triggers. Discourage habitual throat clearing and voice overuse—consider referral to a speech pathologist or ‘cough clinic’.

## Bronchial carcinoma

Lung cancer accounts for 25% of cancer deaths in men and 24% of cancer deaths in women (rapidly rising), with cigarette smoking being the most common cause of lung cancer in both sexes.[13] It is also the most common lethal cancer in both sexes in Australia. Bronchial carcinoma accounts for over 95% of primary lung malignancies. The prognosis is poor—the 5-year overall survival is 17%.[21] The mesothelioma incidence continues to rise.

### Clinical features

- Most present between 50 and 70 years (mean 67 years)
- Nearly all (>90%) are already symptomatic at the time of diagnosis[22]

- If symptomatic—usually advanced and not resectable

### Local symptoms

- Cough (early) (42%)
- Chest pain (22%)
- Wheeze (15%)
- Haemoptysis (7%)
- Dyspnoea (5%)

### General

- Anorexia, malaise
- Weight loss—unexplained



### Others

- Unresolved chest infection
- Hoarseness
- Symptoms from metastases

The possible physical findings are summarised in FIGURE 32.3.

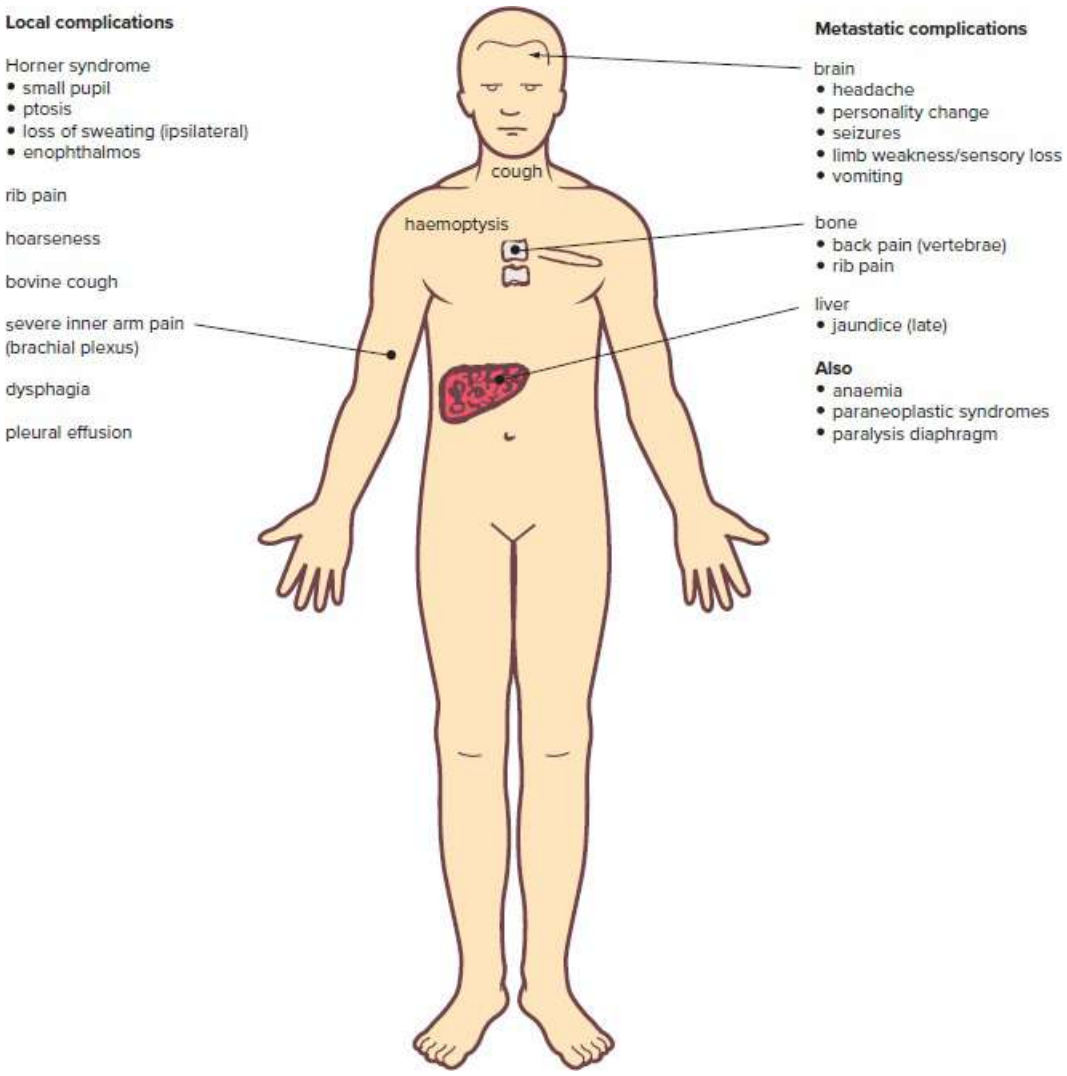


**FIGURE 32.3** Possible physical findings of bronchial carcinoma

## Investigations

- Chest X-ray
- Sputum cytology
- CT scanning
- Fibre-optic bronchoscopy
- PET scanning
- Fluorescence bronchoscopy (helps early detection)

- Tissue diagnosis where possible

*Note:* There is no current recommendation to screen asymptomatic people for lung cancer by any modality, including CXR or low-dose CT chest.[23]

Causes of a solitary pulmonary nodule on X-ray are presented in TABLE 32.7 .



**Table 32.7** Causes of a solitary pulmonary nodule (on X-ray)[24]

| |
|---|
| **Common** |
| Bronchial carcinoma |
| Secondary tumour |
| Solitary metastasis |
| Granuloma (e.g. TB) |
| Hamartoma |
| **Less common** |
| Bronchial adenoma |
| Foreign body |
| AVM |
| Hydatid |
| Others (e.g. haematoma, cyst, carotid tumour) |

### Management

Refer to a respiratory physician to determine the type of cancer. They are usually classified as small cell lung (oat cell) poorly differentiated cancer (about 15% incidence) (SCLC) and non-small cell lung cancer (NSCLC), which includes squamous cell carcinoma, adenocarcinoma and large cell carcinoma (approximately 20–30% of each). The main aim of management is a curative resection of NSCLC in those who can benefit from it. Surgery is not an option for SCLC since it metastasises so rapidly (80% have metastasised at the time of diagnosis).[11] Chemotherapy is suitable for the deadly SCLC but currently only extends life expectancy by a few months.[25] Chemotherapy has an important place in treating NSCLC. The main role of radiotherapy is palliative.

## Mesothelioma

Mesothelioma is a malignant tumour of mesothelial cells usually at the pleura. It is associated with prior asbestos exposure, possibly decades earlier (90% report exposure).

Clinical features include chest pain, dyspnoea, weight loss and recurrent pleural effusions. Diagnosis is based on imaging and on histology after pleural biopsy. Prognosis is poor and treatment is palliative support.

# Bronchiectasis

Bronchiectasis is dilatation of the bronchi when their walls become inflamed, thickened and irreversibly damaged, usually after obstruction followed by infection. Predisposing causes include whooping cough, measles, TB, inhaled foreign body (e.g. peanuts in children), bronchial carcinoma, cystic fibrosis and congenital ciliary dysfunction (Kartagener syndrome). Suspect immune deficiency in these patients. The left lower lobe and lingula are the commonest sites for localised disease. In children, early intervention saves bronchi; refer urgently if suspected.

## Clinical features

- Chronic loose cough—worse on waking
- Mild cases: yellow or green sputum only after infection
- Advanced:

    profuse purulent offensive sputum

    persistent halitosis

    recurrent febrile episodes

    malaise, weight loss
- Episodes of pneumonia
- Sputum production related to posture
- Haemoptysis (bloodstained sputum or massive) possible

## Examination

- Clubbing
- Coarse crackles over infected areas (usually lung base)
- Other respiratory signs are given in TABLE 38.6 (see CHAPTER 38 )

## Investigations

- Chest X-ray (normal or bronchial changes)
- Sputum examination: for resistant pathogens and to exclude TB

- Cytology: to rule out neoplasia
- Main pathogens: *Haemophilus influenzae* (commonest), *Streptococcus pneumoniae, Pseudomonas aeruginosa, Staphylococcus aureus*
- CT scan: can show bronchial wall thickening—high-resolution CT scan is the gold standard for diagnosis
- Spirometry
- Bronchograms: very unpleasant and used only if diagnosis in doubt or possible localised disease amenable to surgery (rare)

### Management

- Explanation and preventive advice. Avoid URTIs, smoking and smoke-filled rooms.
- Physiotherapy and exercise program.
- Postural drainage (e.g. lie over side of bed with head and thorax down for 10–20 minutes three times a day).
- Antibiotics for acute exacerbations (increased cough and sputum volume/purulence) according to organism—it is important to eradicate infection to halt the progress of the disease. Amoxicillin 500 mg (o) tds for 14 days or doxycycline 200 mg (o) daily (if child ≥8 years) is recommended for first presentation. Long-term antibiotic therapy should be guided by respiratory specialists.
- Bronchodilators, if evidence of bronchospasm.[26]



## Tuberculosis

Although cough is a feature, pulmonary TB may be symptomless and detected by X-ray screenings.[8] (Refer to CHAPTER 19 .)

## Symptomatic treatment of cough[5]

Regardless of whether the underlying cause of the cough is being treated or has no treatment (e.g. viral URTI), the symptom of coughing can be distressing at all ages, and GPs are frequently asked to discuss symptomatic relief. Frustratingly, all the many over-the-counter cough remedies have either scant or no evidence for efficacy. These include antihistamines, decongestants, expectorants (e.g. senega with ammonia) and suppressants such as codeine. Cough medicines are generally not recommended in children.

Inhaled asthma medications (LABAs, corticosteroids or MART therapy) only work if there is

underlying hyper-responsiveness. Honey has some evidence, particularly in children, and is safe and easily available. Offer advice around environmental triggers—smoke, dust, pollen, cold air.

## When to refer

- Patients in whom bronchoscopy is necessary to exclude bronchial carcinoma
- Persistent hoarseness in a patient who requires expert laryngeal examination
- Evidence of pulmonary TB

### Practice tips

- Unexplained cough over the age of 50 is bronchial carcinoma until proved otherwise (especially if there is a history of smoking).
- Consider TB in the presence of an unusual cough ± wheezing.
- Bronchoscopy is essential to exclude adequately a suspicion of bronchial carcinoma when the chest X-ray is normal.
- Bright red haemoptysis in a young person may be the initial symptom of pulmonary TB.
- Avoid settling for a diagnosis of bronchitis as an explanation of haemoptysis until bronchial carcinoma has been excluded.
- Coughing may be so severe that it terminates in vomiting or loss of consciousness (post-tussive syncope).
- Large haemoptyses are usually due to bronchiectasis or TB.
- The presence of white cells in the sputum renders it yellow or green (purulent) but does not necessarily imply infection.

## Patient education resources

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

- Common cold
- Croup
- Influenza

- Pneumonia
- Bronchiectasis

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# 33 Deafness and hearing loss

*There are two kinds of deafness. One is due to wax and is curable; the other is not due to wax and is not curable.*

SIR WILLIAM WILDE (1815–1876)

Deafness is defined as impairment of hearing, regardless of its severity.[1] It is a major community health problem requiring a high index of suspicion for diagnosis, especially in children. Deafness may be conductive, sensorineural or a combination of both (mixed).

## Key facts and checkpoints

- Deafness occurs at all ages but is more common in the elderly (see FIG. 33.1 ). Fifty per cent of people over 80 years have deafness severe enough to be helped by a hearing aid.
- The threshold of normal hearing is from 0–20 decibels (dB), about the loudness of a soft whisper.
- 60 dB is the level of normal conversation or a sewing machine.
- One in seven of the adult population suffers from some degree of significant hearing impairment (over 20 dB in the better-hearing ear).[2]
- One child in every 1000 is born with a significant hearing loss. The earlier it is detected and treated, the better.
- Degrees of hearing impairment with vocal equivalent:[2,3]
  - mild = loss of hearing at 20–40 dB (soft-spoken voice is 20 dB)
  - moderate = loss at 40–60 dB (normal voice)
  - severe = loss at 70–90 dB (loud spoken voice)
  -

profound = loss at over 90 dB (shout is 90–120 dB)

- More women than men have a hearing loss.
- People who have worked with high noise levels (>85 dB) are more than twice as likely to be deaf.
- There is a related incidence of tinnitus with deafness.

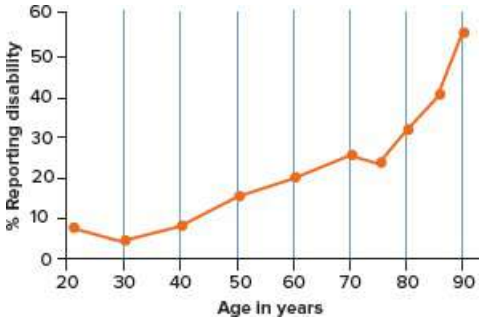


**FIGURE 33.1** Prevalence of hearing problems with increasing age

# A diagnostic approach

It is useful to consider the causes of deafness in terms of pathophysiology (conductive or sensorineural hearing loss) and anatomical sites (see FIG. 33.2 ).

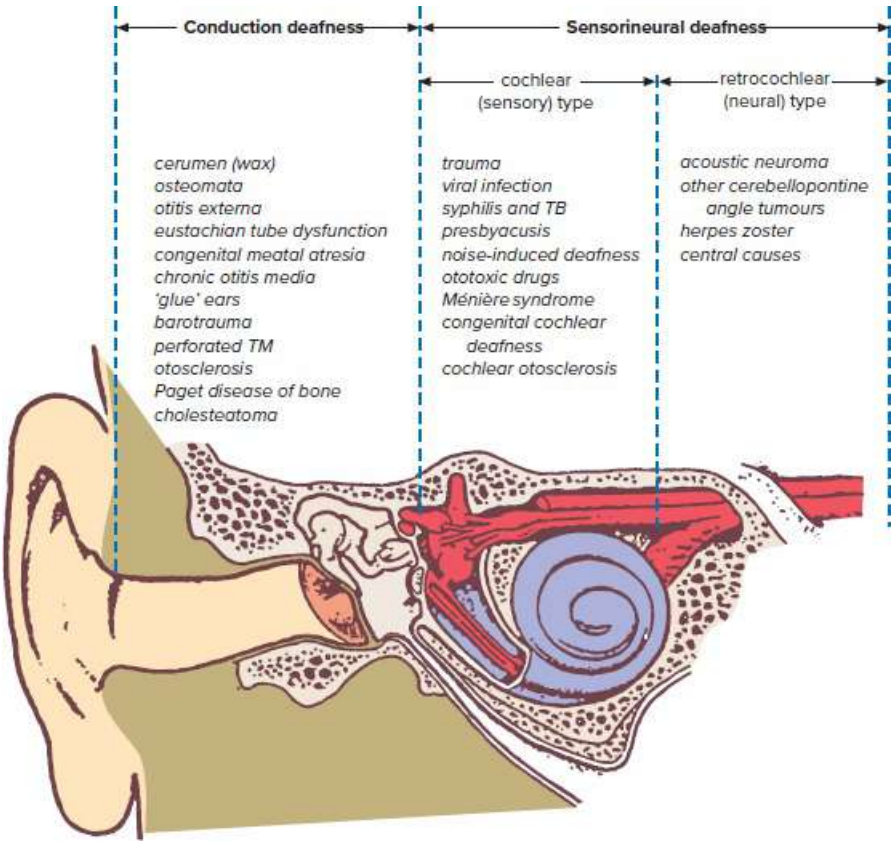


**FIGURE 33.2** Causes of deafness according to anatomical site

Conductive hearing loss is caused by an abnormality in the pathway conducting sound waves from the outer ear to the inner ear,[1] as far as the footplate of the stapes.

Sensorineural hearing loss (SNHL) is a defect central to the oval window involving the cochlea (sensor), cochlear nerve (neural) or, more rarely, central neural pathways.[1] Mixed hearing loss occurs most commonly with severe head injury or chronic infection.

Congenital deafness is an important consideration in children, while presbycusis is very common in the aged. The commonest acquired causes of deafness are impacted cerumen (wax), serous otitis media and otitis externa. Noise-induced deafness is also common.

It is important not to misdiagnose an acoustic neuroma, which can present as acute deafness, although slow progressive loss is more typical. A summary of the diagnostic strategy model, which includes several important causes of deafness, is presented in TABLE 33.1 and a checklist of ototoxic drugs in TABLE 33.2 .



**Table 33.1** Deafness and hearing loss: diagnostic strategy model

**Probability diagnosis**

Impacted cerumen
Serous otitis media
Otitis externa
Congenital (children)
Presbycusis

**Serious disorders not to be missed**

Neoplasia:

- acoustic neuroma
- temporal lobe tumours (bilateral)
- otic tumours
- nasopharyngeal carcinoma

Infection:

- generalised infections (e.g. mumps, measles)
- meningitis
- syphilis

Perforated tympanic membrane
Cholesteatoma
Perilymphatic fistula (post-stapedectomy)
Ménière syndrome

**Pitfalls (often missed)**

Foreign body
Temporal bone fracture
Otosclerosis
Barotrauma
Noise-induced deafness
*Rarities:*

- Paget disease of bone
- multiple sclerosis
- osteogenesis imperfecta

**Seven masquerades checklist**

Diabetes
Drugs
Thyroid disorder (hypo)

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

Unlikely.

**Table 33.2** Known ototoxic drugs

Alcohol

Aminoglycosides:

- amikacin
- gentamicin
- kanamycin
- neomycin
- streptomycin
- tobramycin

Diuretics:

- ethacrynic acid
- frusemide

Chemotherapeutic agents

Quinine and related drugs

Salicylates/aspirin excess

### Symptoms

The symptoms vary so that some barely notice a problem while others are severely disabled.

Common symptoms include inability to:

- hear speech and other sounds loudly enough
- hear speech and music clearly, even when loud enough
- understand speech even when loud enough—a problem of language reception

People with mild hearing loss notice only subtle differences and may have trouble hearing certain high-frequency sounds, such as ‘s’, ‘f’ or ‘th’. They may also have trouble hearing in certain situations, such as at a party or in a crowd where there is a lot of background noise. Those with moderate hearing loss have trouble hearing in many situations.

## The clinical approach

### History

The history should include an account of the onset and progression of any deafness, noise exposure, drug history, a history of swimming or diving, air travel, head injury and family history. A recent or past episode of a generalised infection would be relevant and the presence of associated aural symptoms, such as ear pain, discharge, tinnitus and vertigo. Vertigo may be a symptom of Ménière syndrome, multiple sclerosis, acoustic neuroma or syphilis.

Several important clues can be obtained from the history. The often sudden onset of hearing loss in an ear following swimming or showering is suggestive of wax, which swells to block the ear canal completely.

### Red flags

- Unilateral sensorineural hearing loss
- Cranial nerve abnormalities (other than hearing loss)

Patients with conductive loss may hear better in noisy conditions (paracusis) because we raise our voices when there is background noise. Conversely, people with sensorineural deafness (SND) usually have more difficulty hearing in noise as voices become unintelligible.

## Examination

Inspect the facial structures, skull and ears. The ears are inspected with an otoscope to visualise the external meatus, the tympanic membrane (TM) and the presence of obstructions such as wax, inflammation or osteomata.

The examination requires a clean external auditory canal. Gentle suction is useful for cleaning pus debris. Syringing is reserved for wax in people with an intact TM and a known healthy middle ear.

It is an advantage to have a pneumatic attachment to test drum mobility. Reduction of TM mobility is an important sign in secretory otitis media.

There are several simple hearing tests. The distance at which a ticking watch can be heard can be used but the advent of the digital watch has affected this traditional method.

### Whisper test

Occlude far ear. Have the patient cover near eye with one hand to prevent lip reading. Place your mouth at the near side. Strongly whisper ‘68’ then ‘100’ from a distance of 50 cm. Ask the patient to repeat the words. If not heard, repeat using a normal speaking voice.

### Hair-rubbing method

In children and in adults with a reasonable amount of hair, grab several hairs close to the external auditory canal between the thumb and index finger. Rub the hairs lightly together at 5 cm (high sensitivity) to produce a relatively high-pitched ‘crackling’ sound (see FIG. 33.3 ). If this sound cannot be heard, a moderate hearing loss is likely (usually about 40 dB or greater). Like the whisper test, this test is a rough guide only.

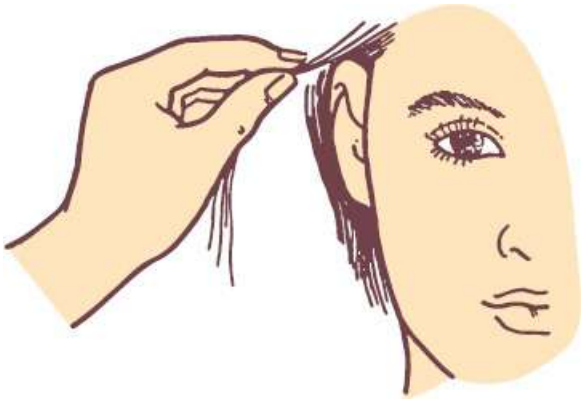

**FIGURE 33.3** Simple hair-rubbing method of testing possible deafness

## Tuning fork tests

If deafness is present, its type (conduction or sensorineural) should be determined by tuning fork testing. The most suitable tuning fork for preliminary testing is the $C_2$ (512 cps) fork. The fork is best activated by striking it firmly on the bent elbow.



### Weber test

The vibrating tuning fork is applied firmly to the midpoint of the skull or to the central forehead or to the teeth.

This test is of value only if the deafness is unilateral or bilateral and unequal (see FIG. 33.4 ). Normally the sound is heard equally in both ears in the centre of the forehead. With sensorineural deafness the sound is heard in the normal ear, while with conduction deafness it is heard better in the abnormal ear.


**FIGURE 33.4** Weber test

Lateralisation of the sound to one ear indicates a conductive loss on that side, or a sensorineural loss on the other side.

### Rinne test

The tuning fork (512 or 256 Hz) is held:

- outside the ear (tests air conduction) and
- firmly against the mastoid bone (tests bone conduction)

Ask which is louder.

It therefore compares air and bone conduction in the same ear (see FIG. 33.5 ). A more commonly used variation of the test includes placing the tuning fork on the mastoid process and the patient indicates when it can no longer be heard. The fork is then placed at the external auditory meatus and the patient indicates whether the sound is now audible. Normally air conduction is better than bone conduction and the sound will again be heard.

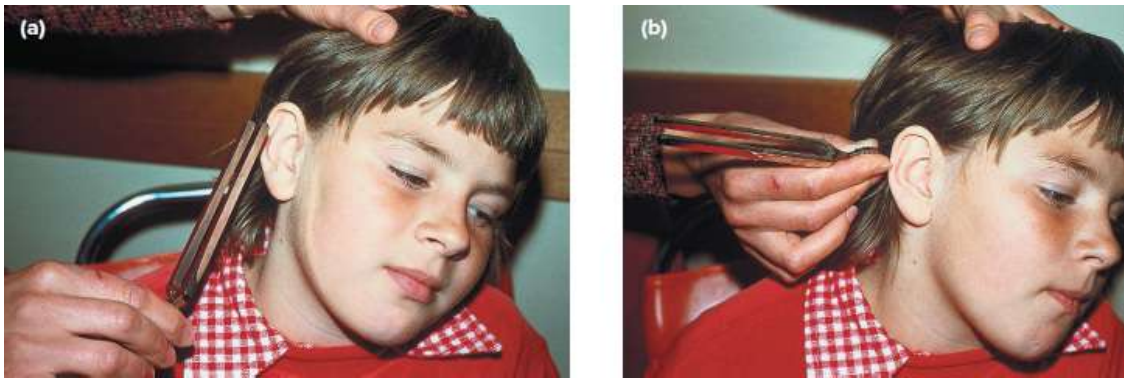

**FIGURE 33.5** Rinne test comparing air conduction **(a)** with bone conduction **(b)**

A comparison of the interpretation of these tests is summarised in TABLE 33.3 .

**Table 33.3** A comparison of the Rinne and Weber tests

| State of the hearing | Rinne test | Weber test |
|---|---|---|
| Normal | Positive: AC > BC | Equal in both ears |
| Conduction deafness | Negative: BC > AC | Louder in the deaf ear |
| Very severe conduction deafness | Negative: BC > AC<br>May hear BC only | Louder in the deaf ear |
| Sensorineural deafness | Positive: AC > BC | Louder in the better ear |
| Very severe sensorineural deafness | 'False' negative (without masking) | Louder in the better ear |

*AC = air conduction; BC = bone conduction*

## Audiometric assessment

Audiometric assessment includes the following:

- pure tone audiometry
- impedance tympanometry
- electric response audiometry
- oto-acoustic emission testing



### Pure tone audiometry[4,5]

Pure tone audiometry is a graph of frequency expressed in hertz versus loudness expressed in decibels. The tone is presented either through the ear canal (a test of the conduction and the cochlear function of the ear) or through the bone (a test of cochlear function).

FIGURES 33.6 and 33.7 are typical examples of pure tone audiograms.

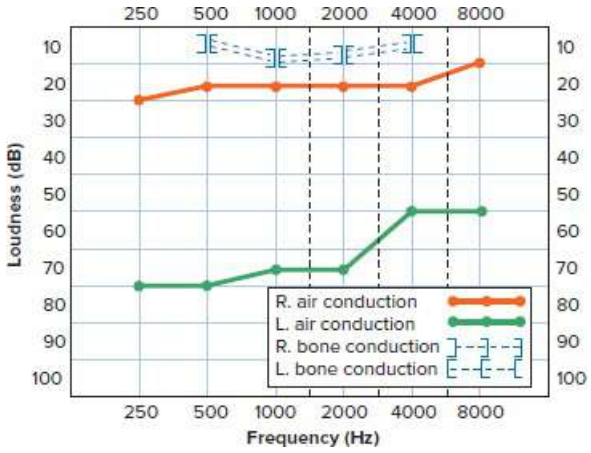


**FIGURE 33.6** Pure tone audiogram for severe conductive deafness in left ear

*Source:* Black[4]

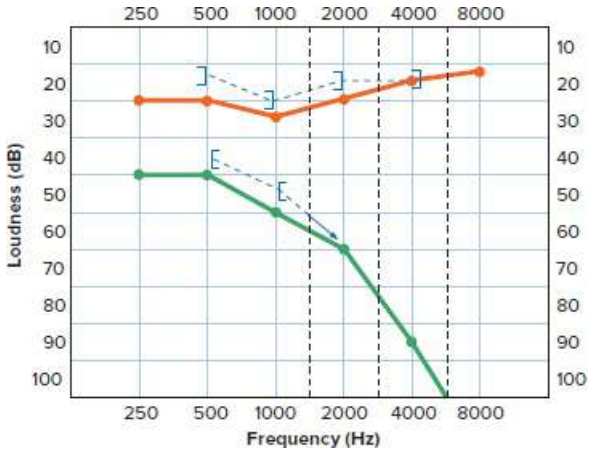


**FIGURE 33.7** Pure tone audiogram for unilateral (left) sensorineural deafness. Suspect a viral or congenital origin in children; check adults for acoustic neuroma.

The difference between the two is a measure of conductance. If the two ears have different thresholds, a white noise masking sound is applied to the better ear to prevent it hearing sound presented to the test ear. The normal speech range occurs between 0 and 20 dB in soundproof conditions across the frequency spectrum.

## Tympanometry

Tympanometry measures the mobility of the tympanic membrane, the dynamics of the ossicular chain and the middle-ear air cushion. The test consists of a sound applied at the external auditory meatus, otherwise sealed by the soft probe tip.

### Imaging

CT and gadolinium-enhanced MRI can identify retrocochlear pathology such as acoustic neuroma and cochlear nerve agenesis.

# Deafness in children

Deafness in childhood is relatively common and often goes unrecognised. One to two of every 1000 newborn infants suffer from sensorineural deafness.[1] Congenital deafness may be due to inherited defects, to prenatal factors such as maternal intra-uterine infection or drug ingestion during pregnancy, or to perinatal factors such as birth trauma, and haemolytic disease of the newborn.

Deafness may be associated with Down syndrome and Waardenburg syndrome. Waardenburg syndrome, which is dominantly inherited, is diagnosed in a person with a white forelock of hair and different coloured eyes.

Acquired deafness accounts for approximately half of all childhood cases. Purulent otitis media and secretory otitis media are common causes of temporary conductive deafness. However, one in 10 children will have persistent middle-ear effusions and mild to moderate hearing loss in the 15–40 dB range.[6]

Permanent deafness in the first few years of life may be due to virus infections, such as mumps or meningitis, ototoxic antibiotics and several other causes.

## Screening[1]

Screening should begin at birth so that language input can allow optimal language development. The aim of screening should be to recognise every deaf child by the age of 8 months to 1 year—before the vital time for learning speech is wasted. High-risk groups should be identified and screened; for example, a family history of deafness, maternal problems of pregnancy, perinatal problems, survivors of intensive care, very low birthweight and gestation <33 weeks, cerebral palsy and those with delayed or faulty speech. The guidelines for early signs of normal hearing are presented in TABLE 33.4 .



**Table 33.4** Early signs of normal hearing

| Age | Typical response |
|---|---|
| 1 month | Should notice sudden constant sounds (e.g. car motor, vacuum cleaner) by pausing and listening. |
| 3 months | Should respond to loud noise (e.g. will stop crying when hands are clapped). |
| 4 months | Should turn head to look for source of sound, such as mother |

| | |
|---|---|
| | speaking behind the child. |
| 7 months | Should turn instantly to voices or even to quiet noises made across the room. |
| 10 months | Should listen out for familiar everyday sounds. |
| 12 months | Should show some response to familiar words and commands, including his or her name. |

Optimal screening times:

- 8–9 months (or earlier)
- school entry

Newborn hearing screening measures the 8th cranial nerves' responses to sound, and is widely available and encouraged in Australia. Screening has improved the average age of detection of deafness from 20 months in 1989 to 0.8 months in 2014.[7]

## Early signs of hearing loss

A high index of suspicion is essential in detecting hearing loss in children and any parental concern should be taken seriously. The presentation of hearing loss will depend on whether it is bilateral or unilateral, its severity and age of onset.

Typical presentations include:

- malformation of skull, ears or face
- failure to respond in an expected way to sounds, especially one's voice
- preference for, or response only to, loud sounds
- no response to normal conversation or to television
- speech abnormality or delay
- absence of 'babbling' by 12 months
- no single words or comprehension of simple words by 18 months
- learning problems at school
- disobedience
- other behavioural problems

- inability to detect sound direction (unilateral loss)
- inability to follow simple commands or using less than 20 spoken words by 2 years

### Screening methods

Hearing can be tested at any age. No child is too young to be tested and this includes the newborn. Informal office assessments, such as whispering in the child's ear or rattling car keys, are totally inadequate for excluding deafness and may be potentially harmful if they lead to false reassurance.

Pneumatic otoscopy is essential to exclude middle-ear effusions.

Pure tone audiometry is unreliable in children under 4 years of age, so special techniques such as tympanometry are required. Tympanometry assesses TM compliance, and is highly sensitive and specific for detecting middle-ear pathology in children beyond early infancy.

Neonates and infants can be tested using Automated Auditory Brainstem Response (AABR) or Transient Evoked Otoacoustic Emissions (TEOAE).

### Management

Children with middle-ear pathology and hearing loss should be referred to a specialist. All children with sensorineural hearing loss (even those with profound deafness), as well as children with conductive losses not correctable by surgery, benefit from amplification. All children need referral to a specialist centre skilled in educational and language remediation.

## Deafness in the elderly[8]

The prevalence of hearing loss increases exponentially with age. The commonest reason for bilateral progressive sensorineural deafness is presbycusis, which is the high-frequency hearing loss of advancing age (see FIG. 33.8 ). There appears to be a genetic predisposition to presbycusis.[8]

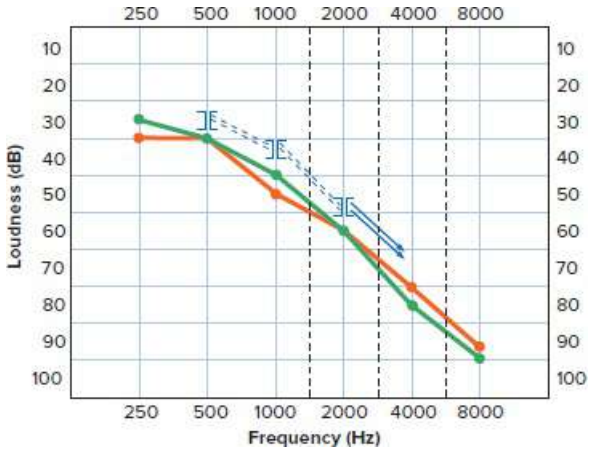


**FIGURE 33.8** Presbycusis: bilateral high-frequency sensorineural deafness



## Presbycusis

Presbycusis is sensorineural hearing loss related to deterioration of hearing with ageing. Some features include:

- loss of high-frequency sounds
- usually associated with tinnitus
- intolerance to very loud sounds
- difficulty picking up high-frequency consonants, e.g. ‘f’, ‘s’—these sounds are often distorted or unheard, and there is confusion with words such as ‘fit’ and ‘sit’, ‘fun’ and ‘sun’

Deafness is associated with various types of mental illness in the aged, including anxiety, depression, paranoid delusions, agitation and confusion because of sensory deprivation. The possibility of deafness should be kept in mind when assessing these problems.

## Signs indicating referral for hearing test

Possible indications for referring the older person:

- speaking too loudly
- difficulty understanding speech
- social withdrawal
- lack of interest in attending parties and other functions

- complaints about people mumbling
- requests to have speech repeated
- complaints of tinnitus
- setting television and radio on high volume

# Sudden deafness

Sudden deafness refers to a sudden sensorineural hearing loss threshold of greater than 30–35 dB with an onset period of between 12 hours and 3 days.[9] It specifically excludes gradual progressive causes of sensorineural deafness, such as cumulative noise trauma or presbycusis, and also excludes causes of sudden deafness that may be related to pathology in the external auditory canal, TM or middle ear.

The main causes are given in TABLE 33.5 .

**Table 33.5** Causes of sudden deafness

| |
|---|
| Trauma:<br>• head injury/blunt head trauma<br>• diving<br>• flying<br>• acoustic blast |
| Postoperative:<br>• previous stapedectomy |
| Viral infections (e.g. mumps, measles, herpes zoster) |
| Ototoxic drugs (e.g. aminoglycosides, gentamicin) |
| Cerebellopontine angle tumours (e.g. acoustic neuroma) |
| Vascular disease:<br>• polycythaemia<br>• diabetes<br>• stroke, especially cerebellar<br>• vasculitis |
| Ménière syndrome |
| Cochlear otosclerosis |

In several instances, despite a careful clinical examination and investigation, an explanation for sudden sensorineural deafness cannot be found and it is considered to be idiopathic, which

accounts for most cases. The cause of deafness in these cases is thought to be either vascular obstruction of the end artery system or viral cochleitis.[8,10] Fortunately, spontaneous recovery usually results.

Patients with sudden sensorineural deafness require immediate referral. It is a difficult problem both in diagnosis and management. Early diagnosis and a high index of suspicion are fundamental.[8] Two important conditions that deserve special reference are perilymphatic fistula, which occurs after stapedectomy, and an acoustic neuroma presumably causing compression of the internal auditory artery by the tumour in the internal auditory meatus.

Investigations: FBE, ESR, ANCA; TB ELISpot; viral titres; evoked response audiometry; MRI.

# Otosclerosis

Otosclerosis is a disease of the bone surrounding the inner ear and is the most common cause of conductive hearing loss in the adult with a normal tympanic membrane. The normal middle-ear bone is replaced by vascular, spongy bone that becomes sclerotic.[11]

## Clinical features[3]

Usually:

- a progressive disease
- develops in the 20s and 30s
- family history (autosomal dominant)
- bilateral or unilateral
- female preponderance
- affects the footplate of the stapes
- may progress rapidly during pregnancy
- conductive hearing loss
- begins in lower frequencies, then progresses



- impedance audiometry shows characteristic features of conductive loss with a mild sensorineural loss
- may be associated with Ménière syndrome

## Management

- Referral to an ENT specialist
- Stapedectomy (approximately 90% effective)
- Hearing aid (less effective alternative)

## Cholesteatoma[10]

A cholesteatoma is a sac of keratinising squamous epithelium that arises from a perforation involving the periphery of the TM. In other words, it is a ‘big sac of skin’ (refer to CHAPTER 39 ). It is dangerous to the ear because it tends to expand and destroy adjacent structures, including the TM, ossicular chain and cochlear. Destruction of the first two may result in conductive hearing loss of up to 60 dB. Irreversible sensorineural deafness caused by otic capsule erosion may also occur. Surgical correction is mandatory.

## Wax impaction[12]

Ear wax impaction occurs in about 5% of the normal population but is more prevalent in older people especially with the use of hearing aids. It is also common in those who use cotton buds (which should be avoided), where cerumen is packed onto the TM leading to a conductive hearing loss. The average wax production is 2.81 mg/week. Most ear wax clears spontaneously without treatment.

Methods of removal include:

- gentle syringing with warm (body temperature) water by trained practitioner (avoid syringing if infection or perforated TM)
- consider cerumenolytic drops for several days before syringing

    carbamide peroxide (Ear Clear)

    docusate sodium (Waxsol)

    hydrogen peroxide

    sodium bicarbonate drops

    oil based (e.g. olive oil, almond oil)

Keratosis obturans is an accumulation of keratin to form a pearly-white plug that requires removal.

## Noise-induced hearing loss

### Clinical features

- Onset of tinnitus after work in excessive noise
- Speech seems muffled soon after work
- Temporary loss initially but becomes permanent if noise exposure continues
- High-frequency loss on audiogram

Sounds exceeding 85 dB are potentially injurious to the cochlea, especially with prolonged exposures. Common sources of injurious noise are industrial machinery, weapons and loud music.

## Tinnitus[13]

The diagnostic strategy is presented in TABLE 33.6 .

**Table 33.6** Diagnostic strategy for tinnitus

| |
|---|
| **Probability diagnosis** |
| Ear wax or debris |
| Sensorineural hearing loss (esp. noise-induced) |
| Otosclerosis |
| Ageing |
| Ear infection (e.g. viral cochleitis) |
| Ménière syndrome |
| **Serious disorders not to be missed** |
| Vascular:<br>• arteriovenous malformation<br>• arterial bruits (esp. carotid)<br>• venous hum (jugular) |
| Infection:<br>• suppurative otitis media |
| Cancer/tumour:<br>• acoustic neuroma (unilateral) |
| Other:<br>• head injury |
| **Pitfalls (often missed)** |
| Impacted wisdom tooth |
| Temporomandibular injury |
| Alcoholism |

*Rarities:*

- superior canal dehiscence
- glomus jugulare tumour
- syphilis

**Seven masquerades checklist**

Anaemia (severe)

Depression

Drugs (aspirin, NSAIDs, loop diuretics, marijuana, quinine, aminoglycosides)

Spinal dysfunction

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

Consider if subjective tinnitus.

Tinnitus is defined as a sound perceived by the ear that arises from an internal source. When pathology in the inner ear is the cause, the tinnitus is non-pulsating, continuous and may have variable frequencies and intensity.



A thorough history and examination should be conducted so that tinnitus can be classified as objective (e.g. heard with stethoscope) or non-objective, and pulsatile or non-pulsatile.

Precautions:

- exclude wax, drugs including marijuana, NSAIDs, salicylates, quinine and aminoglycosides,[9] vascular disease, depression, anaemia, aneurysm, vascular tumours (e.g. glomus tumour), venous hum (jugular vein), acoustic neuroma (progressive and unilateral), Ménière syndrome and infections (e.g. viral cochleitis)
- if pulsatile, consider carotid artery lesions, including a caroticocavernous fistula and an AV fistula
- beware of lonely elderly people living alone (suicide risk)

*Note:* Otosclerosis in young adults causes deafness and tinnitus.

## Investigations

- Audiological examination by audiologist
- Tympanometry and speech discrimination
- MRI or CT scan (if serious cause suspected or head injury)

## Management

- Treat any underlying cause and aggravating factors. Otherwise, minimise symptoms.
- Educate and reassure the patient (tinnitus is nearly always amenable to treatment).
- Encourage a patient support group.

### Holistic approach (options)

Mainly based on acoustic desensitisation:

- Relaxation techniques, address any anxiety
- Tinnitus retraining therapy (clinical psychologist)
- Cognitive behaviour therapy techniques
- Background ‘noise’ (e.g. music played during night for masking)
- Tinnitus maskers
- Hearing aids (based on audiologist assessment)
- Consider hypnotherapy

### Medical

- Clonazepam 0.5 mg nocte (with care)
- Minerals (e.g. zinc and magnesium)
- Betahistine (Serc) 8–16 mg daily (max. 32 mg)
- Carbamazepine or sodium valproate
- Antidepressants if depressed

*Note:* All of the above treatments have unsupportive Cochrane reviews.

### Acute severe tinnitus

- Lignocaine 1% IV slowly (up to 5 mL)

## Hearing aids

Hearing aids are most useful in conductive deafness. This is due to the relative lack of distortion, making amplification simple. In sensorineural deafness, the dual problem of recruitment and the hearing loss for higher frequencies may make hearing aids less satisfactory. Technology is

rapidly advancing, and modern aids selectively amplify higher frequencies and ‘cut out’ excessive volume peaks that would cause discomfort. A trial of such aids should be made by a reliable hearing-aid consultant following full medical assessment.

## Cochlear implants[8,13]

The cochlear implant or ‘bionic ear’ is used in adults and children with severe hearing loss unresponsive to powerful hearing aids. The implant consists of an array of 22 electrodes inserted into the cochlea following mastoidectomy, attached to a receiver implanted in the skull next to the ear. External sounds are detected by an external processor worn behind the ear and connected to the implanted receiver with an external induction coil. Near normal speech and hearing may be achieved in children with congenital or acquired deafness with early implantation. The device is most suitable for children over 2 years and adults with severe deafness.

## Advice for families[14]

Relatives and close friends need considerable advice about coping with deaf family members. They should be told that the deaf person may hear in a quiet room but not in a crowd, and advised of the range of aids and services available and the importance of proper maintenance of any hearing aids (especially with aged people).

### Do

- Face the light when speaking to them.
- Speak directly to them.
- Speak clearly and naturally.
- Speak at a uniform pitch: avoid lowering your voice during or at the end of a sentence.
- Speak within 2 metres.
- Be tolerant and relaxed.
- Be patient with mistakes.
- Write key words on a paper pad when necessary.



### Don’t

- Speak with your back to them.
- Mumble your words.

- Use exaggerated lip movements.
- Shout.
- Put your hand or fingers over your mouth when talking.
- Repeat one word over and over.

### Red flags for priority referral[8]

- Asymmetrical sensorineural hearing loss
- Cranial nerve defects (other than hearing loss)
- Ear canal or middle-ear mass
- Deep ear pain
- Discharging ear

## When to refer

- See ‘Red flags’ above.
- Sudden deafness.
- Any child with suspected deafness, including poor speech and learning problems, should be referred to an audiology centre.
- Any child with middle-ear pathology and hearing loss should be referred to a specialist.
- Unexplained deafness.

### Practice tips

- A mother who believes her child may be deaf is rarely wrong.
- Suspect deafness in an infant with delayed development and in children with speech defects or behavioural problems.
- Audiological assessment should be performed on children born to mothers with evidence of intra-uterine infection by any of the TORCH organisms (toxoplasmosis, rubella, cytomegalovirus and herpes virus).

- No child is too young for audiological assessment. Informal office tests are inadequate for excluding hearing loss.
- Sounds tend to be softer in conductive hearing loss and distorted with sensorineural loss.
- People with conductive deafness tend to speak softly, hear better in a noisy environment, hear well on the telephone and have good speech discrimination.
- People with sensorineural deafness tend to speak loudly, hear poorly in a noisy environment, have poor speech discrimination and hear poorly on the telephone.

## Patient education resources

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

- Hearing problems in children
- Hearing impairment in older people
- Tinnitus

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# 34 Diarrhoea

*A dirty cook gives diarrhoea quicker than rhubarb.*

TUNG-SU PAI (TIME UNCERTAIN)

Diarrhoea is defined as an intestinal disorder characterised by abnormal frequency and liquidity of faecal evacuations.

Acute self-limiting diarrhoea, which is very common and frequently not seen by the medical practitioner, is usually infective and mild, and resolves within days. In Australia most infective cases are viral. The causes of diarrhoea are numerous, thus making a detailed history and examination very important in leading to the diagnosis. 'Chronic' diarrhoea is that lasting at least 4–6 weeks. Important causes are presented in FIGURE 34.1 .

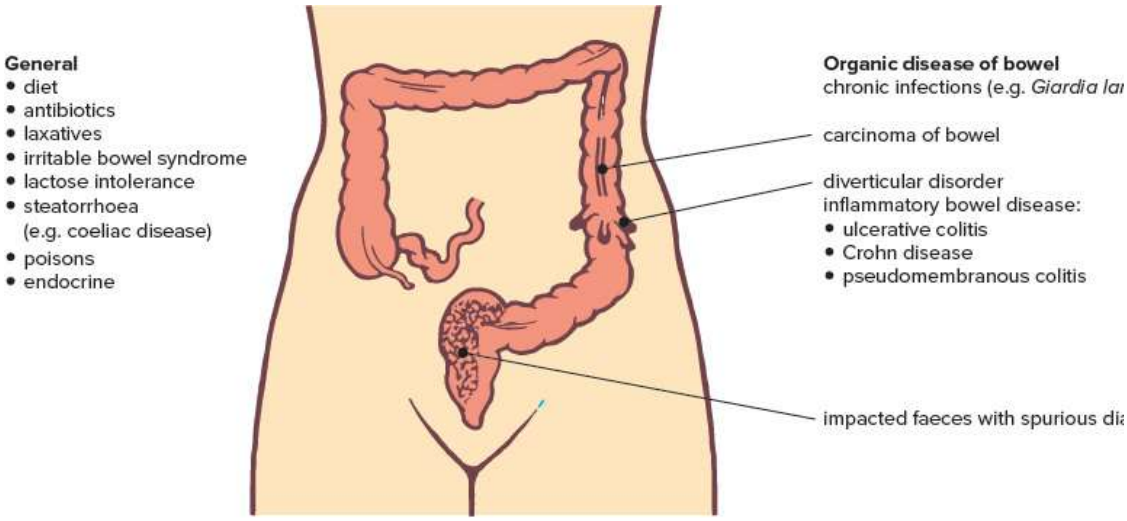


**FIGURE 34.1** Important causes of chronic diarrhoea

The terminology for acute infective diarrhoea can be confusing. A simple classification is:

- vomiting and diarrhoea = gastroenteritis