

- diarrhoea (only) = enteritis

Key facts and checkpoints

- The characteristics of the stool provide a useful guide to the site of the bowel disorder.
- Disorders of the upper GIT tend to produce diarrhoea stools that are copious, watery or fatty, pale yellow or green.
- Colonic disorder tends to produce stools that are small, of variable consistency, brown and may contain blood or mucus.
- Acute gastroenteritis should be regarded as a diagnosis of exclusion.
- Chronic diarrhoea is more likely to be due to protozoal infection (e.g. amoebiasis, giardiasis or *Cryptosporidium*) than bacillary dysentery.
- Asking about a history of travel, especially to countries at risk of endemic bowel infections, is essential.
- Certain antibiotics can cause an overgrowth of *Clostridium difficile*, which produces pseudomembranous colitis.
- Coeliac disease, although a cause of failure to thrive in children, can present at any age.
- In disorders of the colon, the patient experiences frequency and urgency but passes only small amounts of faeces.
- Diarrhoea can be classified broadly into four types:
 - acute watery diarrhoea
 - bloody diarrhoea (acute or chronic)
 - chronic watery diarrhoea
 - steatorrhoea

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A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 34.1 .

Table 34.1 Diarrhoea: diagnostic strategy model

Probability diagnosis

Acute:

- Gastroenteritis/infective enteritis (viral/bacterial)
- Dietary indiscretion
- Antibiotic reaction

Chronic:

- Irritable bowel syndrome
- Drug reactions (e.g. laxatives)
- Chronic infections

Serious disorders not to be missed

Neoplasia:

- colorectal cancer
- ovarian cancer
- peritoneal cancer

HIV infection (AIDS)

Infections:

- cholera
- typhoid/paratyphoid
- amoebiasis
- malaria
- enterohaemorrhagic *E. coli* enteritis

Inflammatory bowel disease:

- Crohn/ulcerative colitis
- pseudomembranous colitis

Non-microbial food poisoning (e.g. death cap mushroom)

Intussusception

Pelvic appendicitis/pelvic abscess

Pitfalls (often missed)

Coeliac disease

Faecal impaction with overflow (spurious) diarrhoea

Lactase deficiency

Giardia lamblia infection

Listeria

Cryptosporidium infection

Cytomegalovirus in immunocompromised
Malabsorption states (e.g. coeliac disease)

Vitamin C and other oral drugs

Nematode infections:

- strongyloides (threadworm)
- whipworm, hookworm

Radiotherapy

Diverticulitis

Post-GIT surgery

Ischaemic colitis (elderly)

Rarities:

- Addison disease (see [CHAPTER 14](#))
- carcinoid tumours
- zinc deficiency in children
- short bowel syndrome
- amyloidosis
- toxic shock
- Zollinger–Ellison syndrome

Seven masquerades checklist

Diabetes

Drugs (e.g. metformin)

Thyroid disorder (hyper)

Is the patient trying to tell me something?

Yes, diarrhoea may be a manifestation of anxiety state or irritable bowel syndrome.

Probability diagnosis

Acute diarrhoea

Common causes are:

- gastroenteritis/enteritis:

bacterial: *Salmonella* sp., *Campylobacter jejuni*, *Shigella* sp., enteropathogenic *Escherichia coli*, *Staphylococcus aureus* (food poisoning)

viral: rotavirus (50% of child hospital admissions),¹ norovirus, astrovirus, adenovirus

- dietary indiscretions (e.g. binge eating)

- antibiotic reactions

Red flag pointers for diarrhoea

- Unexpected weight loss
- Persistent/unresolved
- Blood in stool
- Fever
- Overseas travel
- Severe abdominal pain
- Family history: bowel cancer, Crohn disease

Chronic diarrhoea

Irritable bowel syndrome was the commonest cause of chronic diarrhoea in a UK study.¹

Drug reactions are also important. These include ingestion of laxatives, osmotic agents such as lactose and sorbitol in chewing gum, alcohol, antibiotics, thyroxine and others.

Acute gastroenteritis that persists into a chronic phase is relatively common, especially in travellers returning from overseas. Important considerations are *Giardia lamblia*, *C. difficile*, *Yersinia*, *Entamoeba histolytica*, *Cryptosporidium* and HIV infection.

Serious disorders not to be missed

Colorectal carcinoma must be considered with persistent diarrhoea, especially if of insidious onset.

AIDS due to symptomatic HIV infection needs consideration, especially in those at risk. The serious infectious disorders that can affect international travellers, such as cholera, typhoid, paratyphoid and amoebiasis, should also be kept in mind.

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In children, coeliac disease and cystic fibrosis can present as chronic diarrhoea, while intussusception, although not causing true diarrhoea, can present as loose, redcurrant jelly-like stools and should not be misdiagnosed (as gastroenteritis). Appendicitis must also be considered in the onset of acute diarrhoea and vomiting.

Infection with enterohaemorrhagic strains of *E. coli* (e.g. O157:H7, O111:H8) may lead to the haemolytic uraemic syndrome or thrombotic thrombocytopenic purpura, particularly in children.

What appears to be simple enteritis can eventuate to be fatal. If suspected, avoid giving antibiotics.

Clue: Think of it with atypical gastroenteritis and bloody diarrhoea. Avoid antibiotics.

Death cap mushroom (the world's most lethal)—*Amanita phalloides* (see FIG. 34.2)—causes severe gastroenteritis followed by delayed hepatic failure and AKI.



FIGURE 34.2 *Amanita phalloides* (death cap mushroom)

Source: AleksandarMilutinovic/Shutterstock

Pitfalls

There are many traps in evaluating the patient with diarrhoea, including drug ingestion, especially vitamin C (sodium ascorbate powder), which causes diarrhoea. Faecal impaction with spurious diarrhoea is an age-old pitfall, as is lactase deficiency, which may go undiagnosed for many years. In recent times infection with *G. lamblia* may smoulder on for months with watery, offensive stools before diagnosis.

General pitfalls

- Not considering acute appendicitis in acute diarrhoea—can be retrocaecal or pelvic appendicitis
- Missing faecal impaction with spurious diarrhoea
- Failing to perform a rectal examination
- Failing to consider acute ischaemic colitis in an elderly patient with the acute onset of bloody diarrhoea stools (following sudden abdominal pain in preceding 24 hours)

Seven masquerades checklist

The significant masquerades include diabetes (autonomic neuropathy may cause alternating bouts of constipation and diarrhoea), thyrotoxicosis and drugs. Drugs that can cause diarrhoea are summarised in [TABLE 34.2](#).

Table 34.2 Drugs that can cause or aggravate diarrhoea

- Alcohol, esp. chronic abuse (often overlooked!)
- Antibiotics, esp. penicillin derivatives
- Antihypertensives, selected (e.g. methyldopa)
- Acarbose
- Caffeine
- Cardiac agents (e.g. digoxin, quinidine)
- Colchicine
- Cytotoxic agents (e.g. methotrexate)
- Food and drug additives: sorbitol, mannitol, fructose, lactose
- Heavy metals
- H₂-receptor antagonists
- Iron-containing compounds
- Laxatives
- Magnesium-containing antacids/magnesium supplements
- Metformin
- Misoprostol
- NSAIDs
- Orlistat
- Prostaglandins
- Salicylates
- Sildenafil
- Statins
- Theophylline
- Thyroxine

Pseudomembranous colitis (antibiotic-associated diarrhoea)²

This potentially fatal colitis can be caused by the use of any antibiotic, especially clindamycin,

lincomycin, ampicillin and the cephalosporins (an exception is vancomycin). It is usually due to an overgrowth of *C. difficile*, which produces a toxin that causes specific inflammatory lesions, sometimes with a pseudomembrane. It may occur, uncommonly, without antibiotic usage.

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Clinical features

- Profuse, watery diarrhoea
- Abdominal cramping and tenesmus ± fever
- Within 2 days of taking antibiotic (can start up to 4 to 6 weeks after usage)
- Persists 2 weeks (up to 6) after ceasing antibiotic

Diagnosed by characteristic lesions on sigmoidoscopy and a tissue culture assay and/or PCR for *C. difficile* toxin.

Treatment²

- Cease antibiotic
- Hygiene measures to prevent spread
- Mild to moderate: metronidazole 400 mg (o) tds for 10 days
- Severe: vancomycin 125 mg (o) qid for 10 days
- Consult with specialist. Beware of toxic megacolon.

Psychogenic considerations

Anxiety and stress can cause looseness of the bowel. The irritable bowel syndrome, which is a very common condition, may reflect underlying psychological factors and most patients find that the symptoms are exacerbated by stress. Look for evidence of depression.

In children, chronic diarrhoea can occur with the so-called ‘maternal deprivation syndrome’, characterised by growth and developmental retardation due to adverse psychosocial factors.

The clinical approach

History

As always, the history is the key to the diagnosis. First establish what the patient means by the term ‘diarrhoea’, his or her normal pattern and how the presenting problem varies from normal.

It is important to analyse the nature of the stools, the frequency of diarrhoea, associated

symptoms, including abdominal pain, and constitutional symptoms, such as fever. Food intake in the past 72 hours and recent travel abroad may give a clue to acute gastroenteritis or food poisoning (an acute, self-limiting illness of diarrhoea and vomiting). The difference between food poisoning and infective gastroenteritis is presented in TABLE 34.3 . However, there can be an overlap of features from a specific organism and the exercise may be semantic, but it may provide a clue to food-borne causation. A summary of non-microbial food poisoning is presented in TABLE 34.4 .

Table 34.3 Comparison of acute diarrhoea due to bacterial food poisoning and infective gastroenteritis

	Food poisoning	Infective gastroenteritis
Responsible organisms	Toxins from: <i>Staphylococcus aureus</i> <i>Salmonella</i> sp. <i>Clostridium perfringens</i> <i>Clostridium difficile</i> <i>Vibrio parahaemolyticus</i> <i>Aeromonas hydrophilia</i> <i>Bacillus cereus</i>	Viral Bacterial, e.g. <i>Campylobacter jejuni</i> <i>Escherichia coli</i> <i>Shigella</i> sp. <i>Salmonella</i> sp.
Incubation period (onset from contact)	Short—within 24 hours Average—12 hours <i>S. aureus</i> —2–4 hours	3–5 days
Diarrhoea	Watery	Diarrhoea ± blood
Other features	Abdominal cramps (milder) Dehydration Headache Vomiting	Abdominal cramps
Typical foods	Chicken Meat Seafood Rice	Milk Water Chicken

Custard and cream (*S. aureus*)

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Table 34.4 Non-microbial food poisoning³

Food (specific types)	Toxin	Onset	Features (symptoms)
Mushrooms	Muscarine	Minutes to hours	N, V, D, P: CNS symptoms
Toadstools	Amatoxin	Hours	N, V, D, P: hepatic failure
Immature or sprouting potatoes	Solanine	Within hours	N, V, D, P Throat constriction
Fish	Ichthyosarcotoxin Various (e.g. ciguatera, scombrotoxin)	10–60 minutes (occasionally longer)	N, V, D, P Circumoral tingling CNS symptoms Collapse
Mussels	Mytilotoxism	5–30 minutes	N, V, P CNS: paralysis
Grain, esp. rye	Ergot fungus Alphatoxin	Minutes to 24 hours	N, V, P Circulatory and CNS
Fava beans (favism)	Enzyme deficiency	Rapid	V, D Acute haemolysis

N = nausea; V = vomiting; D = diarrhoea; P = abdominal pain

A drug history is relevant, as is a family history of diarrhoea, which may be significant for coeliac disease, Crohn disease and cystic fibrosis.

People at risk from HIV infection should be discreetly evaluated.

Key questions

Acute diarrhoea

- Where did you eat in the 24 hours before the diarrhoea started?
- What food did you eat during this time?
- Did you have chicken or seafood recently? (Chicken may be contaminated with *Salmonella* or *Campylobacter* and seafood with *Vibrio parahaemolyticus*.)
- Did any other people get the same problem?
- Have you travelled overseas recently? Where?
- Have you noticed any blood or mucus in your motions?
- Have you had any previous attacks?
- Have you noticed fever, weakness or other symptoms?

Chronic diarrhoea

- Have you noticed any blood or mucus in the motion?
- Have you travelled overseas recently? Where?
- Do you have pain and is it relieved by opening your bowels or passing wind?
- Does anyone else in your family have diarrhoea?
- Have you had any operations on your abdomen recently?
- What medications are you taking?
- Are you taking antibiotics?
- Do you take vitamin C for your health?
- Do you take laxatives?
- How much alcohol do you drink? Fruit juice?
- How much milk do you drink?
- What about thick shakes, ice-cream and yoghurt?

- Do you get clammy or shaky, or have you lost weight?
- Have you had trouble with pain in your joints, back pain, eye trouble or mouth ulceration?
- Do you have trouble flushing your motions down the toilet?
- Do you get diarrhoea during the night?
- Are you under a lot of stress?

Significance of symptoms

Abdominal pain

Central colicky abdominal pain indicates involvement of the small bowel, while lower abdominal pain points to the large bowel.

Nature of stools

If small volume, consider inflammation or carcinoma of colon; if large volume, consider laxative abuse and malabsorption.

If there is profuse bright red bleeding, consider diverticulitis or carcinoma of colon, and if small amounts with mucus or mucopus, consider inflammatory bowel disorder. The presence of blood in the stools excludes functional bowel disorder. Diarrhoea at night suggests organic disease. In steatorrhoea the stools are distinctively pale, greasy, offensive, floating and difficult to flush. It is exacerbated by fatty foods.

‘Rice water’ stool is characteristic of cholera and ‘pea soup’ stool of typhoid fever.

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Examination

The extent of the examination depends on the nature of the presenting problem. If it is acute, profuse and associated with vomiting, especially in a child, the examination needs to be general to assess the effects of fluid, electrolyte and nutritional loss. An infant’s life is in danger from severe gastroenteritis and this assessment is a priority. The general nutritional and electrolyte assessment is also relevant in chronic diarrhoea with malabsorption, and this includes looking for evidence of muscle weakness (e.g. hypokalaemia, hypomagnesaemia, tetany [hypocalcaemia], bruising [vitamin K loss]).

The examination should also focus on the abdomen (systematic palpation), the rectum and the skin. Possible helpful signs are included in FIGURE 34.3 .

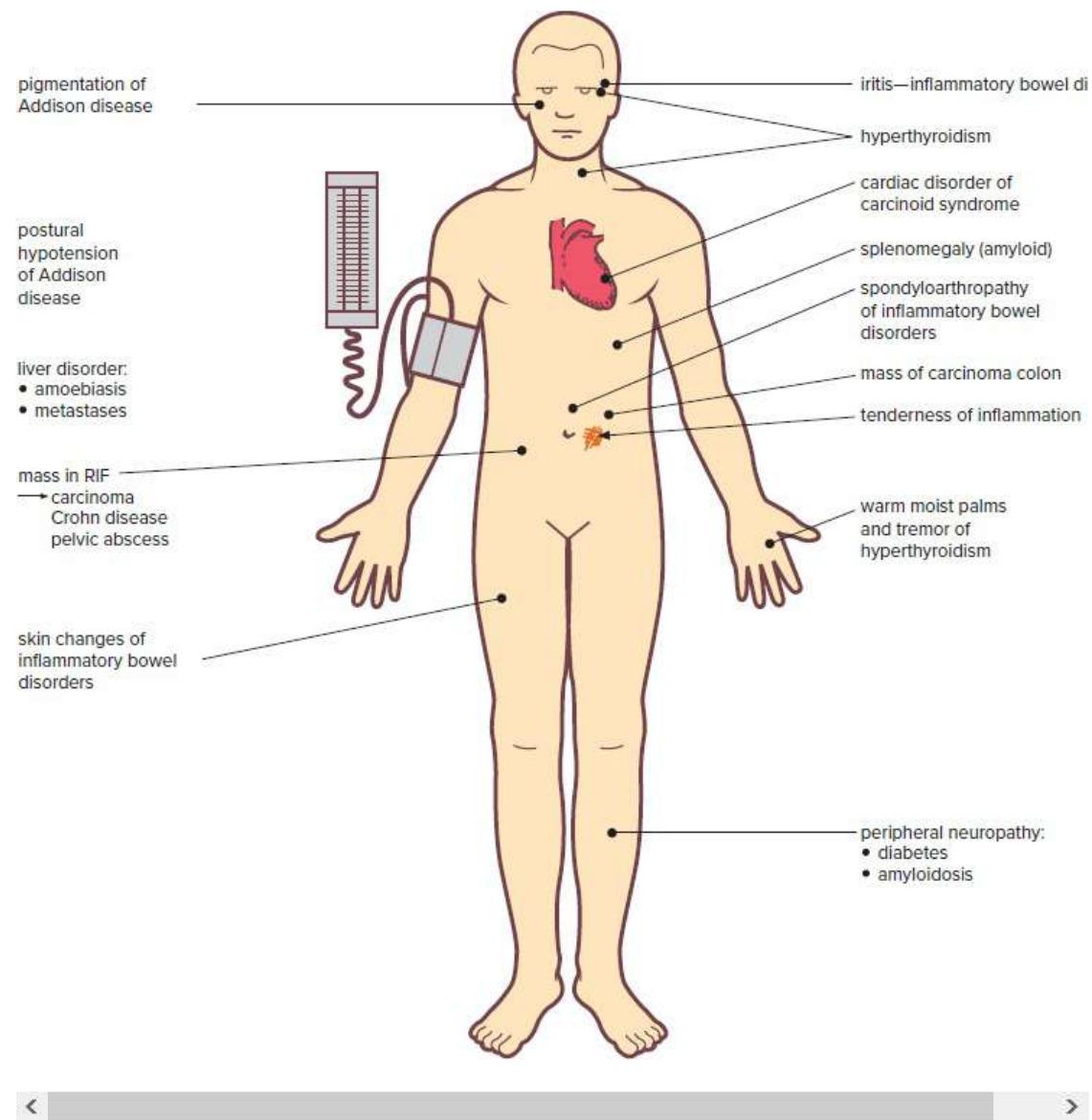


FIGURE 34.3 Possible significant signs in a person with diarrhoea

The stool

Ideally the stool should be examined. The consistency of the stool as an aid to diagnosis^{2,4} is summarised in TABLE 34.5 , the features of the stool in TABLE 34.6 and the characteristics that distinguish between small and large bowel diarrhoea¹ are presented in TABLE 34.7 . Note the presence of blood, mucus or steatorrhoea.

Table 34.5 Stool consistency as an aid to diagnosis

Consistency	Probable cause
-------------	----------------

Liquid and uniform	Small bowel disorder (e.g. gastroenteritis)
Loose with bits of faeces	Colonic disorder
Watery, offensive, bubbly	<i>Giardia lamblia</i> infection
Liquid or semiformed, mucus ± blood	Entamoeba histolytica
Bulky, pale, offensive	Malabsorption
Pellets or ribbons	Irritable bowel syndrome

Table 34.6 Stool features as an aid to diagnosis

Stool appearance	Cause to consider
China clay	Obstructive jaundice
Black stool	Melaena (blood) in faeces
Pea soup	Typhoid fever
Rabbit pellets	Irritable bowel syndrome
Redcurrant jelly	Intussusception
Rice water	Cholera
Silver stool	Carcinoma of ampulla of Vater
Toothpaste	Hirschsprung disease

Table 34.7 Distinction between small and large bowel diarrhoea

	Small bowel	Large bowel
Volume	Large	Small
Pain	Central	Lower/LIF
Borborygmi	++	-
Undigested food	+	-
Steatorrhoea	+/-	-
Blood	-	+
Mucus	-	+
Urgency	-	+

Bloody diarrhoea

Consider: inflammatory bowel disease, colonic polyps, carcinoma, infective especially *Shigella*, *Salmonella*, *Campylobacter*, *E. coli*, amoebiasis, colitis (pseudomembranous, ischaemic).

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Investigations³

The following list includes a range of tests that may be required. Appropriate tests should be judiciously selected and in many instances, particularly acute self-limiting diarrhoea, no investigations are necessary.

- Stool tests:
 - microscopy for parasites and red and white cells (warm specimen for amoebiasis)
 - cultures: routine for *Salmonella* sp., *Shigella* sp., *E. coli* and possibly *Campylobacter*; may need special requests for *Campylobacter* sp., *C. difficile* and toxin, listeria, *Yersinia* sp., *Cryptosporidium* sp., *Aeromonas* sp. (stools must be collected fresh on three occasions)
- Blood tests (especially chronic diarrhoea): haemoglobin; MCV, WCC, ESR, iron, ferritin, folate, vitamin B12, calcium, electrolytes, thyroid function, HIV tests
- Specific tests for organisms
 - Antibody tests, total IgA (e.g. IgA transglutaminase for coeliac disease); PCR tests (where applicable)
 - Haemagglutination tests for amoebiasis
 - C. difficile* tissue culture assay
 - Malabsorption studies
 - Stool elastase for pancreatic insufficiency
- Endoscopy:
 - proctosigmoidoscopy
 - flexible sigmoidoscopy/colonoscopy (with biopsy)
 - small bowel biopsy (coeliac disease)
- Radiology:

plain X-ray abdomen—of limited value

small bowel enema

barium enema, especially double contrast

Note: Those with HIV should be investigated in specialist centres due to complexity.

Complications of diarrhoea

- Fluid loss with dehydration, electrolyte loss (Na^+ , K^+ , Mg^+ , Cl^-)
- Vascular collapse
- Hypokalaemia

Principles of treatment³

When an underlying cause of diarrhoea can be identified, apart from some common infections, management should be directed at that cause. There are a few situations in which the causative bacterial or parasitic pathogen requires specific treatment, for example, giardiasis. The management is determined by the nature of the pathogen and the severity of the illness.

However, in Australia most infective cases are viral. The basic principle therefore is to achieve and maintain adequate hydration until the illness resolves. In adults and children, oral rehydration is indicated unless there is evidence of impending circulatory ‘shock’ demanding intravenous therapy. Oral rehydration solution containing sodium, potassium and glucose should be considered for patients with mild to moderate dehydration. Adults should drink 2 to 3 L of the solution in 24 hours. Normal food intake may start after rehydration.

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In general, treatment should not be directed specifically at altering the frequency and consistency of the stools. The antimotility drugs (loperamide, diphenoxylate and codeine) have a role restricted to short-term control of symptoms in adults during periods of significant social inconvenience, such as travel. It must be emphasised that antimotility drugs should be used with caution, especially for *C. difficile*, *Salmonella* and *Shigella*, and are never indicated for management of acute diarrhoea in infants and children.⁵

The traditional absorbent agents, such as kaolin/pectin mixtures, activated charcoal and other mineral clays, have not been shown to be of value and may interfere with absorption of other drugs. They should not be used.

Specific antibiotics are reserved for the treatment of giardiasis, amoebiasis, antibiotic-associated diarrhoea, cholera and typhoid. Although antibiotics are usually unnecessary, they may be indicated for severe cases of *Campylobacter* enteritis, *Salmonella* enteritis, shigellosis and traveller’s diarrhoea. Lactobacillus has been shown to reduce the duration of diarrhoea in rotavirus-related enteritis and antibiotic-associated diarrhoea.

Diagnostic triads for diarrhoea



DxT acute diarrhoea + colicky abdominal pain ± vomiting → gastroenteritis

DxT (young adult) diarrhoea ± blood and mucus + abdominal cramps → inflammatory bowel disease (UC/Crohn)

DxT as above + constitutional symptoms ± eyes/joints → Crohn disease

DxT pale bulky offensive stools, difficult to flush, weight loss → malabsorption

DxT fatigue + weight loss + iron deficiency → coeliac disease

DxT failure to thrive (child) + recurrent chest infections → cystic fibrosis

DxT altered bowel habit: diarrhoea ± constipation ± rectal bleeding ± abdominal discomfort → colorectal carcinoma

DxT diarrhoea (fluid/incontinent) + constipation + abdominal discomfort + anorexia/nausea → faecal impaction

DxT profuse watery diarrhoea + abdominal cramps and increasing distension (on antibiotics) → pseudomembranous colitis (Girotra's triad)

DxT variable diarrhoea/constipation + abdominal discomfort + mucus PR + flatulence → irritable bowel syndrome

Malabsorption

It is important to distinguish the steatorrhoea of various malabsorption syndromes from diarrhoea. Important causes are presented in [TABLE 34.8](#) .

Table 34.8 Important causes of malabsorption

Primary mucosal disorders

Gluten-sensitive enteropathy (coeliac disease)

Tropical sprue

Lactose intolerance (lactase deficiency)

Crohn disease (regional enteritis)

Whipple disease

Parasite infections (e.g. *Giardia lamblia*)

Lymphoma

Maldigestion states

Lumenal abnormalities:

- postsurgery (e.g. gastrectomy, ileal resection)
 - systemic sclerosis
-

Pancreatic disorders

Chronic pancreatitis

Cystic fibrosis

Pancreatic tumours (e.g. Zollinger–Ellison)

The common causes are coeliac disease, chronic pancreatitis and postgastrectomy.

Clinical features

- Bulky, pale, offensive, frothy, greasy stools
- Stools difficult to flush down toilet
- Weight loss
- Prominent abdomen
- Failure to thrive (in infants)
- Increased faecal fat
- Signs of multiple vitamin deficiencies (e.g. A, D, E, K)
- Sore tongue (glossitis)
- Hypochromic or megaloblastic anaemia (possible)

Refer for specific investigations (e.g. FBE, barium studies, small bowel biopsy, faecal fat [>21 g/3 days]).

Coeliac disease²

Synonyms: coeliac sprue, gluten-sensitive enteropathy.

Note: It can appear at *any* age; refer to coeliac disease in children (see later in chapter).

It is widely underdiagnosed because most patients present with non-GIT symptoms, such as tiredness.

There is a genetic factor in this autoimmune disorder with a 1 in 10 chance if a first-degree relative is affected. Consider screening under 2 years if there is such an association.

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Clinical features

- Classic tetrad: diarrhoea, weight loss, iron/folate deficiency, abdominal bloating
- Malaise, lethargy
- Flatulence
- Mouth ulceration
- Diarrhoea with constipation (alternating)
- Pale and thin patient
- No subcutaneous fat

Diagnosis

- Elevated faecal fat
- Characteristic duodenal biopsy: villous atrophy (key test)
- Total IgA level
- IgA transglutaminase antibodies (>90% sensitivity and specificity)
- Deamidated gliadin peptide (DGP-IgG) also highly sensitive and specific

Associations

- Iron-deficiency anaemia
- Malignancy, especially lymphoma, GIT
- Type 1 diabetes
- Pernicious anaemia
- Primary biliary cirrhosis
- Subfertility
- Dermatitis herpetiformis

- IgA deficiency
- Autoimmune thyroid disease
- Osteoporosis
- Neurological (e.g. seizures, ataxia, peripheral neuropathy)
- Down syndrome

Management

- Diet control: high complex carbohydrate and protein, low fat, lifelong gluten-free (no wheat, barley, rye and oats)
- Treat specific vitamin and mineral deficiencies
- Give pneumococcal vaccination (increased risk of pneumococcus sepsis)
- Coeliac support group and Coeliac Australia

Gluten-free diet

Avoid foods containing gluten either as an obvious component (e.g. flour, bread, oatmeal) or as a hidden ingredient (e.g. dessert mix, stock cube).

Forbidden foods include:

- standard bread, pasta, crispbreads, flour
- standard biscuits and cakes
- breakfast cereals made with wheat or oats
- oatmeal, wheat bran, barley/barley water
- ‘battered’ or breadcrumbed fish, etc.
- meat and fruit pies
- most stock cubes and gravy mixes

Whipple disease

This is a rare malabsorption disorder usually affecting white males. It is caused by the bacillus *Tropheryma whipplei*. It may involve the heart, lungs and CNS. It is fatal if missed.

Clinical features

- Males >40 years
- Chronic diarrhoea (steatorrhoea)
- Arthralgia (migratory seronegative arthropathy mainly of peripheral joints)
- Weight loss
- Lymphadenopathy
- ± Fever

Diagnoses

- PCR for *T. whipplei*
- Jejunal biopsy—stunted villi

Treatment

IV ceftriaxone for 2 weeks then cotrimoxazole or tetracycline for up to 12 months.

This produces a dramatic improvement.

Diarrhoea in the elderly

The older the person, the more likely a late onset of symptoms that reflect serious underlying organic disease, especially malignancy. Colorectal cancer needs special consideration. Frail or bedridden people have an increasing likelihood with age of faecal impaction with spurious diarrhoea. The possibility of drug interactions (e.g. digoxin) and ischaemic colitis should also be considered.

Ischaemic colitis

This is due to atheromatous occlusion of mesenteric vessels (low blood flow) (see CHAPTER 24).

Clinical features

Clinical features include:

- sharp abdominal pain in an elderly person with bloody diarrhoea (low blood flow)

or

- perumbilical pain and diarrhoea about 15–30 minutes after eating

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- may be bruits over central abdomen

- other evidence of generalised atherosclerosis

- barium enema shows ‘thumb printing’ sign due to submucosal oedema

- the definitive test is aortography and selective angiography of mesenteric vessels
- most episodes resolve—may be followed by a stricture

Diarrhoea in children

The commonest cause of diarrhoea in children is acute infective gastroenteritis, followed by antibiotic-induced diarrhoea. However, certain conditions that develop in infancy and childhood require special attention. The presentation of small amounts of redcurrant jelly-like stool with intussusception should be kept in mind. Of the many causes, only a few could be considered common.

Important causes of diarrhoea in children are:

- infective gastroenteritis
- antibiotics
- overfeeding (loose stools in newborn)
- dietary indiscretions
- toddler's diarrhoea
- sugar (carbohydrate) intolerance
- food allergies (e.g. milk, soy bean, wheat, eggs)
- maternal deprivation
- malabsorption states: cystic fibrosis, coeliac disease

Note: Exclude surgical emergencies (e.g. acute appendicitis), infections (e.g. pneumonia), septicaemia, otitis media <5 years.

Acute gastroenteritis

Note: Dehydration from gastroenteritis is an important cause of death, particularly in obese infants (especially if vomiting accompanies the diarrhoea).

Definition

It is an illness of acute onset of less than 10 days' duration, associated with fever, diarrhoea and/or vomiting, where there is no other evident cause for the symptoms.⁵

Causes

- Mainly rotavirus (developed countries) and adenovirus: viruses account for about 80%
- Bacterial: *C. jejuni* and *Salmonella* sp. (two commonest), *E. coli* and *Shigella* sp.
- Protozoal: *G. lamblia*, *E. histolytica*, *Cryptosporidium*
- Food poisoning—staphylococcal toxin

Differential diagnoses. These include septicaemia, urinary tract infection, intussusception, appendicitis, pelvic abscess, partial bowel obstruction, type 1 diabetes and antibiotic reaction⁴ (see TABLE 34.9).

Table 34.9 Differential diagnosis of acute diarrhoea and vomiting in children

Bowel infection:

- viruses
- bacteria
- protozoal
- food poisoning—staphylococcal toxin

Systemic infection

Abdominal disorders:

- appendicitis
- pelvic abscess
- intussusception
- malrotation

Urinary tract infection

Antibiotic reaction

Diabetes

Note: Exclude acute appendicitis and intussusception in the very young.

Symptoms

- Diarrhoea, anorexia, nausea, poor feeding, vomiting, fever (vomiting and fever may be absent)
- Fluid stools (often watery) 10–20 per day
- Crying—due to pain, hunger, thirst or nausea
- Bleeding—uncommon (usually bacterial)

- Anal soreness

Viral indication: large volume, watery, typically lasts 2–3 days, systemic symptoms uncommon.

Bacterial indication: small motions, blood, mucus, abdominal pain and tenesmus.

Dehydration: must be assessed (see TABLE 34.10).

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Table 34.10 Assessment of hydration²

	Mild	Moderate	Severe
Body weight loss	3–5%	6–9%	≥9%
Symptoms/general observations	Thirsty Alert Restless	Thirsty Restless Lethargic Irritable	Infants: drowsy, limp, cold, sweaty, cyanotic limbs, comatose Older: apprehensive, cold and sweaty, cyanotic limbs
Signs	Normal	Dry mucous membranes, absent tears Tachycardia Mildly sunken eyes	Rapid feeble pulse Hypotensive Sunken eyes and fontanelles Very dry mucous membranes
Pinched skin test	Normal (<1 second)	Retracts slowly (1–2 seconds)	Retracts very slowly (>2 seconds)
Urine output	Normal	Decreased	Nil
Treatment	Oral rehydration: <ul style="list-style-type: none"> • small amounts of fluids often • continue breastfeeding • solids after 24 hours • provide maintenance 	Oral rehydration: <ul style="list-style-type: none"> • consider nasogastric tube for steady fluid infusion or • IV infusion 	Admit to hospital Urgent IV infusion: isotonic fluid (0.9% saline) Start with bolus 20 mL/kg

fluid and loss

Complications:

- febrile convulsions
- sugar (lactose) intolerance (common)
- septicaemia, especially *Salmonella*

Management

Management is based on the assessment and correction of fluid and electrolyte loss.^{5,6} Since dehydration is usually isotonic with equivalent loss of fluid and electrolytes, serum electrolytes will be normal.

Note: The most accurate way to monitor dehydration is to weigh the child, preferably without clothes, on the same scale each time. However, the easiest is clinical assessment (e.g. vomiting, no urine, lethargy and thirst). Perform faecal microbiological testing for routine pathogens if there are features of severe disease.

Commercially available oral rehydration solutions (ORS) must have levels of glucose and sodium/potassium salts that meet WHO standards. Most brand names end in ‘-lyte’. They come either as sachets that must be reconstituted with a specific volume of water, or ready-made liquids, including frozen ice sticks. ‘Sports drinks’ are not designed for this purpose. One trial in mildly dehydrated children >2 years old showed that dilute apple juice was better tolerated and resulted in less treatment failure than ORS.⁷

If acute invasive or persistent *Salmonella* are present, give antibiotics (ciprofloxacin or azithromycin).

Avoid

- Drugs: antidiarrhoeals, anti-emetics and antibiotics
- Full-strength lemonade or similar sugary soft drink: osmotic load too high, can use if diluted 1 part to 4 parts water but sugar may be poorly tolerated

To treat or not to treat at home

- Treat at home—if family can cope, vomiting is not a problem and no dehydration.
- Admit to hospital—if dehydration or persisting vomiting or family cannot cope; also infants <6 months and high-risk patients.

Advice to parents (for mild-to-moderate diarrhoea)

If applicable, remove child from day care or school and keep away from food preparation areas. Advise about hygiene, including handwashing and napkin disposal. If children are not vomiting, encourage eating and drinking as tolerated.

General rules^{6,8}

- Give small amounts of fluids often
- Return to an age-appropriate diet as soon as possible after rehydration
- Start solids after 24 hours
- Continue breastfeeding (should be increased in frequency, e.g. hourly)
or
- Continue formula feeding if tolerated or resume it after 24 hours
- Consider stool culture and test for rotavirus for symptoms that persist and worsen

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Day 1

Give fluids, a little at a time and often (e.g. 5 mL every 1–2 minutes by spoon or syringe or 50 mL every 15 minutes if vomiting a lot). A good method is to give 200 mL (about 1 cup) of fluid every time a watery stool is passed or a big vomit occurs.

Use ORS if tolerated, or alternatives such as diluted apple juice if the child prefers.

Warning: Do not use straight lemonade or mix up powders with lemonade or fluids other than water.

Alternatives are:

• lemonade (not low-calorie)	1 part to 4 parts water
• sucrose (table sugar)	1 teaspoon to 120 mL water
• glucose	1 teaspoon to 120 mL water
• cordials (not low-calorie)	1 part to 16 parts water
• fruit juice (esp. apple)	1 part to 4 parts water

Method of assessing fluid requirements:³

- Fluid loss (mL) = % dehydration × body weight (kg) × 10
- Maintenance (mL/kg/24 h): 1–3 mo: 120 mL; 4–12 mo: 100 mL; >12 mo: 80 mL

- Allow for continuing loss.

Example: 8 month 10 kg child with 5% dehydration:

$$\text{Fluid loss} = 5 \times 10 \times 10 = 500 \text{ mL}$$

$$\text{Maintenance} = 100 \times 10 = 1000 \text{ mL}$$

$$\text{Total 24-hour requirement (min.)} = 1500 \text{ mL}$$

$$\text{Approximate average hourly requirement} = 60 \text{ mL}$$

- Aim to give more (replace fluid loss) in the first 6 hours.
- Rule of thumb: give 100 mL/kg (infants) and 50 mL/kg (older children) in first 6 hours.

Days 2 and 3

Reintroduce your baby's milk or formula diluted to half strength (i.e. mix equal quantities of milk or formula and water). Their normal food can be continued but do not worry that your child is not eating food. Solids can be commenced after 24 hours. Best to start with bread, plain biscuits, jelly, stewed apple, rice, porridge or non-fat potato chips. Avoid fatty foods, fried foods, raw vegetables and fruit, and wholegrain bread.

Day 4

Increase milk to normal strength and gradually continue reintroduction to usual diet.

Breastfeeding. If your baby is not vomiting, continue breastfeeding but offer extra fluids (preferably ORS) between feeds. If vomiting is a problem, express breast milk for the time being while you follow the oral fluid program.

Note: Watch for lactose intolerance as a sequela—explosive diarrhoea after introducing formula. Replace with a lactose-free formula.

Chronic diarrhoea in children⁶

⌚ Sugar intolerance

Synonyms: carbohydrate intolerance, lactose intolerance.

The commonest offending sugar is lactose.

Diarrhoea often follows acute gastroenteritis when milk is reintroduced into the diet (some recommend waiting for 2 weeks). Stools may be watery, frothy, smell like vinegar and tend to excoriate the buttocks. They contain sugar. Exclude giardiasis.

A simple test follows.

- Line the napkin with thin plastic and collect fluid stool.
- Mix 5 drops of liquid stool with 10 drops of water and add a Clinitest tablet (detects lactose and glucose but not sucrose).
- A positive result suggests sugar intolerance.

Diagnosis: lactose breath hydrogen test.

Treatment

- Remove the offending sugar from the diet.
- Use milk preparations in which the lactose has been split to glucose and galactose by enzymes, or use soy protein.

Note: Most milk allergies improve with age.

⌚ Toddler's diarrhoea ('cradle crap')

A clinical syndrome of loose, bulky, non-offensive stools with fragments of undigested food in a well, thriving child. The onset is usually between 8 and 20 months. Associated with high fructose intake (fruit juice diarrhoea).

Diagnosis by exclusion; treatment by dietary adjustment.

⌚ Cow's milk protein intolerance⁸

This is not as common as lactose intolerance. Diarrhoea is related to taking a cow's milk formula and relieved when it is withdrawn.

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Allergic responses to cow's milk protein may result in a rapid or delayed onset of symptoms. Delayed onset may be more difficult to diagnose, presenting with diarrhoea, malabsorption or failure to thrive.

It is diagnosed by unequivocal reproducible reactions to elimination and challenge. If diagnosed, remove cow's milk from the diet and replace with either soy milk or a hydrolysed or an elemental formula (see [CHAPTER 72](#)).

⌚ Inflammatory bowel disorders

These disorders, which include Crohn disease and ulcerative colitis, can occur in childhood. A high index of suspicion is necessary to make an early diagnosis. Approximately 5% of cases of chronic ulcerative colitis have their onset in childhood.⁵

Chronic enteric infection

Responsible organisms include *Salmonella* sp., *Campylobacter*, *Yersinia*, *G. lamblia* and *E. histolytica*. With persistent diarrhoea, it is important to obtain microscopy of faeces and aerobic and anaerobic stool cultures. *G. lamblia* infestation is not an uncommon finding and may be associated with malabsorption, especially of carbohydrate and fat. Giardiasis can mimic coeliac disease.

Coeliac disease

(See earlier in chapter.)

Clinical features in childhood:

- usually presents at 9–18 months, but any age
- previously thriving infant
- anorexia, lethargy, irritability
- failure to thrive
- malabsorption—abdominal distension
- offensive frequent stools

Diagnosis: duodenal biopsy (definitive).

Treatment: remove gluten from diet.

Cystic fibrosis

Cystic fibrosis, which presents in infancy, is the commonest of all inherited disorders (1 per 2500 live births). Refer to [CHAPTER 23](#).

Acute gastroenteritis in adults

Features

- Invariably a self-limiting problem (1–3 days)
- Abdominal cramps
- Possible constitutional symptoms (e.g. fever, malaise, nausea, vomiting)
- Other meal-sharers affected → food poisoning

- Consider dehydration, especially in the elderly
- Consider possibility of enteric fever

Traveller's diarrhoea

The symptoms are usually as above, but very severe diarrhoea, especially if associated with blood or mucus, may be a feature of a more serious bowel infection such as amoebiasis. Possible causes of diarrhoeal illness are presented in [CHAPTER 129](#). Most traveller's diarrhoea is caused by *E. coli*, which produces a watery diarrhoea within 14 days of arrival in a foreign country. Another organism is *Cryptosporidium parvum*. If moderate to severe, azithromycin is recommended for 2–3 days. (For specific treatment refer to the section on Traveller's diarrhoea in [CHAPTER 129](#).)

Persistent traveller's diarrhoea

Any traveller with persistent diarrhoea after visiting less developed countries, especially India and China, may have a protozoal infection such as amoebiasis or giardiasis.

If there is a fever and blood or mucus in the stools, suspect amoebiasis. Giardiasis is characterised by abdominal cramps, flatulence and bubbly, foul-smelling diarrhoea.

Principles of treatment of diarrhoea

Acute diarrhoea

- Maintenance of hydration:

anti-emetic injection (for severe vomiting) prochlorperazine IM, statim

or

metoclopramide IV, statim

- Antidiarrhoeal preparations:

(avoid if possible, but loperamide preferred) loperamide (Imodium) 2 caps statim then 1 after each unformed stool (max. 8 caps/day)

or

diphenoxylate with atropine (Lomotil) 2 tabs statim then 1–2 (o) 8 hourly

General advice to patient

Rest

Your bowel needs a rest and so do you. It is best to reduce your normal activities until the diarrhoea has stopped.

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Diet

Eat as normally as possible but drink small amounts of clear fluids such as water, tea, lemonade and yeast extract (e.g. Vegemite). Then eat low-fat foods such as stewed apples, rice (boiled in water), soups, poultry, boiled potatoes, mashed vegetables, dry toast or bread, biscuits, most canned fruits, jam, honey, jelly, dried skim milk or condensed milk (reconstituted with water).

At first, avoid alcohol, coffee, strong tea, fatty foods, fried foods, spicy foods, raw vegetables, raw fruit (especially with hard skins), wholegrain cereals and cigarette smoking.

On the third day introduce dairy produce, such as a small amount of milk in tea or coffee and a little butter or margarine on toast. Add also lean meat and fish (either grilled or steamed).

Treatment (antimicrobial drugs)^{2,9}

Bacterial diarrhoea in adults and older children is usually self-limiting and does not require antibiotic treatment (they may be used to shorten the course of a persistent infection).

Campylobacter, Salmonella, Shigella and *E. coli* are the most common causes. As a rule, use oral rehydration solution 2–3 L orally over 24 hours if mild to moderate dehydration. If severe, intravenous rehydration with N saline is recommended.

It is advisable not to use antimicrobials except where the following specific organisms are identified. The drugs should be selected initially from the list below or modified according to the results of culture and sensitivity tests.³ Only treat if symptoms have persisted for more than 48 hours. Adult doses are shown for the following specific enteric infections based on faecal culture. Recommended empirical therapy is ciprofloxacin or norfloxacin.

***Shigella* dysentery (moderate to severe)**

cotrimoxazole (double strength) 1 tab (o) 12 hourly for 5 days: use in children (children's doses)

or

norfloxacin 400 mg (o) 12 hourly for 5 days (preferred for adults)

or

ciprofloxacin 500 mg (o) bd for 5 days

Giardiasis

This protozoal infestation is often misdiagnosed. It should be considered for a persistent profuse, watery, bubbly, offensive diarrhoea (see CHAPTER 129).

tinidazole 2 g (o), single dose (may need repeat)

or

metronidazole 400 mg (o) tds for 7 days

(in children: 30 mg/kg/day [to max. 1.2 g/day] as single daily dose for 3 days)

Salmonella enteritis

Antibiotics are not generally advisable, but if severe or prolonged, use:

ciprofloxacin 500 mg (o) bd for 5–7 days

or

azithromycin 1 g (o) day, then 500 mg for 6 days

or

ceftriaxone IV or ciprofloxacin IV if oral therapy not tolerated

Note: *Salmonella* is a notifiable disease; infants under 15 months are at risk of invasive *Salmonella* infection.

Campylobacter

A zoonosis that is usually self-limiting.

Antibiotic therapy indicated in severe or prolonged cases:

azithromycin 500 mg (o) 12 hourly for 3 days

or

ciprofloxacin 500 mg (o) 12 hourly for 3 days

or

norfloxacin 400 mg (o) 12 hourly for 5 days

***Cryptosporidium* species**

Usually self-limiting, may need fluid and electrolytes and antimotility agents.

If severe, nitazoxanide (shared care).

Amoebiasis (intestinal)

See [CHAPTER 129](#) .

metronidazole 600–800 mg (o) tds for 6–10 days

plus

diloxanide furoate 500 mg (o) tds for 10 days

***Blastocystis hominis* (a parasitic infection)**

Pathogenicity is disputed: give therapy only if severe. Associated with poor hygiene (travel, pets, dam/tank water, oysters).

metronidazole for 7 days

Specialist advice should be sought.

Treatment for special enteric infections

Typhoid/paratyphoid fever

See [CHAPTER 129](#) .

azithromycin 1 g (o) daily for 7 days

or

(if not acquired in the Indian subcontinent or South-East Asia)

ciprofloxacin 500 mg (o) 12 hourly for 7–10 days (use IV if oral therapy not tolerated)

If ciprofloxacin is contraindicated (e.g. in children) or not tolerated, then use:

ceftriaxone 3 g IV daily until culture and sensitivities available, then choose oral regimens

If severe: administer same drug and dosage IV for first 4–5 days.

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Cholera

Antibiotic therapy reduces the volume and duration of diarrhoea. Rehydration is the key.

azithromycin 1 g (child 20 mg/kg up to 1 g) (o) as a single dose

or

ciprofloxacin 1 g (o) as a single dose

For pregnant women and children:

amoxicillin (child: 10 mg/kg up to) 250 mg (o) 6 hourly for 4 days

Inflammatory bowel disease³

Two important disorders are ulcerative colitis (UC) and Crohn disease, which have equal sex incidence and can occur at any age, but onset peaks between 20 and 40 years.

Inflammatory bowel disease (IBD) should be considered when a young person presents with:

- bloody diarrhoea and mucus
- colonic pain and fever
- urgency to visit toilet and feeling of incomplete defecation
- constitutional symptoms including weight loss and malaise
- extra-abdominal manifestations such as arthralgia, low back pain (spondyloarthropathy), eye problems (iritocyclitis), liver disease and skin lesions (pyoderma gangrenosum, erythema nodosum)

Investigations include FBE, vitamin B12 and folate, LFTs (abnormal enzymes), HLA-B₂₇, faecal calprotectin (if normal, no intestinal inflammation; if abnormal, needs colonoscopy) and lactoferrin.

Ulcerative colitis

Clinical features

- Mainly a disease of Western societies
- Mainly in young adults (15–40 years)
- High-risk factors—family history, previous attacks, low-fibre diet
- Recurrent attacks of loose stools
- Blood, or blood and pus, or mucus in stools
- Abdominal pain slight or absent
- Fever, malaise and weight loss uncommon

- Begins in rectum (continues proximally)—affects only the colon: it usually does not spread beyond the ileocaecal valve
- An increased risk of carcinoma after 7–10 years

Main symptom

- Bloody diarrhoea

Diagnosis

- Faecal calprotectin: a sensitive test
- Proctosigmoidoscopy: a granular red proctitis with contact bleeding
- Barium enema: characteristic changes

Prognosis

- Mortality rates are comparable to the general population without UC¹⁰
- Recurrent attacks common

Crohn disease

Synonyms: regional enteritis, granulomatous colitis.

The cause is unknown but there is a genetic link.

Clinical features

- Recurrent diarrhoea in a young person (15–40 years)
- Blood and mucus in stools (less than UC)
- Colicky abdominal pain (small bowel colic)
- Right iliac fossa pain (confused with appendicitis)
- Constitutional symptoms (e.g. fever, weight loss, malaise, anorexia, nausea)
- Signs include perianal disorders (e.g. anal fissure, fistula, ischiorectal abscess), mouth ulcers
- Skip areas in bowel: $\frac{1}{2}$ ileocolic, $\frac{1}{4}$ confined to small bowel, $\frac{1}{4}$ confined to colon, 4% in upper GIT

Main symptom

- Colicky abdominal pain

Diagnosis

- Sigmoidoscopy: ‘cobblestone’ appearance (patchy mucosal oedema)
- Colonoscopy: useful to differentiate from UC
- Biopsy with endoscopy

Prognosis

- Less favourable than UC with both medical and surgical treatment.
- A 20-year Norway study showed a 1.3 times mortality risk compared to a matched population without Crohn disease.¹¹

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Management principles of both

- Education and support, including support groups
- Treat under consultant supervision
- Treatment of acute attacks depends on severity of the attack and the extent of the disorder:
 - mild attacks: manage out of hospital
 - severe attacks: hospital, to attend to fluid and electrolyte balance
- Role of diet controversial: consider a high-fibre diet but maintain adequate nutrition
- Pharmaceutical agents (the following can be considered):
 - 5-aminosalicylic acid derivatives (mainly UC): sulfasalazine (mainstay), olsalazine, mesalazine. Usually start with these agents
 - corticosteroids (mainly for acute flares): oral, parenteral, topical (rectal foam, suppositories or enemas)
 - for severe disease, immunomodifying drugs (e.g. azathioprine, cyclosporin, methotrexate) and anti-TNF and biological agents (e.g. adalimumab, vedolizumab, infliximab)
- Surgical treatment: reserve for complications; avoid surgery if possible

Alternating diarrhoea and constipation

Alternating diarrhoea and constipation are well-known symptoms of incomplete bowel obstruction (cancer of colon and diverticular disease) and irritable bowel syndrome.

Irritable bowel syndrome (IBS)^{3,9,12}

Clinical features

- Typically in younger women (21–40 years)
- Any age or sex can be affected
- May follow attack of gastroenteritis/traveller's diarrhoea
- Cramping abdominal pain (central or iliac fossa)—see FIGURE 34.4
- Pain usually relieved by passing flatus or by defecation
- Variable bowel habit (constipation more common)
- Diarrhoea usually worse in morning—several loose, explosive bowel actions with urgency
- The Bristol stool chart was devised to assist with the subclassification of stool types and bowel habits (see: www.continence.org.au/pages/bristol-stool-chart.html)
- Often precipitated by eating
- Faeces sometimes like small hard pellets or ribbon-like
- Anorexia and nausea (sometimes)
- Bloating, abdominal distension, borborygmi
- Tiredness common

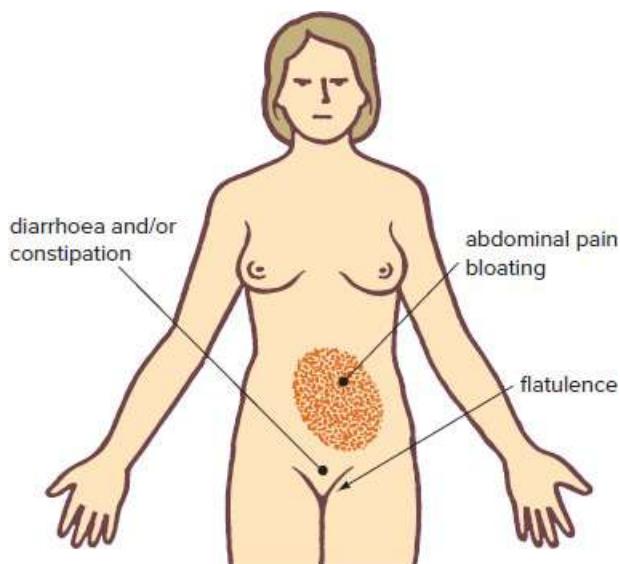


FIGURE 34.4 Classic symptoms of irritable bowel syndrome

The Rome III diagnostic criteria for IBS are presented in [TABLE 34.11](#).

Table 34.11 Rome III diagnostic criteria for irritable bowel syndrome^{*13}

In the preceding 3 months, the patient has had abdominal discomfort for at least 3 days per month with two of the following three features:

- relieved by defecation
- onset associated with a change in stool frequency
- onset associated with a change in form (appearance) of stool (loose, watery or pellet-like)

Symptoms that cumulatively support the diagnosis of irritable bowel syndrome:

- abnormal stool frequency (for research purposes may be defined as more than three bowel movements per day or fewer than three bowel movements per week)
- abnormal stool form (lumpy/hard or watery/mushy)
- abnormal stool passage (straining, urgency or feeling of incomplete evacuation)
- passage of mucus
- bloating or feeling of abdominal distension

Note: Red flags must be excluded

*in absence of structural or metabolic abnormalities to explain symptoms

IBS is a diagnosis of exclusion. A thorough physical examination, investigations (FBE, ESR and stool microscopy or culture) and colonoscopy are necessary. Insufflation of air at colonoscopy may reproduce the abdominal pain of IBS.

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Possible related causes

These include bowel infection, food irritation (e.g. spicy foods), lactose (milk) intolerance, excess-fibre wheat products, high fatty foods, carbonated drinks, laxative overuse, use of antibiotics and codeine-containing analgesics, psychological factors.

Management

The patient must be reassured and educated with advice that the problem will not cause malignancy or inflammatory bowel disease and will not shorten life expectancy. The basis of initial treatment is simple dietary modification (FODMAPs),¹⁴ exercise, fluids (2–3 L water daily) and non-fermentable fibre.

Red flag pointers for non-IBS disease¹⁵

- Age of onset >50 years
- Fever
- Unexplained weight loss
- Rectal bleeding
- Pain waking at night
- Persistent daily diarrhoea/steatorrhoea
- Recurrent vomiting
- Major change in symptoms
- Mouth ulcers
- ↑ CRP, ESR
- Anaemia
- Family history of bowel cancer or IBD

Self-help advice to the patient

Anyone with IBS should try to work on the things that make the symptoms worse. If you recognise stresses and strains in your life, try to develop a more relaxed lifestyle. You may have to be less of a perfectionist in your approach to life.

Focus on establishing a regular eating pattern. Try to avoid any foods that you can identify as causing the problem. You may have to cut out smoking and alcohol and avoid laxatives and codeine (in painkillers). A high-fibre (non-fermentable) and low-carbohydrate diet and 2–3 L of water a day may be the answer to your problem.

A low-FODMAP diet can produce good benefits.^{11,13,16} FODMAP refers to fermentable oligosaccharides, disaccharides, monosaccharides and polyols, which are poorly absorbed. All of these carbohydrates need to be eliminated (under a dietitian's guidance), then reintroduced one at a time.

See: www.med.monash.edu/cecs/gastro/fodmap, available as an app—the Monash University Low-FODMAP Diet.

Diverticular disorder

Diverticular disorder is a problem of the colon (90% in descending colon) and is related to lack of fibre in the diet. It is usually symptomless.

Clinical features

- Typical in middle-aged or elderly—over 40 years
- Increases with age
- Present in one in three people over 60 years (Western world)
- Diverticulosis—symptomless
- Diverticulitis—infected diverticula and symptomatic (refer [CHAPTER 24](#))
- Constipation or alternating constipation/diarrhoea
- Intermittent cramping lower abdominal pain in LIF
- Tenderness in LIF
- Rectal bleeding—may be profuse (\pm faeces)
- May present as acute abdomen or subacute obstruction
- Usually settles in 2–3 days

Complications (of diverticulitis)

- Bleeding—may cause massive lower GIT bleeding
- Abscess
- Perforation
- Peritonitis
- Obstruction (refer [CHAPTER 24](#))
- Fistula—bladder, vagina

Investigations

- WBC and ESR—to determine inflammation
- Sigmoidoscopy

- Barium enema

Management

- It usually responds to a high-fibre diet.
- Avoidance of constipation.

Advice to the patient

The gradual introduction of fibre with plenty of fluids (especially water) will improve any symptoms you may have and reduce the risk of complications. Your diet should include:

1. cereals, such as bran, shredded wheat, muesli or porridge
2. wholemeal and multigrain breads
3. fresh or stewed fruits and vegetables

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Bran can be added to your cereal or stewed fruit, starting with 1 tablespoon and gradually increasing to 3 tablespoons a day. Fibre can make you feel uncomfortable for the first few weeks, but the bowel soon settles with your improved diet.

When to refer

Children with diarrhoea

- Infant under 3 months
- Moderate to severe dehydration
- Diagnosis of diarrhoea and vomiting in doubt (e.g. blood in vomitus or stool, bile-stained vomiting, high fever or toxæmia, abdominal signs suggestive of appendicitis or obstruction)
- Failure to improve or deterioration
- A pre-existing chronic illness

Adults with diarrhoea

- Patient with chronic or bloody diarrhoea
- Any problem requiring colonoscopic investigation
- Patients with anaemia

- Patients with weight loss, abdominal mass or suspicion of neoplasia
- Patients with anal fistulae
- Patients not responding to treatment for giardiasis
- Infection with *E. histolytica*
- Long-term asymptomatic carrier of typhoid or paratyphoid fever
- Patient with persistent undiagnosed nocturnal diarrhoea
- Patients with IBS with a significant change in symptoms
- Patients with inflammatory bowel diseases with severe exacerbations, possibly requiring immunosuppressive therapy and with complications
- Patients with ulcerative colitis of more than 7 years' duration (screening by colonoscopy for carcinoma)

Practice tips

- Oral antidiarrhoeal drugs are contraindicated in children; besides being ineffective they may prolong intestinal recovery.
- Anti-emetics can readily provoke dystonic reactions in children, especially if young and dehydrated.
- Acute diarrhoea is invariably self-limiting (lasts 2–5 days). If it lasts longer than 7 days, investigate with culture and microscopy of the stools.
- If diarrhoea is associated with episodes of facial flushing or wheezing, consider carcinoid syndrome.
- Recurrent pain in the right hypochondrium is usually a feature of IBS (not gall bladder disease).
- Recurrent pain in the right iliac fossa is more likely to be IBS than appendicitis.
- Beware of false correlations or premature conclusions (e.g. attributing the finding of diverticular disorder on barium meal to the cause of the symptoms).
- Undercooked chicken is a common source of enteropathic bacterial infection.
- Consider alcohol abuse if a patient's diarrhoea resolves spontaneously on hospital admission.

Patient education resources

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

- Coeliac disease in adults
- Coeliac disease in children
- Cystic fibrosis
- Diarrhoea—acute diarrhoea in adults
- Diverticular disease
- Gastroenteritis in children
- Inflammatory bowel disease

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References

- 1 Bolin T, Riordan SM. Acute and persistent diarrhoea. *Current Therapeutics*, 2001; May: 47–57.
- 2 Cheng AC et al. Australasian Society for Infectious Diseases guidelines for the diagnosis and treatment of *Clostridium difficile* infection. *Med J Aust*, 2011; 194(7): 353–8.
- 3 Supportive measures of gastroenteritis [published 2016]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2016. www.tg.org.au, accessed October 2019.
- 4 Dalton C. Foodborne illness: how to treat. *Australian Doctor*, 15 April; 2005: 39–46.
- 5 Robinson MJ, Roberton DM. *Practical Paediatrics* (5th edn). Edinburgh: Churchill Livingstone, 2003: 675–90.
- 6 Oberklaid F. Management of gastroenteritis in children. In: *The Australian Paediatric Review*. Melbourne: Royal Children's Hospital, 1990: 1–2.
- 7 Freedman S et al. Effect of dilute apple juice and preferred fluids versus electrolyte maintenance solution on treatment failure among children with mild gastroenteritis: a randomized clinical trial. *JAMA*, 2016; 315(18): 1966–74.
- 8 Gwee A, Rimer R, Marks M. *Paediatric Handbook* (9th edn). Oxford: Wiley-Blackwell, 2015: 90–5.
- 9 Onwuezobe IA et al. Antimicrobials for treating symptomatic non-typhoidal *Salmonella*

infection (Cochrane Review). Cochrane Database Syst Rev, 2012; Issue 11: Art No. CD001167.

- 10** Manninen P et al. Mortality in ulcerative colitis and Crohn's disease: a population-based study in Finland. *J Crohns Colitis*, June 2012; 6(5): 524–8.
- 11** Hovde Ø et al. Mortality and causes of death in Crohn's disease: results from 20 years of follow-up in the IBSEN study. *Gut*, 2014; 63: 771–5.
- 12** NICE. Irritable bowel syndrome in adults: diagnosis and management of irritable bowel syndrome in primary care, 2008. Available from: www.nice.org.uk, accessed 25 May 2018.
- 13** Rome Foundation. Rome III diagnostic criteria for functional gastrointestinal disorders. *J Gastrointest Liver Dis*, 2006; 15(3): 307–12.
- 14** Gibson PR. Irritable bowel syndrome. *Australian Doctor*, 13 April 2012: 17–34.
- 15** Ellard K, Malcolm A. Irritable bowel syndrome. *Medical Observer*, 30 March 2007: 29–32.
- 16** Gibson PR, Shepherd SJ. Evidence-based dietary management of functional gastrointestinal symptoms: the FODMAP approach. *J Gastroenterol Hepatology*, 2010; Feb 25(2): 2528.

35 Dizziness/vertigo

I got my giddiness in 1690 (at the age of 23) by eating 100 golden pippins at a time at Richmond. Four years later at a place 20 miles further on in Surrey I got my deafness; and these two ‘friends’ have visited me one or other year since, and being old acquaintances have often sought fit to come together.

JONATHAN SWIFT (1667–1745), DESCRIBING HIS MÉNIÈRE SYNDROME

When patients complain of ‘dizziness’, they can be using this term to describe many different phenomena, and hence a careful history is required to unravel the problem. Others may use different terms, such as ‘giddiness’, ‘swimming in the head’, ‘my brain spinning’, ‘whirling’ and ‘swinging’.

‘Dizzy’ comes from an old English word, *dysig*, meaning foolish or stupid. Strictly speaking, it means unsteadiness or lightheadedness—without movement, motion or spatial disorientation.

‘Vertigo’, on the other hand, comes from the Latin *vertere* (to turn) and *-igo* for a condition. It should describe a hallucination of rotation of self or the surroundings in a horizontal or vertical direction.¹

The term ‘dizziness’, however, is generally used collectively to describe all types of equilibrium disorders and, for convenience, can be classified as shown in FIGURE 35.1 .

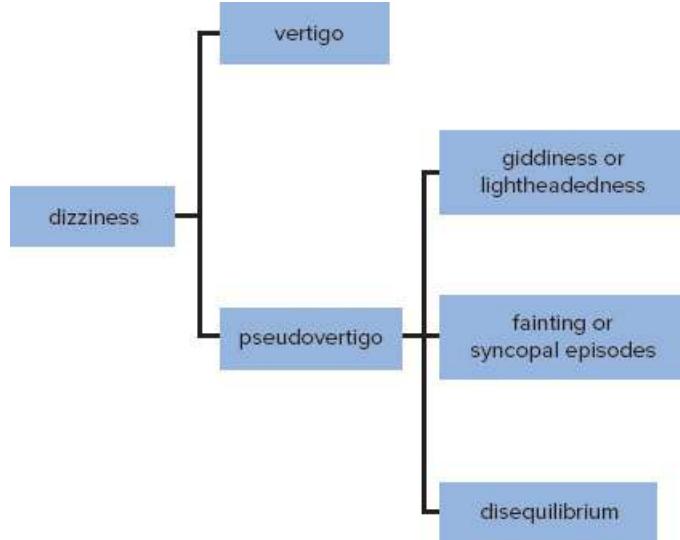


FIGURE 35.1 Classification of dizziness

Key facts and checkpoints

- Approximately one-third of the population will have suffered from significant dizziness by age 65 and about a half by age 80.²
- The commonest causes in family practice are postural hypotension and hyperventilation.
- The ability to examine and interpret the sign of nystagmus accurately is important in the diagnostic process.
- A drug history is very important, including prescribed drugs and others such as alcohol, cocaine, marijuana and illicit drugs.
- Ménière syndrome is overdiagnosed. It has the classic triad: vertigo–tinnitus–deafness (sensorineural).
- Vertebrobasilar insufficiency is also overdiagnosed as a cause of vertigo. It is a rare cause but may result in dizziness and sometimes vertigo but rarely in isolation.

Defined terminology

Vertigo²

Vertigo is defined as an episodic sudden sensation of circular motion of the body or of its surroundings or an illusion of motion, usually a rotatory sensation. Other terms used to describe this symptom include ‘everything spins’, ‘my head spins’, ‘the room spins’, ‘whirling’, ‘reeling’, ‘swaying’, ‘pitching’ and ‘rocking’. It is frequently accompanied by autonomic symptoms such as nausea, retching, vomiting, pallor and sweating.

Vertigo is characteristically precipitated by standing, by turning the head or by movement. Patients have to walk carefully and may become nervous about descending stairs or crossing the road, and usually seek support. Therefore, the vertiginous person is usually very frightened and tends to remain immobile during an attack and may feel their feet being lifted under them.

Patients may feel as though they are being impelled by some outside force that tends to pull them to one side, especially while walking.

True vertigo is a symptom of disturbed function involving the vestibular system or its central connections. It invariably has an organic cause. Important causes are presented in TABLE 35.1 , while FIGURE 35.2 illustrates central neurological centres that can cause vertigo. Some features of central vertigo include gait ataxia out of proportion to vertigo, diplopia, hemisensory loss, slurred speech, difficulty swallowing and abnormal eye movements. With peripheral vertigo, hearing loss, tinnitus, ear fullness and a positive head impulse test may be present.²

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Table 35.1 Causes of vertigo

Peripheral disorders

Labyrinth:

- labyrinthitis: viral or suppurative
- Ménière syndrome
- benign paroxysmal positional vertigo (BPPV)
- drugs
- trauma
- chronic suppurative otitis media

Eight nerve:

- vestibular neuritis
- acoustic neuroma
- drugs

Cervical vertigo

Central disorders

Brain stem (TIA or stroke):

- vertebrobasilar insufficiency
- infarction

Cerebellum:

- degeneration
- tumours

Migraine

Multiple sclerosis

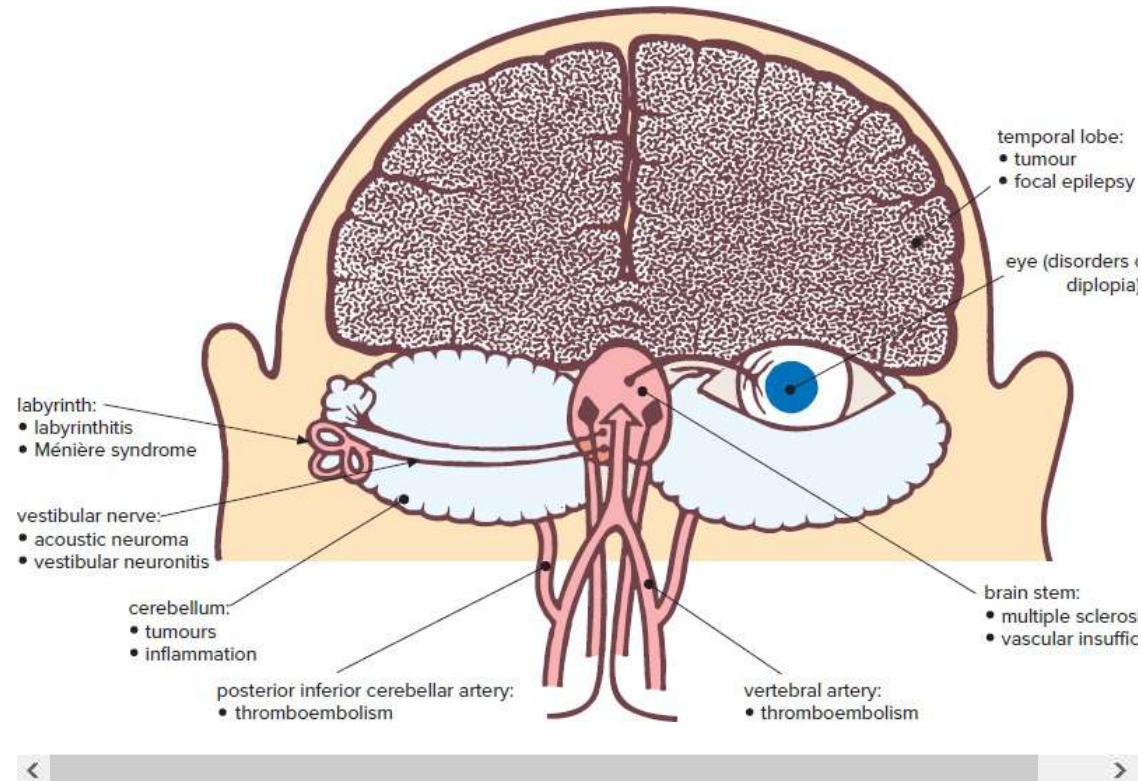


FIGURE 35.2 Diagrammatic illustration of central centres that can cause vertigo

Nystagmus is often seen with vertigo and, since 80–85% of causes are due to an ear problem, tinnitus and hearing disorders are also occasionally associated. In acute cases there is usually a reflex autonomic discharge producing sweating, pallor, nausea and vomiting.

Giddiness

Giddiness is a sensation of uncertainty or ill-defined lightheadedness. Other terms used include ‘a swimming sensation’, ‘walking on air’ and ‘ground going beneath me’. It usually contains no elements of rotation, impulsion, tinnitus, deafness, nausea or vomiting.

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The patient with giddiness, although fearful of falling or swooning, can nonetheless walk without difficulty if forced to do so.

Giddiness is also a typical psychoneurotic symptom.

Syncopal episodes

Syncope may present as a variety of dizziness or lightheadedness in which there is a sensation of impending fainting or loss of consciousness. Presyncope is a sensation of feeling faint. Common causes are cardiogenic disorders and postural hypotension, which are usually drug-induced.

Disequilibrium

Disequilibrium implies a condition in which there is a loss of balance or instability while walking, without any associated sensations of spinning. Other terms used to describe this include 'unsteadiness on feet', 'the staggers', 'swaying feeling' and 'dizzy in the feet'.

Disequilibrium is usually of neurogenic origin.

A diagnostic approach

A summary of the diagnostic strategy model is presented in [TABLE 35.2](#) .

Table 35.2 Dizziness/vertigo: diagnostic strategy model

Probability diagnosis

- Anxiety-hyperventilation (G)
- Postural hypotension (G/S)
- Simple faint—vasovagal (S)
- Acute vestibulopathy (V)
- Benign paroxysmal positional vertigo (V)
- Motion sickness (V)
- Vestibular migraine (V)
- Cervical dysfunction/spondylosis

Serious disorders not to be missed

Neoplasia:

- acoustic neuroma
- posterior fossa tumour
- other brain tumours, primary or secondary

Intracerebral infection (e.g. abscess)

Cardiovascular:

- arrhythmias
- myocardial infarction
- aortic stenosis

Cerebrovascular:

- vertebrobasilar insufficiency
- brain-stem infarct (e.g. PICA thrombosis)

Multiple sclerosis

Carbon monoxide poisoning

Pitfalls (often missed)

Ear wax (G)

Otosclerosis

Arrhythmias

Hyperventilation

Alcohol and other drugs

Cough or micturition syncope

Vestibular migraine/migrainous vertigo

Parkinson disease

Ménière syndrome (overdiagnosed)

Rarities:

- Addison disease ([CHAPTER 14](#))
- neurosyphilis
- autonomic neuropathy
- hypertension
- subclavian steal
- perilymphatic fistula
- Shy–Drager syndrome

Seven masquerades checklist

Depression

Diabetes (possible: hypo/hyper)

Drugs

Anaemia

Thyroid disorder (possible)

Spinal dysfunction

UTI (possible)

Is the patient trying to tell me something?

Very likely. Consider anxiety and/or depression.

Probability diagnosis

In medical school we gain the wrong impression that the common causes of dizziness or vertigo are the relatively uncommon causes, such as Ménière syndrome, aortic stenosis, Stokes–Adams attacks, cerebellar disorders, vertebrobasilar disease and hypertension. In the real world of medicine, one is impressed by how often dizziness is caused by relatively common benign conditions, such as hyperventilation associated with anxiety, simple syncope, postural hypotension due to drugs and old age, inner ear infections, wax in the ears, post head injury, motion sickness and alcohol intoxication. In most instances making the correct diagnosis (which, as ever, is based on a careful history) is straightforward, but finding the underlying cause of true vertigo can be very difficult.

The common causes of vertigo seen in general practice are benign paroxysmal positional vertigo (BPPV), accounting for about 25% of cases, acute vestibulopathy (vestibular neuritis) and vestibular migraine.

Viral labyrinthitis is basically the same as vestibular neuritis, except that the whole of the inner ear is involved so that deafness and tinnitus arise simultaneously with severe vertigo. The most common causes of recurrent spontaneous vertigo are vestibular migraine and Ménière syndrome.

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Serious disorders not to be missed

Neoplasia

The important serious disorders to keep in mind are space-occupying tumours, such as acoustic neuroma, medulloblastoma and other tumours (especially posterior fossa tumours) capable of causing vertigo, intracerebral infections and cardiovascular abnormalities.

It is important to bear in mind that the commonest brain tumour is a metastatic deposit from lung cancer.³

Red flags for dizziness/vertigo

- Neurological signs
- Ataxia out of proportion to vertigo
- Nystagmus out of proportion to vertigo
- Central nystagmus

- Central eye movement abnormalities

Acoustic neuroma

This uncommon tumour should be suspected in the patient presenting with the symptoms shown in the diagnostic triad below. Headache may occasionally be present.



DxT (unilateral) tinnitus + hearing loss + unsteady gait → acoustic neuroma

Diagnosis is best clinched by high-resolution MRI. Audiometry and auditory evoked responses are also relevant investigations.

Cardiac disorders

Cardiac disorders that must be excluded for giddiness or syncope are the various arrhythmias, such as Stokes–Adams attacks caused by complete heart block, aortic stenosis and myocardial infarction.

Cerebrovascular causes

The outstanding cerebrovascular causes of severe vertigo are vertebrobasilar insufficiency and brain-stem infarction. Vertigo is the commonest symptom of transient cerebral ischaemic attacks in the vertebrobasilar distribution.¹

Severe vertigo, often in association with hiccoughs and dysphagia, is a feature of the variety of brain-stem infarctions known as the lateral medullary syndrome due to posterior inferior cerebellar artery (PICA) thrombosis. There is a dramatic onset of vertigo with cerebellar signs, including ataxia and vomiting. There are ipsilateral cranial nerve (brain stem) signs with contralateral spinothalamic sensory loss of the face and body. Diagnosis is by CT or MRI scanning.

Neurological causes

Important neurological causes of dizziness are multiple sclerosis and complex partial seizures.

The lesions of multiple sclerosis may occur in the brain stem or cerebellum. Young patients who present with a sudden onset of vertigo with ‘jiggly’ vision but without auditory symptoms should be considered as having multiple sclerosis. Five per cent of cases of multiple sclerosis present with vertigo.

Pitfalls

A list of conditions causing dizziness that may be misdiagnosed is presented in TABLE 35.2 .

Wax in the ear certainly causes dizziness, though its mechanism of action is controversial. Cough and micturition syncope do occur, although they are uncommon.

Ménière syndrome is a pitfall in the sense that it tends to be overdiagnosed.

Seven masquerades checklist

Of these conditions, drugs and vertebral dysfunction (of the cervical spine) stand out as important causes. Depression demands attention because of the possible association of anxiety and hyperventilation.

Diabetes mellitus has an association through the possible mechanisms of hypoglycaemia from therapy or from an autonomic neuropathy.

Drugs

Drugs usually affect the vestibular nerve rather than the labyrinth. Drugs commonly associated with dizziness are presented in TABLE 35.3 .

Table 35.3 Drugs that can cause dizziness

Alcohol

Antibiotics: streptomycin, gentamicin, kanamycin, tetracyclines

Antidepressants

Anti-epileptics: phenytoin

Antihistamines

Antihypertensives

Aspirin and salicylates

Cocaine, cannabis

Diuretics in large doses: intravenous frusemide, ethacrynic acid

Glyceryl trinitrate

Quinine: quinidine

Tranquillisers: phenothiazines, phenobarbitone, benzodiazepines

Cervical spine dysfunction

It is not uncommon to observe vertigo in patients with cervical spondylosis or post cervical spinal injury. It has been postulated⁴ that this may be caused by the generation of abnormal impulses from proprioceptors in the upper cervical spine, or by osteophytes compressing the vertebral arteries in the vertebral canal. Some instances of BPPV are associated with disorders of the cervical spine.

Psychogenic considerations

This may be an important aspect to consider in the patient presenting with dizziness, especially if the complaint is giddiness or lightheadedness. An underlying anxiety, particularly agoraphobia and panic disorder, may be the commonest cause of this symptom in family practice and clinical investigation of hyperventilation may confirm the diagnosis. The possibility of depression must also be kept in mind.⁵ Many of these patients harbour the fear that they may be suffering from a serious disorder, such as a brain tumour or multiple sclerosis, or face an impending stroke or insanity. Appropriate reassurance to the contrary is often positively therapeutic for that patient.

The clinical approach

The essentials of the diagnostic approach include careful attention to the history and physical examination, and judicious selection of specific office tests and special investigations.

History

It is important to get patients to explain the precise nature of the symptoms, even asking their opinion as to the cause of their dizziness.

Key questions

The following questions should be addressed:

- Is it vertigo or pseudovertigo?
- Symptom pattern:
 - paroxysmal or continuous?
 - effect of position and change of posture?
- Any aural symptoms? Tinnitus? Deafness?
- Any visual symptoms?
- Any neurological symptoms?
- Any nausea or vomiting?
- Any symptoms of psychoneurosis?
- Any recent colds?
- Any recent head injury (even trivial)?

- Any drugs being taken?

alcohol?

marijuana?

hypotensives?

psychotropics?

other drugs?

Examination

A full general examination is appropriate with particular attention being paid to the cardiovascular and central nervous systems and the auditory and vestibular mechanisms.

Guidelines

Examination guidelines are:

1. ear disease:

- auroscopic examination: ?wax, ?drum
- hearing tests
- Weber and Rinne tests

2. the eyes:

- visual acuity
- test movements for nystagmus

3. cardiovascular system:

- evidence of atherosclerosis
- blood pressure: supine, standing, sitting
- cardiac arrhythmias

4. cranial nerves:

- 2nd, 3rd, 4th, 6th and 7th
- corneal response for 5th

- 8th—auditory nerve
- 5. the cerebellum or its connections:
 - gait
 - coordination
 - reflexes
 - Romberg test
 - finger–nose test: ?past pointing
- 6. the neck, including cervical spine
- 7. general search for evidence of:
 - anaemia
 - polycythaemia
 - alcohol dependence

Office tests for dizziness

- Ask the patient to perform any manoeuvre that may provoke the symptom.
- Carry out head positional testing to induce vertigo and/or nystagmus (e.g. Hallpike manoeuvre in FIG. 35.3 or head impulse test/head thrust). Avoid if prominent spontaneous vertigo and nystagmus.
- The 3-part HINTS test (head impulse, nystagmus, test of skew) is useful for distinguishing the cause of vertigo as either central (urgent) or peripheral. Page 434
- Take blood pressure measurements in three positions.
- Perform forced hyperventilation (20–25 breaths per minute) for 2 minutes.
- Carry out palpation of carotid arteries and carotid sinus (with care).

Investigations

Appropriate laboratory tests should be selected from TABLE 35.4 .

Investigations (select only if indicated)

Table 35.4

Haemoglobin
Blood glucose
ECG: ?Holter monitor
Audiometry
Brain-stem evoked audiometry
Caloric test
Visual evoked potentials (MS)
Electrocochleography
Electro-oculography (electronystagmography)
Rotational tests
Radiology:

- chest X-ray (?bronchial carcinoma)
- cervical spine X-ray
- CT scan
- MRI (the choice to locate acoustic neuroma or other tumour—may detect MS and vascular infarction)

Diagnostic guidelines

- A sudden attack of vertigo in a young person following a recent URTI is suggestive of vestibular neuritis.
- Dizziness is a common symptom in menopausal women and is often associated with other features of vasomotor instability.
- Phenytoin therapy can cause cerebellar dysfunction.
- Postural and exercise hypotension are relatively common in the older atherosclerotic patient.
- Acute otitis media does not cause vertigo but chronic otitis media can, particularly if the patient develops a cholesteatoma, which then erodes into the internal ear causing a perilymphatic fistula.

Dizziness in children

Dizziness is not a common symptom in children. Vertigo can have sinister causes and requires referral because of the possibility of tumours, such as a medulloblastoma. A study by Eviatar⁶ of vertigo in children found that the commonest cause was a seizure focus particularly affecting the temporal lobe. Other causes included psychosomatic vertigo, vestibular migraine and vestibular neuritis.

Apart from the above causes it is important to consider:

- infection (e.g. meningitis, meningoencephalitis, cerebral abscess)
- trauma, especially to the temporal area
- middle-ear infection
- labyrinthitis (e.g. mumps, measles, influenza)
- BPPV (short-lived attacks of vertigo in young children between 1 and 4 years of age: tends to precede adulthood migraine)⁷
- hyperventilation
- drugs—prescribed
- illicit drugs (e.g. cocaine, marijuana)
- cardiac arrhythmias
- alcohol toxicity

A common trap is the acute effect of alcohol in curious children who can present with the sudden onset of dizziness.

‘Dizzy turns’ in girls in late teens

- These are commonly due to blood pressure fluctuations.
- Give advice related to reducing stress, lack of sleep and excessive exercise.
- Reassure that it settles with age (rare after 25 years).

Dizziness in the elderly

Dizziness is a relatively common complaint of the elderly. Common causes include postural hypotension related mainly to drugs prescribed for hypertension or other cardiovascular problems. Cerebrovascular disease, especially in the areas of the brain stem, is also relevant in this age group. True vertigo can be produced simply by an accumulation of wax in the external auditory meatus, being more frequent than generally appreciated.

Middle-ear disorder is also sometimes the cause of vertigo in an older person but disorder of the auditory nerve, inner ear, cerebellum, brain stem and cervical spine are common underlying factors.

Malignancy, primary and secondary, is a possibility in the elderly. The possibility of cardiac

‘Dizzy turns’ in elderly women

If no cause such as hypertension is found, advise them to get up slowly from sitting or lying, and to wear firm elastic stockings.

Common general conditions

⌚ Acute vestibulopathy (vestibular failure)

Causes:

- vestibular neuritis
- stroke—AICA or PICA

Vestibular neuritis covers both vestibular neuronitis and labyrinthitis, which are considered to be a viral infection of the vestibular nerve and labyrinth respectively, causing a prolonged attack of vertigo that can last for several days and be severe enough to require admission to hospital.⁸ This is more likely with labyrinthitis.

It is analogous to a viral infection of the 7th nerve causing Bell palsy. The attack is similar to Ménière syndrome except that there is no hearing disturbance.



DxT acute vertigo + nausea + vomiting → vestibular neuronitis



DxT same symptoms + hearing loss ± tinnitus → acute labyrinthitis

Characteristic features

- Single attack of vertigo without tinnitus or deafness
- Usually preceding ‘flu-like’ illness
- Mainly in young adults and middle age
- Abrupt onset with vertigo, ataxia, nausea and vomiting
- Generally lasts days to weeks
- Examination shows lateral or unidirectional nystagmus—rapid component away from side of

lesion (no hearing loss)

- Caloric stimulation confirms impaired vestibular function

It is basically a diagnosis of exclusion.

Treatment

- Rest in bed, lying very still
- Gaze in the direction that eases symptoms

The following drugs can be used for the first 2 days (see TABLE 35.5):

prochlorperazine (Stemetil) 5–10 mg (o) 6–8 hrly or 12.5 mg IM (if severe vomiting), but may slow recovery

or

promethazine 10–25 mg IM or slow IV, then 10–25 mg (o) for 48 hrs

or

ondansetron 4–8 mg (o) 2–3 hrly

or (recommended as best)

diazepam (which decreases brain-stem response to vestibular stimuli)² 5–10 mg IM for the acute attack (care with respiratory depression), then 5 mg (o) tds for 2–3 days

Table 35.5 Symptomatic relief of acute vertigo:
pharmaceutical options¹⁰

Anti-emetics:

- prochlorperazine
- metoclopramide
- ondansetron

Antihistamines:

- promethazine
- betahistine

Benzodiazepines (short period use for vertigo):

- diazepam
- lorazepam

A short course of corticosteroids often promotes recovery (e.g. prednisolone 1 mg/kg (up to 100 mg) orally daily in morning for 5 days, then taper over 15 days and cease).^{1,9}

Outcome

Both are self-limiting disorders and usually settle over 5–7 days or several weeks. Labyrinthitis usually lasts longer and during recovery rapid head movements may bring on transient vertigo.

Benign paroxysmal positional vertigo

BPPV is a common type of acute vertigo that is induced by changing head position—particularly tilting the head backwards, changing from a recumbent to a sitting position or turning to the affected side.

Features

- Affects all ages, especially the elderly
- The female to male ratio is 2:1
- Recurs periodically for several days
- Each attack is brief, usually lasts 10–60 seconds and subsides rapidly
- Severe vertigo on getting out of bed
- Can occur on head extension and turning head in bed
- Attacks are not accompanied by vomiting, tinnitus or deafness (nausea may occur)
- In one large series 17% were associated with trauma, 15% with viral labyrinthitis, while about 50% had no clear predisposing factor other than age. One accepted theory of causation is that fine pieces of floating crystalline calcium carbonate deposits (otoconia) that are loose in the labyrinth settle in the posterior semicircular canal and generate endolymphatic movement.¹¹ It may also be a variation of cervical dysfunction.
- Diagnosis is confirmed by head position testing: head impulse (head thrust) test or Hallpike manoeuvre (refer to YouTube for these manoeuvres).¹² In the latter, from a sitting position, the patient's head is rapidly taken to a head-hanging position 30° below the level of the couch —do three times, with the head (1) straight, (2) rotated to the right, (3) rotated to the left. Hold on for 30 seconds and observe the patient carefully for vertigo and nystagmus. There is a latent period of a few seconds before the onset of the symptoms—see FIGURE 35.3 .
- Tests of hearing and vestibular function are normal

- There is usually spontaneous recovery in weeks (most return to regular activity after 1 week)
- Recurrences are common: attacks occur in clusters

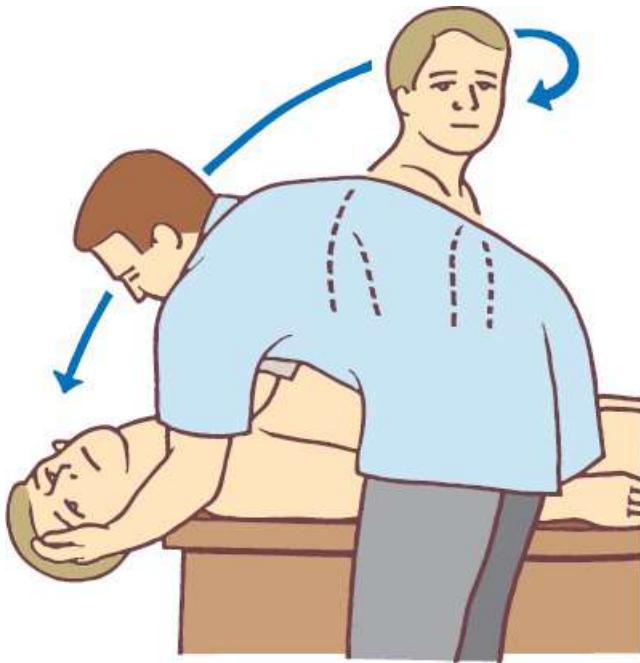


FIGURE 35.3 Hallpike manoeuvre: positional testing for benign paroxysmal positional vertigo (head rotated to 45° then taken rapidly from a sitting position to a hanging position). Repeat with head turned to the opposite side. A positive response is the onset of symptoms ± nystagmus with the affected ear lowermost.

Management

- Give appropriate explanation and reassurance
- Avoidance measures: encourage the patient to move in ways that avoid the attack
- Drugs are not recommended—usually ineffective
- Special exercises
- Cervical traction may help

Particle repositioning manoeuvres

Patient-performed exercises. Most patients appear to benefit from exercise, such as the Brandt–Daroff procedure¹³ or the Cawthorne–Cooksey exercises¹⁰ that consist essentially of repeatedly inducing the symptoms of vertigo. Rather than resorting to avoidance measures, the patient is

instructed to perform positional exercises to induce vertigo, hold this position until it subsides, and repeat this many times until the manoeuvre does not precipitate vertigo. The attacks then usually subside in a few days.

Therapist-performed exercises. Physical manoeuvres performed as an office procedure include the Epley and Semont manoeuvres, which aim to dislodge otoliths from a semicircular canal. The Epley manoeuvre has a high (77%) success rate on the initial attempt and up to 100% on further attempts.²

Surgical treatment

Rarely surgical treatment is required; it involves occlusion of the posterior semicircular canal rather than selective neurectomy.

Ménière syndrome

This is caused by a build-up of endolymph.

- It is an uncommon condition which is commonest in the 30–50 years age group.
- It is characterised by paroxysmal attacks of vertigo, tinnitus, nausea and vomiting, sweating and pallor, deafness (progressive).
- Onset is abrupt—patient may fall and then be bedridden for 1–2 hours. Patient doesn't like moving head.
- Attacks last 30 minutes to several hours.
- There is a variable interval between attacks (twice a month to twice a year).
- Nystagmus is observed only during an attack (often to side opposite affected inner ear).
- Examination:
 - sensorineural deafness (low tones)
 - caloric test: impaired vestibular function
 - audiometry: sensorineural deafness, loudness recruitment
 - special tests
- There are characteristic changes in electrocochleography.

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DxT vertigo + vomiting + tinnitus + sensorineural deafness → Ménière syndrome

Treatment

The aim is to reduce endolymphatic pressure by reducing the sodium and water content of the endolymph.

Prophylaxis

hydrochlorothiazide 25 mg (o) daily or with triamterene or amiloride combination (o) once daily.

Acute attack^{1,14}

- Anticipation of attack (fullness, tinnitus):

prochlorperazine 25 mg suppository

Treatment:

For severe attack, diazepam 5 mg IV ± prochlorperazine 12.5 mg IM, or if episodic, a diuretic, e.g. hydrochlorothiazide 25 mg (o) daily

Long term

- Reassurance with a careful explanation of this condition to the patient, who often associates it with malignant disease
- Excess intake of salt, tobacco and coffee to be avoided
- A low-salt diet is the mainstay of treatment (<3 g per day)
- Alleviate abnormal anxiety by using stress management, meditation or possibly long-term sedation (fluid builds up with stress)
- Referral for neurological assessment
- Diuretic (e.g. hydrochlorothiazide 50 mg/amiloride 5 mg once daily)—check electrolytes regularly

Surgery may be an option for intractable cases.

⌚ Vestibular migraine (migrainous vertigo)¹

Migraine is a relatively common cause of vertigo (up to 25% association) and often unrecognised because of its many guises. It should be strongly suspected if there is a past and/or family history of migraine and also where there is a history of recurrent bouts of spontaneous vertigo or ataxia that persist for hours or days in the absence of aural symptoms.¹⁵ Vertigo, which is usually not violent, may take the place of the aura that precedes the headache or may be a migraine

equivalent whereby the vertigo replaces the symptoms of headache, which may be an inconspicuous feature in some cases. Nausea and vomiting may be present. Pizotifen or propranolol are recommended for prophylaxis and a triptan for an attack.¹

When to refer¹⁶

- Vertigo of uncertain diagnosis, especially in children
- Possibility of tumour or bacterial infection
- Vertigo in presence of suppurative otitis media despite antibiotic therapy
- Presumed viral labyrinthitis not abating after 3 months
- Vertigo following trauma
- Presumed Ménière syndrome, not responding to conservative medical management
- Evidence of vertebrobasilar insufficiency
- Other neurological symptoms (including headache) and signs
- BPPV persisting for more than 12 months despite treatment with particle repositioning exercises

Practice tips

- A careful drug history often pinpoints the diagnosis.
- Always consider cardiac arrhythmias as a cause of acute dizziness.
- Consider phenytoin as a cause of dizziness.
- If an intracerebral metastatic lesion is suspected, consider the possibility of carcinoma of the lung as the primary source.
- Three important office investigations to perform in the evaluation are blood pressure measurement (lying, sitting and standing), hyperventilation and head positional testing (or HINTS test).
- Cervical vertigo is common and appropriate cervical mobilisation methods, with care, have been shown to be beneficial in a systematic review.¹⁷
- BPPV is also common and prescribing a set of exercises to desensitise the labyrinth is recommended. Use either the Brandt–Daroff procedure or the Cawthorne–Cooksey program.¹¹

- Think of migraine particularly in a younger patient presenting with unexplained recurrent vertigo.

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Patient education resources

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

- Labyrinthitis
- Ménière syndrome
- Vertigo: benign positional vertigo
- Vertigo: exercises for benign positional vertigo

References

- 1 Vestibular neuritis [published 2017]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2017. www.tg.org.au, accessed September 2017.
- 2 Dommaraju S, Perera E. An approach to vertigo in general practice. *Aus Fam Physician*, 2016; 45(4): 190–4.
- 3 Kuo C-H, Pang L, Chang R. Vertigo: assessment in general practice. *Aust Fam Physician*, 2008; 37: 341–7.
- 4 Lance JW. *A Physiological Approach to Clinical Neurology*. London: Butterworths, 1970: 162–79.
- 5 Paine M. Dealing with dizziness. *Australian Prescriber*, 2005; 28: 94–7.
- 6 Eviatar L, Eviatar A. Vertigo in children: differential diagnosis and treatment. *Paediatrics*, 1977; 59: 833–7.
- 7 Tunnessen WW Jr. *Signs and Symptoms in Paediatrics*. Philadelphia: Lippincott, 1988: 591–4.
- 8 Waterson J. Dizziness: how to treat. *Australian Doctor*, 7 March; 2003: 1–8.
- 9 Strupp M et al. Methylprednisolone, valacyclovir or the combination for vestibular neuritis. *N Engl J Med*, 2004; 351: 354–61.
- 10 Hain TC, Yacovino D. Pharmacologic treatment of persons with dizziness. *Neurol Clin*, 2005; 23: 831–53.

- 11** Brandt T, Daroff DB. Physical therapy for BPPV. *Arch Otolaryngol*, 1980; 106: 484–5.
- 12** Kuo CH, Pang L, Chang R. Vertigo—part 1—assessment in general practice. *Aust Fam Physician*, 2008; 37(5): 341–7.
- 13** Froehling IA et al. The canalith repositioning procedure for BPPV: a randomised controlled trial. *Mayo Clin Proc*, 2000; 75: 695–700.
- 14** Tonkin JP. Meniere’s disease. *Current Therapeutics*, 1995; 36: 39–43.
- 15** Pohl D. Vertigo. In: *MIMS Disease Index* (2nd edn). Sydney: IMS Publishing, 1996: 568–71.
- 16** Matthews T. Peripheral vertigo in general practice. *Cont Med Education*, 2006; 33: 267–70.
- 17** Yaseen K et al. The effectiveness of manual therapy in treating cervicogenic dizziness: a systematic review. *J Phys Ther Sci*, 2018; 30(1): 96–102.

36 Dyspepsia (indigestion)

Half the patients who get you up in the middle of the night and think they are dying are suffering from wind!

FRANCIS YOUNG (1884–1954), ADVICE TO A YOUNGER DOCTOR

Dyspepsia or indigestion is a difficult, sometimes vague, symptom to define or evaluate and requires very careful questioning to clarify the exact nature of the complaint.

Dyspepsia embraces the following:

- nausea
- heartburn/regurgitation
- upper abdominal discomfort
- lower chest discomfort
- acidity
- epigastric fullness, bloating or unease
- abdominal distension

The discomfort can sometimes amount to pain. Diagnoses to consider in dyspepsia are summarised in [TABLE 36.1](#) .

Table 36.1 Diagnoses to consider in dyspepsia¹

Gastrointestinal disorders

Gastro-oesophageal reflux, including hiatus hernia

Functional (non-ulcer) dyspepsia

Oesophageal motility disorders (dysmotility)
Peptic ulcer
Upper GIT malignancies (e.g. oesophagus, stomach, pancreas)
Hepatobiliary disease (e.g. hepatitis, biliary dyskinesia, cholelithiasis)
Pancreatitis
Upper GIT inflammation

- oesophagitis
- gastritis
- duodenitis

Irritable bowel syndrome

Non-gastrointestinal disorders

Myocardial ischaemia

Drug reaction

Alcohol effect

Somatisation

Anxiety/stress

Depression

Glossary of terms

Dyspepsia Pain or discomfort centred at the upper abdomen that is chronic or recurrent in nature.

Flatulence Excessive wind. It includes belching, abdominal bloating or passing excessive flatus.

Heartburn A central retrosternal or epigastric burning sensation that spreads upwards to the throat.

Flatulence

Excessive belching

- Usually functional

- Organic disease uncommon
- Due to air swallowing (aerophagy)
- Common in anxious people who gulp food and drink
- Associated hypersalivation

Management tips

- Make patient aware of excessive swallowing
- Avoid fizzy (carbonated) soft drinks
- Avoid chewing gum
- Don't drink with meals
- Don't mix proteins and starches
- Eat slowly and chew food thoroughly before swallowing
- Eat and chew with the mouth closed

If persistent: simethicone preparation (e.g. Mylanta II, Phazyme).

Excessive flatus

Flatus arises from two main sources:

- swallowed air
- bacterial fermentation of undigested carbohydrate

Exclude:

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- malabsorption
- irritable bowel syndrome
- anxiety → aerophagy
- drugs, especially lipid-lowering agents
- lactose intolerance

Management

- Assess diet (e.g. high fibre, beans and legumes, cabbage, onions, grapes and raisins)
- Avoid drinking with eating, especially with leafy vegetables
- Cook vegetables thoroughly
- Trial a lactose-free diet
- Consider simethicone preparations (e.g. De-Gas)

Key facts and checkpoints

- Dyspepsia or indigestion is a common complaint; 80% of the population will have experienced it at some time.
- Consider heartburn as ischaemic heart disease until proved otherwise.
- The presence of oesophagitis is suggested by pain on swallowing hot or cold liquids (odynophagia).
- Not all reflux is due to hiatus hernia.
- Many of those with hiatus hernia do not experience heartburn.
- All dysphagia must be investigated to rule out malignancy.
- Each year, 1–2 people per 1000 have a diagnosed peptic ulcer (PU).²
- The major feature of PU disease is epigastric pain.
- The pain of duodenal ulcer (DU) classically occurs at night.
- At any time, 10–20% of chronic NSAIDs users have peptic ulceration.³
- NSAIDs and *Helicobacter pylori* infection are the most important risk factors for upper GIT disease.
- NSAIDs mainly cause gastric ulcers (GU, gastric antrum and prepyloric region), with the duodenum affected to a lesser extent.
- Dyspeptic symptoms correlate poorly with NSAID-associated ulcer.

A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 36.2 .

Table 36.2 Dyspepsia: diagnostic strategy model

Probability diagnosis

Irritable upper GIT (functional dyspepsia)
Gastro-oesophageal reflux
Drugs
Oesophageal motility disorder (e.g. gastroparesis)

Serious disorders not to be missed

Neoplasia:

- cancer: stomach, pancreas, oesophagus

Cardiovascular:

- ischaemic heart disease
- congestive cardiac failure

Pancreatitis
Peptic ulcer (PU) disease

Pitfalls (often missed)

Myocardial ischaemia
Food allergy (e.g. lactose intolerance)
Pregnancy (early)
Hepatobiliary disease
Other gall bladder disease
Post vagotomy
Achalasia
Duodenitis
Autoimmune gastritis

Rarities:

- hyperparathyroidism
- mesenteric ischaemia
- Zollinger–Ellison syndrome
- kidney failure
- scleroderma

Seven masquerades checklist

Depression
Diabetes (rarely)
Drugs

Is this patient trying to tell me something?

Anxiety and stress are common associations of which patients are often unaware.
Consider irritable bowel syndrome; somatisation.

It is best to consider dyspepsia as:

- ulcer-like—localised pain
- dysmotility-like—diffuse discomfort, feeling full after meals (early satiety), nausea, bloating
- acid-reflux-like—indigestion or heartburn with acid reflux or regurgitation

The ulcer-like category may be due to an ulcer, and if not is termed functional (non-ulcer) dyspepsia.

Pitfalls

Perhaps the most common serious mistake is to attribute the discomfort of myocardial ischaemia to a disorder of the GIT. A sense of fullness or pressure in the epigastrium can certainly accompany ischaemia.

General pitfalls

- Reflux oesophagitis and PU can mimic ischaemic heart disease.
- Overlooking gastric cancer as a cause of dyspepsia.
- Failing to stress that weight reduction to ideal level will generally alleviate gastro-oesophageal reflux.
- Overlooking drugs as a cause (see TABLE 36.3).

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Table 36.3 Drugs that may cause dyspepsia

Alcohol
Anticholinergics
Aspirin
Bisphosphonates, esp. alendronate
Calcium-channel blockers
Corticosteroids
Digitalis
Lipid-lowering agents

Narcotics
Nicotine
NSAIDs
Potassium supplements (slow release)
Tetracycline; erythromycin
Theophylline
Tricyclic antidepressants

The clinical approach

History

It is worthwhile spending some time clarifying the exact nature of the presenting complaint: what the patient means by ‘indigestion’ or ‘heartburn’.¹ The relationship of the symptom to eating is very important, and whether it occurs after each meal or after specific meals.

In particular, care should be taken to consider and perhaps exclude ischaemic heart disease.

Key questions

- How would you describe the discomfort?
- Can you show me exactly where it is and where it radiates?
- What makes your discomfort worse?
- What relieves your discomfort?
- What effect do food, milk and antacids have?
- What effect do coffee, onions or garlic have?
- What effect does a big meal have?
- What about drinking alcohol? Wine?
- What effect does exercise have?
- Do fried or fatty foods make it worse?
- Do hot spicy foods affect it?
- Does the problem come on at night soon after you go to bed?

- Does it wake you up at night?
- Does bending over (e.g. gardening) make it worse?
- Do you have periods of freedom from the problem?
- Are you under a lot of stress or have a lot of worry?
- Do you go flat out all day?
- Do you rush your meals?
- Do you chew your food properly?
- What drugs or medicines do you take?
- How much alcohol do you have? Do you smoke?
- Have you noticed anything else when you have the problem?
- Do you get constipated or have diarrhoea?
- Have you lost weight recently?
- Do you feel the discomfort between your shoulder blades, or in your shoulders or throat?
- Do you experience shortness of breath, syncope or dizziness?

Symptoms analysis

Site and radiation

The site and radiation of pain or discomfort can provide a lead to the diagnosis. Refer to FIGURE 30.9 in CHAPTER 30. If it is felt in the interscapular area, consider oesophageal spasm, gall bladder disease or a DU. Retrosternal discomfort indicates oesophageal disorders or angina, while epigastric discomfort suggests disorders of the biliary system, stomach and duodenum.

Character of the pain

There tends to be considerable overlap in the character of the pain from the various disorders but some general characteristics apply:

- burning pain → gastro-oesophageal reflux (GORD)
- constricting pain → ischaemic heart disease or oesophageal spasm
- deep gnawing pain → PU

- heavy ache or ‘killing’ pain → psychogenic pain

Aggravating and relieving factors

Examples of these factors include:

- eating food may aggravate a GU but relieve a DU
- eating fried or fatty foods will aggravate biliary disease, functional dyspepsia and oesophageal disorders
- bending will aggravate GORD
- alcohol may aggravate GORD, oesophagitis, gastritis, PU, pancreatitis

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Associated symptoms

Relevant examples:

- difficulty in swallowing → oesophageal disorders
- lump or constriction in throat → psychogenic
- acid regurgitation → GORD, oesophagitis
- anorexia, weight loss → stomach cancer
- water brash → GORD, hiatus hernia, PU
- symptoms of anaemia → chronic oesophagitis or gastritis, PU, cancer (stomach, colon)
- flatulence, belching, abnormal bowel habits → irritable bowel syndrome
- diarrhoea 30 minutes after meal → mesenteric ischaemia

Examination

The physical examination does not often provide the key to the diagnosis but it is important to perform very careful palpation and inspection. Look for evidence of clinical anaemia and jaundice. Diffuse mild abdominal tenderness and a pulsatile abdominal aorta (especially in thin people) are common findings but do not necessarily discriminate between organic and functional problems. Specific epigastric tenderness suggests peptic ulceration while tenderness over the gall bladder area (Murphy sign) indicates gall bladder disease. An epigastric mass indicates stomach cancer.

Investigations

Do not overinvestigate. Investigations tend to be unrewarding in most instances of dyspepsia and could be postponed if the history is suggestive of a functional cause and the symptoms are not severe.¹ A trial of treatment such as changing adverse lifestyle factors, dietary modification and antacids could be the initial approach. Age is important in determining the extent of investigations, which are more relevant in those over 40 years.

The investigation of choice is endoscopy, which is superior to barium studies in investigation of the upper GIT. Gastroscopy is indicated for the *alarm* symptoms (see [Red flags box](#)).

***Helicobacter pylori* tests⁴**

Helicobacter pylori has been proved to cause ulcers. Most DUs and about two-thirds of GUs have been attributed to *H. pylori* infection.

Red flags

- Abnormal symptoms of reflux/dyspepsia
- Change of symptoms
- Dysphagia
- Anorexia
- Unexplained weight loss
- GIT bleeding (melaena, haematemesis)
- Pain radiating to back
- Pain waking at night
- Abnormal signs on examination
- Other tests: fasting serum gastrin (?hypersecretion)

Non-invasive tests:

- serological—IgG antibodies (sensitivity 85–90%, specificity 90–99%); excellent for diagnosis, not for follow-up; affected by PPIs—may be negative
- urea breath test (high sensitivity 97% and specificity 96%), good for follow-up
- stool antigen test (sensitivity 96%, specificity 95%)

Invasive tests:

- gastric mucosal biopsy during endoscopy can detect *H. pylori* through histology (gold standard) or rapid urease testing or *H. pylori* culture

Dyspepsia in the elderly

An organic disorder is more likely in the older patient, in whom it is important to consider stomach cancer. Symptoms such as anorexia, vomiting and weight loss point to such a problem.

Other conditions causing dyspepsia that are more prevalent in this age group are:

- constipation
- mesenteric artery ischaemia
- congestive cardiac failure

Dyspepsia in children

Dyspepsia is an uncommon problem in children but can be caused by drugs, cow's milk protein allergy, oesophageal disorders and gastro-oesophageal reflux in particular.⁵ Reflux can be considered to be physiological or pathological.

Gastro-oesophageal reflux

Regurgitation of feeds because of gastro-oesophageal reflux is a common physiological event in newborn infants. A mild degree of reflux is normal in babies, especially after they burp; this condition is called *posseting*.

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Symptoms

Milk will flow freely from the mouth soon after feeding, even after the baby has been put down for a sleep. Sometimes the flow will be forceful and may even be out of the nose.

Despite this vomiting or regurgitation, the babies are usually comfortable and thrive. Some infants will cry, presumably because of heartburn.⁵

In a small number the reflux may be severe enough (pathological) to cause serious problems such as oesophagitis with haematemesis or anaemia, stricture formation, failure to thrive, apnoea and aspiration.

Prognosis

Reflux gradually improves with time and usually ceases soon after solids are introduced into the diet. Most cases clear up completely by the age of 9 or 10 months, when the baby is sitting. At

12 months, only 5% have symptoms. Severe cases tend to persist until 18 months of age.

Investigations

These are not necessary in most cases but in those with persistent problems or complications referral to a paediatrician is recommended. The specialist investigations include barium meal with cine scanning, oesophageal pH monitoring or endoscopy and biopsy.

Management

Appropriate reassurance with parental education is important. It should be pointed out that changes in feeding practice and positioning will control most reflux.

The infant should be placed on the left side for sleeping with the head of the cot elevated about 20–30 degrees. The old method of placing the child in a bucket is no longer considered acceptable!

Smaller, more frequent feeds and thickening agents such as corn flour are appropriate.

Thickening of feeds

In infants under 6 months of age with confirmed, significant reflux, giving thickened formula feeds moderately decreases occurrences of regurgitation and parent-reported symptoms, and improves weight gain compared with non-thickened feeds.⁶

Bottle-fed babies (powdered milk formula):

Carobel: Add slightly less than 1 full scoop per bottle.

Gaviscon: Mix slightly less than ½ teaspoon of Infant Gaviscon Powder with 120 mL of formula in the bottle.

Cornflour (maize based): Mix 1 teaspoon with each 120 mL of formula.

Prethickened formulas, e.g. Karicare and S26 AR: Easier to use but more expensive.

Breastfed babies:

Carobel: Add slightly less than 1 full scoop to 20 mL cool boiled water or 20 mL expressed breast milk and give just before the feed.

Gaviscon: Mix slightly less than ½ teaspoon of Infant Gaviscon Powder with 20 mL cool boiled water or expressed breast milk and give just after the feed.

For persistent or complicated reflux, including painful oesophagitis, specialist-monitored treatment will include the use of antacids and H₂-receptor blocking agents (e.g. ranitidine).⁷

Dyspepsia in adults

Gastro-oesophageal reflux disease (GORD)^{8,9} in adults

Clinical features

- Nausea
- Bloating and belching
- Heartburn
- Acid regurgitation, especially lying down at night
- Water brash (mouth fills with saliva)
- Nocturnal cough with possible asthma-like symptoms
- Diagnosis usually made on history
- Investigation usually not needed (reserve for alarm features as described in the red flag box and non-responsive treatment)

Red flag pointers for upper GIT endoscopy

- Anaemia (new onset)
- Dysphagia
- Odynophagia (painful swallowing)
- Haematemesis or melaena
- Unexplained weight loss > 10%
- Vomiting
- Older age >50 years
- Chronic NSAID use
- Severe frequent symptoms
- Family history of upper GIT or colorectal cancer
- Short history of symptoms

- Unresponsive *H. pylori* treatment
- Barrett oesophagus screening in high-risk patients

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Complications

- Oesophagitis ± oesophageal ulcer
- Iron-deficiency anaemia
- Oesophageal stricture
- Respiratory: chronic cough, asthma, hoarseness
- Barrett oesophagus (from prolonged reflux)

Investigations⁸

- Endoscopy (see [Red flag pointers](#))—perform prior to empirical therapy. Limited role—about one-third negative
- Barium swallow and meal
- 24-hour ambulatory oesophageal pH monitoring

Management of GORD^{8,9,10,11}

Stage 1

- Patient education/appropriate reassurance
- Consider acid suppression or neutralisation
- Attend to lifestyle:
 - reduce weight if overweight (this alone may abolish symptoms)
 - reduce or cease smoking
 - reduce or cease alcohol (especially with dinner)
 - avoid fatty foods (e.g. pastries, french fries)
 - reduce or cease coffee, tea and chocolate
 - avoid coffee and alcohol late at night

avoid gaseous drinks

leave at least 3 hours between the evening meal and retiring

increase fibre intake (e.g. high-fibre cereals, fruit and vegetables)

small regular meals and snacks

eat slowly and chew food well

sleep on the left side

main meal at midday; light evening meal

avoid spicy foods and tomato products

- Drugs to avoid: anticholinergics, theophylline, nitrates, calcium-channel blockers, doxycycline. Pill-induced (i.e. before absorption) oesophagitis occurs, especially with tetracyclines, slow-release potassium, iron sulphate, corticosteroids, NSAIDs—avoid taking dry; use ample fluids
- Elevation of head of bed or wedge pillow: if GORD occurs in bed, sleep with head of bed elevated 10–20 cm on wooden blocks or use a wedge pillow (preferable)
- Antacids (see TABLES 36.4 and 36.5): best is liquid alginate/antacid mixture, e.g. Gaviscon/Mylanta plus 20 mL on demand or 1–2 hours after meals and at bedtime

Table 36.4 Antacids in common use

Antacids

Water soluble: Calcium carbonate

Sodium:

- bicarbonate
- citrotartrate

Note: Excess is prone to cause alkalosis—apathy, mental changes, stupor, kidney dysfunction, tetany

Water insoluble: Aluminium:

- hydroxide
- glycinate
- phosphate

Magnesium:

- alginate

- carbonate
- hydroxide
- trisilicate

Combination antacids

Antacid + alginic acid

Antacid + oxethazaine

Antacid + simethicone

Table 36.5 Side effects of common antacids

Aluminium hydroxide	Constipation
Magnesium trisilicate	Diarrhoea
Sodium bicarbonate	Alkalosis Milk-alkali syndrome Aggravation of hypertension
Calcium carbonate	Alkalosis Constipation Milk-alkali syndrome Hypercalcaemia

Antacids are appropriate for rapid relief of mild intermittent or occasional breakthrough symptoms but are ineffective for long-term management.

Stage 2⁸

If no relief after several weeks, the following approaches are recommended by the Gastroenterological Society of Australia (GESO).⁸

Reduce acid secretion. Select from:

- Proton-pump inhibitor (PPI) for 4 weeks (preferred agent) 30–60 minutes before food

lansoprazole 30 mg mane

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or

omeprazole 20 mg mane

or

pantoprazole 40 mg mane

or

esomeprazole 20 mg mane

or

rabeprazole 20 mg mane

- H₂-receptor antagonists (oral use for 8 weeks)

famotidine 20 mg bd

or

nizatidine 150 mg bd or 300 mg nocte

or

ranitidine 150 mg bd pc or 300 mg nocte

- Antacids are useful for daytime symptoms

Although the more traditional step-up approach of 1. Antacids → 2. H₂-receptor antagonists → 3. PPI can be used, there has been a change to favour a high-level (more potent) initial therapy with PPIs at standard dose (a step-down approach; see FIG. 36.1). This is based on the grounds of outcomes, speed of response and total cost. May need to eradicate *H. pylori* if present, although there is no consistent evidence of an association with GORD.¹⁰

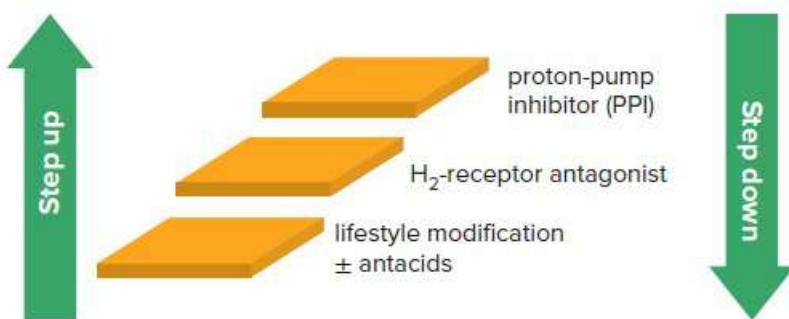


FIGURE 36.1 The stepwise approach to the management of dyspeptic symptoms⁷

Surgery is usually for young people with severe reflux. The gold standard is a short, loose 360-degree laparoscopic fundoplication.

Hiatus hernia

See FIGURE 36.2 .

- Common, especially in obese and >50 years.
- Most asymptomatic but GORD common.
- Diagnosis by barium swallow.

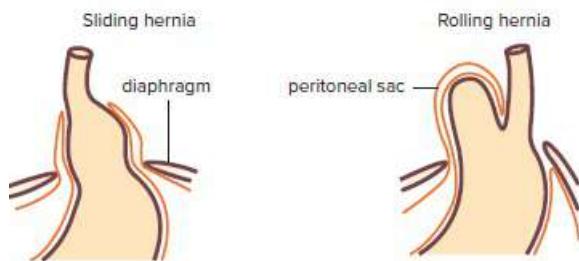


FIGURE 36.2 Hiatus hernia: rolling and sliding

Source: Longmore M, Wilkinson IB et al. *Oxford Handbook of Clinical Medicine* (9th edn). Oxford, 2014: 245. Reproduced with permission of the Licensor through PLSclear.

Types

- Sliding: GO junction slides into chest. Acid reflux common.
- Rolling (paraoesophageal): bulge of stomach herniates into chest. GORD uncommon but prone to strangulation.

Treatment

- Weight loss, esp. for GORD (treat reflux symptoms). Consider PPIs.
- Surgery for intractable symptoms and for repair of rolling hernia.

⌚ Functional (non-ulcer) dyspepsia^{9,12}

This term applies to the 60% of patients presenting with dyspepsia in which there is discomfort on eating in the absence of demonstrable organic disease. This can be considered in two categories (although there is overlap):

- ulcer-like dyspepsia—localised pain
 - or
 - dysmotility-like dyspepsia—diffuse discomfort

Ulcer-like dyspepsia

Treat as for GORD. A practical approach is to commence with a 4-week trial of a PPI or an H₂-receptor antagonist and cease if symptoms resolve.⁹

Dysmotility-like dyspepsia

Clinical features

- Discomfort with early sense of fullness on eating
- Nausea
- Overweight
- Emotional stress
- Poor diet (e.g. fatty foods)
- Similar lifestyle guidelines to GORD

Management

- Treat as for GORD (stage 1).
- Include antacids.
- If not responsive:

Step 1: H₂-receptor antagonists

Step 2: prokinetic agents

domperidone 10 mg tds

or

metoclopramide 10 mg tds

- Consider possibility of Barrett oesophagus or gastroparesis (see [CHAPTER 49](#)).

Barrett oesophagus

- Usually a metaplastic response to prolonged reflux
- A premalignant condition (adenocarcinoma)

- Lower oesophagus lined with gastric mucosa (at least 3 cm) of metaplastic columnar epithelium
- Prone to ulceration
- Needs careful management, which includes PPIs for symptoms of oesophagitis + reflux
- Consider 2-yearly endoscopies with biopsies
- Diagnosed by endoscopy and biopsy

Peptic ulcer disease^{12,13}

Features (general)

- Common: 10% incidence over a lifetime, but decreasing
- Peptic ulcers accounted for 1 in every 500 deaths in Australia¹⁴ in 2018
- DU:GU = 4:1
- DUs common in men 3:1
- Risk factors:

male sex

family history

smoking (cause and delayed healing)

stress

more common in blood group O

NSAIDs 2–4 times increase in GU and ulcer complications

H. pylori

- Unproven risk factors:

corticosteroids

alcohol (except for gastric erosion)

diet (does reduce recurrence of PU)

- Types of ulcers:

lower oesophageal
gastric
stomal (postgastric surgery)
duodenal

Note: If NSAIDs and *H. pylori* are not implicated, it is referred to as idiopathic (affects a small population group).

Clinical features

- Episodic burning epigastric pain related to meals (1–2 hours after)
- Relieved by food or antacids (generally)
- Dyspepsia common
- May be ‘silent’ in elderly on NSAIDs
- Physical examination often unhelpful

Investigations

- Endoscopy (investigation of choice):¹⁵ 92% predictive value
- Barium studies: 54% predictive value
- Serum gastrin (consider if multiple ulcers)
- *H. pylori* test: serology or urea breath test; diagnosis usually based on urease test performed at endoscopy

Complications

- Perforation
- Bleeding → haematemesis and melaena
- Obstruction—pyloric stenosis
- Anaemia (blood loss)
- Cancer (in GU)
- Oesophageal stenosis

Bleeding peptic ulcer

This can be treated with endoscopic haemostasis with electrocautery heater probe or injection of adrenaline or both. Also IV omeprazole 80 mg bolus, then 8 mg/hr IV infusion for 3 days. Surgery is an option. IV esomeprazole, omeprazole or pantoprazole can also be used.

Management of peptic ulcer disease

Aims of treatment:

- relieve symptoms
- accelerate ulcer healing
- prevent complications
- minimise risk of relapse

The treatment of a GU is similar to that for a DU except that GUs take about 2 weeks longer to heal and the increased risk of malignancy has to be considered.

Stage 1⁹

General measures: (lifestyle and symptom relief)

- same principles as for GORD
- stop smoking
- avoid irritant drugs: NSAIDs, aspirin
- normal diet but avoid foods that upset
- antacids

If *H. pylori* positive—eradicate with combined therapy. Confirm eradication with a urea breath test (DU) or repeat gastroscopy (GU) and repeat if still present.¹⁰

If *H. pylori* negative—treat with full-dose PPI.

Proton-pump inhibitors

Proton-pump inhibitors (PPIs) provide more potent acid suppression and heal GUs and DUs more rapidly than H₂-receptor antagonists.

- 4–8-week oral course

PPIs are frequently used for longer than needed, with many people on long-term high

doses which are unnecessary and potentially harmful (risk of *C. difficile*, osteoporotic fractures, pneumonia, nutritional deficiencies). Consider deprescribing for those without Barrett oesophagus, high-grade oesophagitis or GI bleeding. Long-term users may experience rebound symptoms upon cessation; reduce gradually and offer prn occasional use.¹⁶

Use with caution in:

- the elderly
- those on drugs, especially warfarin, anticonvulsants, beta blockers
- liver disease

Therapy to eradicate *Helicobacter pylori*^{17,18}

This organism has a proven link with PU disease (both DU and benign non-drug induced GU), gastric cancer and MALToma (a gastric lymphoma) because of mucosal infection. This hypothesis is supported by a very low relapse of DU in subjects eradicated of *H. pylori*. Most infected people are asymptomatic but infection leads to a lifetime risk of peptic ulcer disease in 15–20% and of gastric cancer in up to 2%.¹² Twenty per cent of people have a variety of symptoms including those from gastritis and duodenitis. Treatment is based on combination triple or quadruple therapy, which can achieve a successful eradication rate of 85–90%.

Drug treatment regimens (examples)⁹

First-line therapy:

PPI (e.g. omeprazole or esomeprazole 20 mg)

plus

clarithromycin 500 mg

plus

amoxicillin 1 g

All orally twice daily for 7 days; this is the preferred regimen (available as a combination pack)

Note: A 10–14-day course improves eradication rate by approx. 5%.¹

or

PPI + clarithromycin + metronidazole 400 mg (twice daily for 7 days)—if hypersensitive to penicillin

or

PPI + amoxicillin + levofloxacin (for salvage therapy)

or

other combinations: quadruple therapy, e.g. bismuth + PPI + tetracycline + metronidazole (for failed triple combination)

Note: Resistance to metronidazole is common (>50%) and to clarithromycin is increasing (about 5% plus), but uncommon with tetracycline and amoxicillin.⁶

Antacids are good for daytime relief.

Maintenance anti-secretory therapy is usually unnecessary for *H. pylori* ulcers after successful eradication.⁹

For children with confirmed *H. pylori*:

PPI + amoxicillin + clarithromycin

Surgical treatment

Indications (now uncommon) include:

- failed medical treatment after 1 year
- complications:

uncontrollable bleeding

perforation

pyloric stenosis

- suspicion of malignancy in GU

NSAIDs and peptic ulcers^{9,19}

1. Ulcer identified in NSAID user:

- stop NSAID (if possible)
- check smoking and alcohol use
- try alternative anti-inflammatory analgesic:
 - paracetamol
 - enteric-coated, slow-release aspirin

corticosteroids intra-articular or oral

- PPI for 4 weeks (gives best results)

Note: Healing time is doubled if NSAID continued.³ About 90% heal within 12 weeks. Check healing by endoscopy at 12 weeks. Do *H. pylori* test.

1. Prevention of ulcers in NSAID user:¹⁹

Primary prophylaxis is usually reserved for those at significantly increased risk, e.g. older persons (>75 years) and past history PU.

Use one of the following PPIs:⁹

esomeprazole 20 mg bd for 7 days

or

omeprazole 20 mg daily

or

pantoprazole 40 mg daily

Increased dietary fibre assists DU healing and prevention.

Note: Do *H. pylori* test and, if present, it should be eradicated with combination therapy after the ulcer has healed, especially in people who continue to take NSAIDs.¹

Autoimmune gastritis⁹

This is an inflammatory condition with antibodies to parietal cells and intrinsic factor. It is asymptomatic and may lead to pernicious anaemia. Diagnosis is confirmed by histology or endoscopy. *H. pylori* is absent.

Treat with iron and vitamin B12 if they are low.

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Stomach cancer

This is the fourth most common cancer worldwide.

Clinical features

- Male to female ratio = 3:1
- Usually asymptomatic early

- Consider if upper GIT symptoms in patients over 40 years, especially weight loss
- Recent-onset dyspepsia in middle age
- Dyspepsia unresponsive to treatment
- Vague fullness or epigastric distension
- Anorexia, nausea ± vomiting
- Dysphagia—a late sign
- Onset of anaemia
- Changing dyspepsia in GU
- Changing symptoms in pernicious anaemia
- *H. pylori* is implicated as a cause. Its treatment reduces the risk¹ and is recommended in high-risk groups

Also implicated in gastric mucosa-associated lymphoid tissue (MALT) lymphoma

Risk factors: ↑ age, blood group A, smoking, sugar, atrophic gastritis, diet—high in salted and smoked foods

Limited physical findings

- Palpable abdominal mass (20%)
- Signs (see FIG. 36.3) in advanced cases

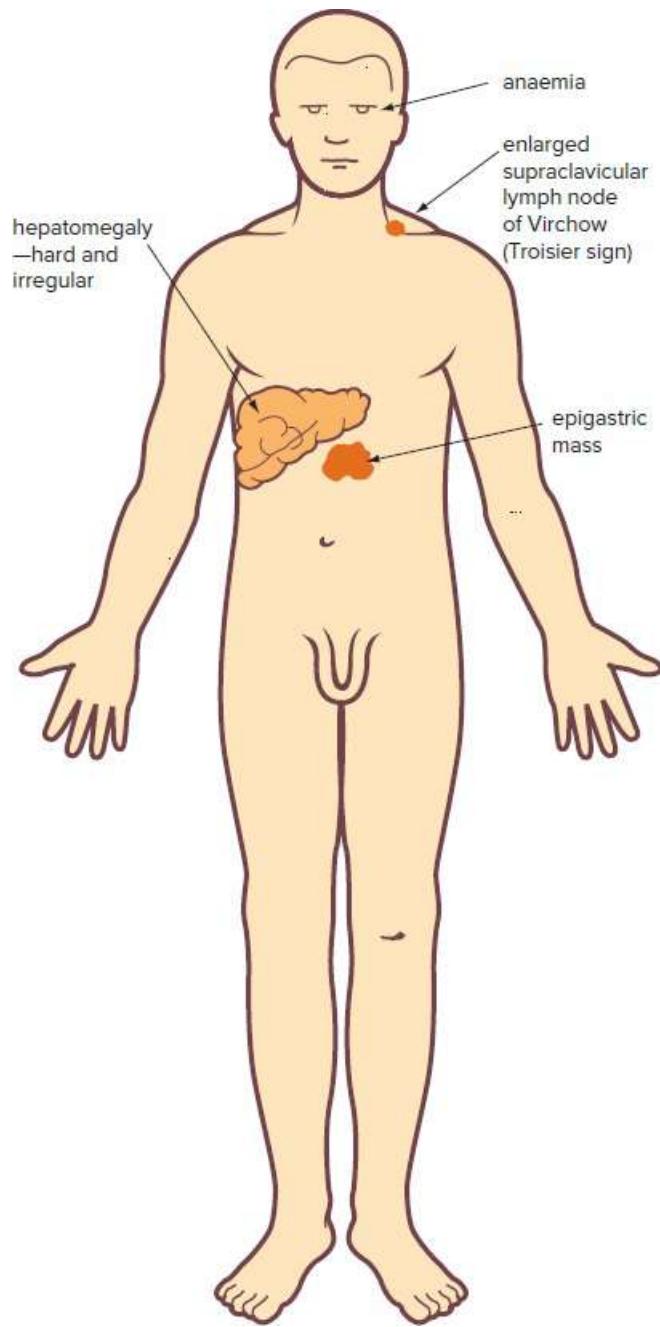


FIGURE 36.3 Late signs of stomach cancer



DxT malaise + anorexia + dyspepsia + weight loss → stomach cancer



DxT triple loss of appetite + weight + colour → stomach cancer

Investigations

- Endoscopy and biopsy is optimal test
- Barium meal—false negatives

Treatment

- Surgical excision: may be curative if diagnosed early but overall survival is poor (22% at 5 years)

When to refer

- Infants with persistent gastro-oesophageal reflux not responding to simple measures
- Failure to respond to stage 1 therapy for heartburn, when endoscopy is required
- Patients with persistent or recurrent ulcers
- Any patient with a PU complication, such as haemorrhage, obstruction or perforation

Practice tips

- Scleroderma is a rare but important cause of oesophagitis.
- Advise patients never to 'dry swallow' medications.
- Persistent dysphagia always warrants investigation, not observation.
- Beware of attributing anaemia to oesophagitis.
- Epigastric pain aggravated by any food, relieved by antacids = chronic GU.
- Epigastric pain before meals, relieved by food = chronic DU.
- Keep in mind the malignant potential of a GU.
- A change in the nature of symptoms with a GU suggests the possibility of malignant change.
- Avoid the long-term use of water-soluble antacids.
- Investigate the alarm symptoms—dysphagia, bleeding, anaemia, weight loss, waking at night, pain radiating to the back.

Patient education resources

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

- Barrett oesophagus
- Heartburn
- Hiatus hernia
- Peptic ulcer
- Reflux disease
- Reflux in infants

References

- 1 Buckley N (Chair). *Australian Medicines Handbook*. Adelaide: Australian Medicines Handbook Pty Ltd, 2018: 502–19.
- 2 Sung JJY et al. Systematic review: the global incidence and prevalence of peptic ulcer disease. *Alimentary Pharmacology & Therapeutics*, 2009; 29: 938–46.
- 3 Pritchard P. The management of upper gastrointestinal problems in patients taking NSAIDs. *Aust Fam Physician*, 1991; 20: 1739–41.
- 4 McGarity B, Morgia M. Peptic ulcer disease: an update on diagnosis and treatment. *Medicine Today*, 2001; December: 33–7.
- 5 Sewell J. Gastro-oesophageal reflux. *Australian Paediatric Review*, 1991; 3: 2.
- 6 Finck A, Morris L. Thickened feedings for infants with gastroesophageal reflux. *Am Fam Physician*, 1 Oct 2019;100(7): 437.
- 7 Gwee A, Rimer R, Marks M. *Paediatric Handbook* (9th edn). Oxford: Wiley-Blackwell, 2015: 96–7.
- 8 *Gastro-oesophageal Reflux Disease in Adults: Guidelines* (5th edn). Sydney: Gastroenterological Society of Australia, 2011.
- 9 Gastric disorders [published 2016]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2016. www.tg.org.au, accessed October 2019.
- 10 Ness-Jensen E et al. Weight loss and reduction in gastroesophageal reflux. *The HUNT*

study. Am J Gastroenterol, 2014; 109(2): 171–7.

- 11 Fock KM et al. Asia-Pacific consensus on the management of gastroesophageal reflux disease: update. J Gastroenterol Hepatol, 2008; 23: 8–22.
- 12 Katelaris P. Dyspepsia: update. Australian Doctor, 2005; 7 October: 23–5.
- 13 Madge S, Yeomans N. Stomach and duodenal ulcers. Current Therapeutics, 2001; September: 69–72.
- 14 Australia: peptic ulcer disease. World Health Rankings. Available from: <https://www.worldlifeexpectancy.com/australia-peptic-ulcer-disease>, accessed February 2021.
- 15 Korman M, Sievert W. Peptic ulcers. In: *MIMS Disease Index* (2nd edn). Sydney: IMS Publishing, 1996: 400–2.
- 16 RACGP Choosing Wisely panel. The Royal Australian College of General Practitioners Recommendations: 1. Don't use proton pump inhibitors (PPIs) long term in patients with uncomplicated disease without regular attempts at reducing dose or ceasing. April 2005. Available from: <https://www.choosingwisely.org.au/recommendations/racgp>, accessed March 2021.
- 17 Ford A et al. Eradication therapy for peptic ulcer disease in *Helicobacter* positive patients (Cochrane Review). Cochrane Database. Syst Rev, 2004; Issue 4: Art No. CD003840.
- 18 Sugano K et al. Kyoto global consensus report on *Helicobacter pylori* gastritis. Gut, 2015; 64(9): 1353–67.
- 19 Chan FK, To KF, Wu JC. Eradication of *Helicobacter pylori* and risk of peptic ulcers in patients starting long term treatment with NSAIDs: a randomised trial. Lancet, 2002; 359(9300): 9–13.

37 Dysphagia

We swallow approximately 1200 times daily, largely subconsciously. While we take the fundamental function for granted, disordered swallowing can be a devastating condition, with substantial morbidity for those affected.

IAN COOK 1996

Dysphagia is difficulty in swallowing. It is a common problem affecting up to 22% of patients at some point in the general practice setting.¹ It is usually associated with a sensation of hold-up of the swallowed bolus and is sometimes accompanied by pain.

Its origin is considered as either oropharyngeal or oesophageal. Oropharyngeal dysphagia is usually related to neuromuscular dysfunction and is commonly caused by stroke. Oesophageal dysphagia is usually due to motor disorders, such as achalasia or diffuse oesophageal spasm, and to peptic oesophageal strictures often secondary to reflux. In this type of dysphagia there is a sensation of a hold-up, which may be experienced in either the cervical or retrosternal region.¹ Causes are usually classified as functional, mechanical and neurological (see TABLE 37.1).

Table 37.1 Causes of dysphagia

Functional	Examples: muscle tension, 'express swallowing'
Neurological	Examples: stroke, myasthenia, MND
Mechanical	
• luminal	• Example: foreign body
• mural	• Example: stricture, tumour
• extramural	• Example: extrinsic compression (i.e. goitre)

Dysphagia must not be confused with globus sensation, which is the sensation of the constant 'lump in the throat' although there is no actual difficulty swallowing food. If dysphagia is progressive or prolonged then urgent attention is necessary.

There are only a few common causes of dysphagia and these are usually readily diagnosed on the history and two or three investigations. A careful history is very important, including a drug history and psychosocial factors.

Diagnostic guidelines

- Any disease or abnormality affecting the tongue, pharynx or oesophagus can cause dysphagia.
- Patients experience a sensation of obstruction at a definite level with swallowing food or water; hence, it is convenient to subdivide dysphagia into oropharyngeal and oesophageal.
- Pain from the oropharynx is localised to the neck.
- Pain from the oesophagus is usually felt over the T2–6 area of the chest.
- Oropharyngeal causes: difficulty initiating swallowing; food sticks at the suprasternal notch level; regurgitation; aspiration.
- Oesophageal causes: food sticks to mid to lower sternal level; pain on swallowing solid foods, especially meat, potatoes and bread, and then eventually liquids.
- A pharyngeal pouch usually causes regurgitation of undigested food and gurgling may be audible over the side of the neck.
- Neurological disorders typically result in difficulty swallowing or coughing or choking due to food spillover, especially with liquids.
- Dysphagia for solids only indicates a structural lesion, such as a stricture or tumour.
- Dysphagia for liquids and solids is typical of an oesophageal motility disorder, namely achalasia.²
- GORD tends to exclude achalasia.
- Scleroderma may lead to a peptic stricture.
- Gastroenterologists suggest the ‘big three’ common causes referred for specialist investigation are benign peptic stricture, cancer and achalasia.³
- Intermittent dysphagia for both liquids and solids is characteristic of a motility disorder such as oesophageal achalasia.
- Malignant oesophageal obstruction is usually evident when there is a short history of rapidly progressive dysphagia and significant weight loss.⁴

Red flag pointers for dysphagia

- Age >50 years
- Recent or sudden onset
- Unexplained weight loss
- Painful swallowing
- Progressive dysphagia
- Dysphagia for solids
- Hiccoughs
- Hoarseness
- Neurological symptoms/signs

A summary of the diagnostic strategy model is presented in [TABLE 37.2](#) .

Table 37.2 Dysphagia: diagnostic strategy model (excluding oropharyngeal infections and strokes)

Probability diagnosis

Functional (e.g. ‘express’ swallowing, psychogenic)

Tablet-induced irritation

Pharyngotonsillitis

GORD/reflux oesophagitis

Serious disorders not to be missed

Neoplasia:

- cancer of the pharynx, oesophagus, stomach
- extrinsic tumour

AIDS (opportunistic oesophageal infection in immunocompromised, also candidiasis, herpes and viral oesophagitis)

Stricture, usually benign peptic stricture

Oesophageal food bolus obstruction

Scleroderma

Neurological causes:

- pseudobulbar palsy
- multiple sclerosis/myasthenia gravis
- motor neurone disease (amyotrophic sclerosis)

- Parkinson disease

Pitfalls (often missed)

Foreign body

Drugs (e.g. phenothiazines, bisphosphonates)

Subacute thyroiditis

Extrinsic lesions (e.g. lymph nodes, goitre)

Upper oesophageal web (e.g. Plummer–Vinson syndrome)

Diffuse oesophageal spasm

Eosinophilic oesophagitis

Radiotherapy

Achalasia

Upper oesophageal spasm (mimics angina)

Rarities (some):

- Sjögren syndrome
- aortic aneurysm
- aberrant right subclavian artery
- lead poisoning
- cervical osteoarthritis (large osteophytes)
- other neurological causes
- other mechanical causes

Seven masquerades checklist

Depression

Drugs

Thyroid disorder

Is the patient trying to tell me something?

Yes. Could be functional. ?Globus sensation.

Examination

It is worthwhile focusing on the following features:

- general examination including hands and skin ?scleroderma
- assess nutritional status including BMI
- mouth, pharynx, larynx (look for paralysis) ?dentition
- neck, especially for lymph nodes and thyroid