Pharyngeal pouch Schatzki (lower oesophageal) ring Foreign body *Extrinsic (outside oesophagus)* Goitre with retrosternal extension Mediastinal tumours, bronchial carcinoma, vascular compression (rare)
Neuromuscular motility disorders (hints from the history: solids and liquids equally difficult, symptoms intermittent) Achalasia Diffuse oesophageal spasm Scleroderma
Pharyngeal dysphagia (hints: aspiration, fluid regurgitation into the nose) Cricopharyngeal dysfunction—Zenker's diverticulum Neurological diseases: bulbar or pseudobulbar palsy, myasthenia gravis, polymyositis, myotonic dystrophy

Questions box 6.2

Questions to ask the patient with acid reflux or suspected gastro-oesophageal reflux disease (GORD)

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. Do you have heartburn (a burning pain under the sternum radiating up towards the

throat)? How often does this occur?—More than once a week suggests GORD

2. Does your heartburn occur after meals or when you lean forward or lie flat in bed (typical of acid reflux)?

3. Does the pain radiate across the chest down the left arm or into the jaw?—Suggests myocardial ischaemia

4. Is the pain relieved by antacids or over-the-counter acid-suppressing medicines?

5. Do you experience suddenly feeling bitter tasting fluid in the mouth?—Acid regurgitation

6. Have you experienced the sudden appearance of a salty tasting or tasteless fluid in the mouth?—Waterbrash

7. Have you had trouble swallowing?—Dysphagia (see *Questions box 6.3*)

8. Have you been troubled by a cough when you lie down?

oesophagus, caustic damage to the oesophagus or oesophageal perforation.

If the patient complains of difficulty initiating swallowing, fluid regurgitating into the nose or choking on trying to swallow, this suggests that the cause of the dysphagia is in the pharynx (pharyngeal dysphagia). Causes of pharyngeal dysphagia can include neurological disease (e.g. motor neurone disease, resulting in bulbar or pseudobulbar palsy).

If the patient complains of food sticking in the oesophagus, it is important to consider a number of anatomical causes of oesophageal blockage.[1] Ask the patient to point to the site where the solids stick. If there is a mechanical obstruction at the lower end of the oesophagus, most often the patient will localise the dysphagia to the lower retrosternal area. However, obstruction higher in the oesophagus may be felt anywhere in the retrosternal area. If heartburn is also present, for example, this suggests that gastro-oesophageal reflux with or without stricture formation may be the cause of the dysphagia. The actual course of the dysphagia is also a very important part of the history to obtain. If the patient states that the dysphagia is intermittent or is present only with the first few swallows of food, this suggests either a lower oesophageal ring or oesophageal spasm. However, if the patient complains of progressive difficulty swallowing, this suggests a stricture, carcinoma or achalasia. If the patient states that both solids and liquids stick, then a motor disorder of the oesophagus is more likely, such as achalasia or diffuse oesophageal spasm.

Diarrhoea

The symptom diarrhoea can be defined in a number of different ways. Patients may complain of frequent stools (more than three per day being abnormal) or they may complain of a change in the consistency of the stools, which have become loose or watery. There are a large number of possible causes of diarrhoea.

Some patients pass small amounts of formed stool more than three times a day because of an increased desire to defecate. The stools are not loose and stool volume is not increased. This is not true diarrhoea. It can occur because of local rectal pathology, incomplete rectal emptying, or because of a psychological disturbance that leads

Questions box 6.3

Questions to ask a patient who reports difficulty swallowing

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. Do you have trouble swallowing solids, liquid or both?—Solids and liquids suggests a motor problem, e.g. achalasia

2. Where does the hold-up occur (please point to the area)?—Oesophageal carcinoma

3. Is the trouble swallowing intermittent or persistent?—Intermittent suggests eosinophilic oesophagitis

4. Has the problem been getting progressively worse?

5. Do you cough or choke on starting to swallow (oropharyngeal dysphagia)?

6. Is it painful to swallow (odynophagia)?

7. Do you have any heartburn or acid regurgitation?

8. Have you been losing weight?

to an increased interest in defaecation.

When a history of diarrhoea is obtained, it is also important to determine if this has occurred acutely or whether it is a chronic problem. Acute diarrhoea is more likely to be infectious in nature, while chronic diarrhoea has a large number of causes.

Clinically, diarrhoea can be divided into a number of different groups based on the likely disturbance of physiology.[2]

1. Secretory *diarrhoea* is likely if the diarrhoea is of high volume

1. *Secretory diarrhoea* is likely if the diarrhoea is of high volume (commonly more than 1 litre per day) and persists when the patient fasts; there is no pus or blood, and the stools are not excessively fatty. Secretory diarrhoea occurs when net secretion in the colon or small bowel exceeds absorption; some of the causes include infections (e.g. *E. coli*, *Staphylococcus aureus*, *Vibrio cholerae*), hormonal conditions (e.g. vasoactive intestinal polypeptide-secreting tumour, Zollinger-Ellison[a] syndrome, carcinoid syndrome) and villous adenoma.

2. *Osmotic diarrhoea* is characterised by its

Questions box 6.4

Questions to ask the patient presenting with diarrhoea

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. How many stools a day do you pass now normally?
2. What do the stools look like (stool form e.g. loose and watery)?
3. Do you have to race to the bathroom to have a bowel movement?—Urgency in colonic disease
4. Have you been woken from sleep during the night by diarrhoea?—Organic cause more likely
5. Have you seen any bright-red blood in the stools or mucus or pus?—Suggests colonic disease
6. Are you passing large volumes of stool every day?—Suggests small bowel disease
7. Are your stools pale, greasy, smelly and difficult to flush away (steatorrhoea)? Have you seen oil droplets in the stool?—Pancreatic disease
8. Have you had problems with leakage of stool (faecal incontinence)?
9. Have you lost weight?—e.g. cancer, malabsorption
10. Have you had any abdominal pain or vomiting?
11. Have you had treatment with antibiotics recently?
12. Have you had any recent travel? Where to?
13. Have you a personal history of inflammatory bowel disease or prior gastrointestinal surgery?
14. Have you any history in the family of coeliac disease or inflammatory bowel disease?
15. Have you had any problems with arthritis?—Inflammatory bowel disease, Whipple's disease
16. Have you had recent fever, rigors, or chills (e.g. infection, lymphoma)? Have you had frequent infections?—Immunoglobulin deficiency

disappearance with fasting and by large-volume stools related to the ingestion of food. Osmotic diarrhoea occurs due to excessive solute drag; causes include lactose intolerance (disaccharidase deficiency), magnesium antacids

or gastric surgery.

3. *Abnormal intestinal motility* (e.g. thyrotoxicosis, the irritable bowel syndrome) can also cause diarrhoea.

4. *Exudative diarrhoea* occurs when there is inflammation in the colon. Typically the stools are of small volume but frequent, and there may be associated blood or mucus (e.g. inflammatory bowel disease, colon cancer).

5. *Malabsorption* of nutrients can result in steatorrhoea. Here the stools are fatty, pale coloured, extremely smelly, float in the toilet bowel and are difficult to flush away. Steatorrhoea is defined as the presence of more than 7 g of fat in a 24-hour stool collection. There are many causes of steatorrhoea (page 190).

Constipation

It is important to determine what patients mean if they say they are constipated.[3] Constipation is a common symptom and can refer to the passage of infrequent stools (fewer than three times per week), hard stools or stools that are difficult to evacuate. This symptom may occur acutely or may be a chronic problem. In many patients, chronic constipation arises because of habitual neglect of the impulse to defecate, leading to the accumulation of large, dry faecal masses. With constant rectal distension from faeces, the patient may grow less aware of rectal fullness, leading to chronic constipation. Constipation may arise from ingestion of drugs (e.g. codeine, antidepressants and aluminium or calcium antacids), and with various metabolic or endocrine diseases (e.g. hypothyroidism, hypercalcaemia, diabetes mellitus, phaeochromocytoma, porphyria, hypokalaemia) and neurological disorders (e.g. aganglionosis, Hirschsprung's[b] disease, autonomic neuropathy, spinal cord injury, multiple sclerosis). Constipation can also arise after partial colonic obstruction from carcinoma; it is, therefore, very important to determine whether there has been a recent change in bowel habit, as this may indicate development of a malignancy. Patients with very severe constipation in the absence of structural disease may be found on a transit study to have slow colonic transit; such slow-transit constipation is most common in young women.

Constipation is common in the later stages of pregnancy.

Difficulty with evacuation of faeces may occur with disorders of the pelvic floor muscles or nerves, or anorectal disease (e.g. fissure, or stricture). Patients with this problem may complain of straining, a feeling of anal blockage or even the need to self-digitate to perform manual evacuation of

faeces.

A chronic but erratic disturbance in defaecation (typically alternating constipation and diarrhoea) associated with abdominal pain, in the absence of any structural or biochemical abnormality, is very common; such patients are classified as having the *irritable bowel syndrome*.[4] Patients who report abdominal pain plus two or more of the following symptoms—abdominal pain relieved by defaecation, looser or more frequent stools with the onset of abdominal pain, passage of mucus per rectum, a feeling of incomplete emptying of the rectum following defaecation and visible abdominal distension—are more likely to have the irritable bowel syndrome than organic disease.

Mucus

The passage of mucus (white slime) may occur because of a solitary rectal ulcer, fistula or villous adenoma, or in the irritable bowel syndrome.

Bleeding

Patients may present with the problem of haematemesis (vomiting blood), melaena (passage of jet-black stools) or haematochezia (passage of bright-red blood per rectum). Sometimes patients may present because routine testing for occult blood in the stools is positive (page 183). It is important to ensure that if vomiting of blood is reported, this is not the result of bleeding from a tooth socket or the nose, or coughing up of blood.

Haematemesis indicates that the site of the bleeding is proximal to or at the duodenum. Ask about symptoms of peptic ulceration; haematemesis is commonly due to bleeding chronic peptic ulceration, particularly from a duodenal ulcer. Acute peptic ulcers often bleed without abdominal pain. A Mallory-Weiss tear usually occurs with repeated vomiting; typically the patient reports first the vomiting of clear gastric contents and then the vomiting of blood. Less-common causes of upper gastrointestinal bleeding are presented in Table 6.3.

TABLE 6.3 Causes of acute gastrointestinal bleeding

Upper gastrointestinal tract

More common

1. Chronic peptic ulcer: duodenal ulcer, gastric ulcer
2. Acute peptic ulcer (erosions)

Less common

3. Mallory-Weiss* syndrome (tear at the gastro-oesophageal junction)

4. Oesophageal and/or gastric varices

5. Erosive or ulcerative oesophagitis

6. Gastric carcinoma, polyp, other tumours

7. Dieulafoy's† ulcer (single defect that involves an ectatic submucosal artery)

8. Watermelon stomach (antral vascular ectasias)

9. Aortoenteric fistula (usually aortoduodenal and after aortic surgery)

10. Vascular anomalies—angiodysplasia, arteriovenous malformations, blue rubber bleb naevus syndrome, hereditary haemorrhagic telangiectasia, CRST syndrome

11. Pseudoxanthoma elasticum, Ehlers-Danlos‡ syndrome

12. Amyloidosis

13. Vasculitis

14. Ménétrier's§ disease

15. Bleeding diathesis

16. Pseudohaematemesis (nasopharyngeal origin)

Lower gastrointestinal tract

More common

1. Angiodysplasia
2. Diverticular disease
3. Colonic carcinoma or polyp
4. Haemorrhoids or anal fissure

Less common

5. Massive upper gastrointestinal bleeding

6. Inflammatory bowel disease

7. Ischaemic colitis

8. Meckel's[#] diverticulum

9. Small bowel disease, e.g. tumour, diverticula, intussusception

10. Haemobilia (bleeding from the gallbladder)

11. Solitary colonic ulcer

CRST = calcinosis, Raynaud's phenomenon, sclerodactyly and telangiectasia.

[*] George Kenneth Mallory (b. 1900), professor of pathology, Boston, and Soma Weiss (1898–1942), professor of medicine, Boston City Hospital described this syndrome in 1929.

[†] Georges Dieulafoy (1839–1911), Paris physician.

[‡] Edvard Ehlers (1863–1937), German dermatologist, described the syndrome in 1901, and Henri Alexandre Danlos (1844–1912), French dermatologist, described the syndrome in 1908.

[§] Pierre Ménétrier (1859–1935), French physician.

[#] Johann Friedrich Meckel the younger (1781–1833), Professor of Surgery and Anatomy at Halle. His father and grandfather were also professors of anatomy.

Haemorrhoids and local anorectal diseases such as fissures will commonly present with passing small amounts of bright-red blood per rectum. The blood is normally not mixed in the stools but is on the toilet paper, on top of the stools or in the toilet bowl. Melaena usually results from bleeding

Questions box 6.5

Questions to ask a patient presenting with constipation

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. How often do you have a bowel movement?
2. Are your stools hard or difficult to pass?
3. What do the stools look like (stool form e.g. small pellets)?
4. Do you strain excessively on passing stool?
5. Do you feel there may be a blockage at the anus area when you try to pass stool?
6. Do you ever press your finger in around the anus (or vagina) to help stool pass?
7. Has your bowel habit changed recently?
8. Any recent change in your medications?
9. Any blood in the stools?
10. Any abdominal pain?
11. Recent weight loss?
12. Do you ever have diarrhoea?
13. Do you have a history of colon polyps or cancer? Any family history of colon cancer?

Questions box 6.6

Questions to ask the patient who presents with vomiting blood (haematemesis)

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem. !

1. Was there fresh blood in the vomitus? Or was the vomitus coffee-grain stained?
2. Have you passed any black stools or blood in the stools?
3. Before any blood was seen in the vomitus, did you experience intense retching or vomiting? – Mallory-Weiss tear

vomiting?—Mallory-Weiss tear

4. Have you been taking aspirin, non-steroidal anti-inflammatory drugs or steroids?

5. Do you drink alcohol?

6. Have you ever had a peptic ulcer?

7. Have you lost weight?

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from the upper gastrointestinal tract, although right-sided colonic and small bowel lesions can occasionally be responsible. Massive rectal bleeding can occur from the distal colon or rectum, or from a major bleeding site higher in the gastrointestinal tract. With substantial lower gastrointestinal tract bleeding, it is important to consider the presence of angiodysplasia or diverticular disease (where bleeding more often occurs from the right rather than the left colon, even though diverticula are more common in the left colon). Less-common causes of lower gastrointestinal bleeding are presented in Table 6.3.

Spontaneous bleeding into the skin, or from the nose or mouth, can be a problem for patients with coagulopathy resulting from liver disease.

Jaundice

Usually the relatives notice a yellow discoloration of the sclerae or skin before the patient does. Jaundice is due to the presence of excess bilirubin being deposited in the sclerae and skin. The causes of jaundice are described on page 185. If there is jaundice, ask about the colour of the urine and stools; pale stools and dark urine occur with obstructive or cholestatic jaundice because urobilinogen is unable to reach the intestine. Also ask about abdominal pain; gallstones, for example, can cause biliary pain and jaundice.[5]

Pruritus

This symptom means itching of the skin, and may be either generalised or localised. Cholestatic liver disease can cause pruritus which tends to be worse over the extremities. Other causes of pruritus are discussed on page 445.

Abdominal bloating and swelling

A feeling of swelling (bloating) may be a result of excess gas or a hypersensitive intestinal tract (as occurs in the irritable bowel syndrome). Persistent swelling can be due to ascitic fluid accumulation; this is discussed

Persistent swelling can be due to ascitic fluid accumulation, this is discussed on page 175. It may be associated with ankle oedema.

Lethargy

Tiredness and easy fatiguability are common symptoms for patients with acute or chronic liver disease, but the mechanism is not known. This can also occur because of anaemia due to gastrointestinal or chronic inflammatory disease. Lethargy is also very common in the general population and is not a specific symptom.

Treatment

The treatment history is very important. Traditional non-steroidal anti-inflammatory drugs (NSAIDs), including aspirin, can induce bleeding from acute or chronic damage to the gastrointestinal tract. As described above, many drugs can result in disturbed defaecation. A large number of drugs are also known to affect the liver. For example, acute hepatitis can occur with halothane, phenytoin or chlorothiazide. Cholestasis may occur from a

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Questions box 6.7

Questions to ask the patient presenting with jaundice
! denotes symptoms for the possible diagnosis of an urgent or dangerous problem. !

1. Is your urine dark? Are your stools pale?—Obstructive jaundice
2. Do you have any skin itching (pruritus)?
3. Have you had any fever?
4. Have you had a change in your appetite or weight?—Malignancy
5. Have you had any abdominal pain or change in bowel habit?
6. Have you had any vomiting of blood or passage of dark stools?
7. Do you drink alcohol? How much? How long? (CAGE questions, page 7)
8. Have you ever used intravenous drugs?
9. Have you ever had a blood transfusion?
10. Have you started any new medications recently?

10. Have you started any new medications recently?

11. Have you had any recent contact with patients with jaundice or liver problems?

12. Any history of recent high-risk sexual behaviours?

13. Have you travelled overseas to areas where hepatitis A is endemic?

14. Have you been immunised against hepatitis B?

15. Any history of inflammatory bowel disease?

16. What is your occupation (contact with hepato-toxins)?

17. Is there any family history of liver disease?

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hypersensitivity reaction to chlorpromazine or other phenothiazines, sulfonamides, sulfonylureas, phenylbutazone, rifampicin or nitrofurantoin. Anabolic steroids and the contraceptive pill can cause dose-related cholestasis. Fatty liver can occur with alcohol use, tetracycline, valproic acid or amiodarone. Large blood-filled cavities in the liver called peliosis hepatis can occur with anabolic steroid use or the contraceptive pill. Acute liver cell necrosis can occur if an overdose of paracetamol (acetaminophen) is taken.

Past history

Surgical procedures can result in jaundice from the anaesthesia (e.g. multiple uses of halothane), hypoxaemia of liver cells (hypotension during the operative or postoperative period) or direct damage to the bile duct during abdominal surgery. A history of relapsing and remitting epigastric pain in a patient who presents with severe abdominal pain may indicate that a peptic ulcer has perforated. A past history of inflammatory bowel disease (either ulcerative colitis or Crohn's disease) is important as these are chronic diseases that tend to flare up.

Social history

The patient's occupation may be relevant (e.g. healthcare workers may be exposed to hepatitis). Toxin exposure can also be important in chronic liver disease (e.g. carbon tetrachloride, vinyl chloride). If a patient has symptoms suggestive of liver disease, ask about recent travel to countries where hepatitis is endemic.

The alcohol history is very important, particularly as alcoholics often

deny or understate the amount they consume (see Table 1.3, page 7).[6] Contact with anybody who has been jaundiced should always be noted. The sexual history should be obtained. A history of any injections (e.g. intravenous drugs, plasma transfusions, dental treatment or tattooing) in a patient who presents with symptoms of liver disease is important, particularly as hepatitis B or C may be transferred in this way.

Family history

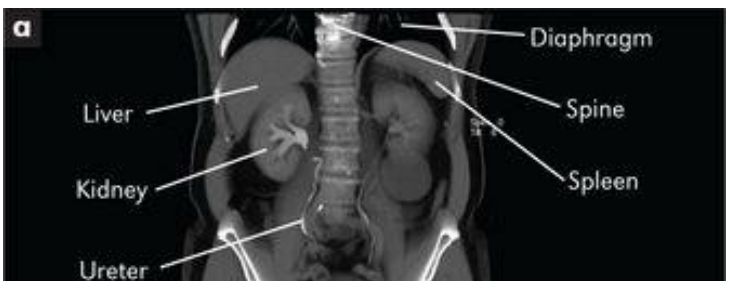
A family history of colon cancer, especially of familial polyps, or inflammatory bowel disease is important. Ask about coeliac disease in the family. A positive family history of jaundice, anaemia, splenectomy or cholecystectomy may occur in patients with haemolytic anaemia (due to haemoglobin abnormalities or auto-immune disease) or congenital or familial hyperbilirubinaemia.

The gastrointestinal examination

Examination of the gastrointestinal system includes a complete examination of the abdomen. It is also important to search for the peripheral signs of gastrointestinal and liver disease. Some signs are more useful than others.[7]

Examination anatomy

An understanding of the structure and function of the gastrointestinal tract and abdominal organs is critical for the diagnosis of gastrointestinal disease (Figure 6.1). The mouth is the gateway to the gastrointestinal tract. It and the anus and rectum are readily accessible to the examiner, and both must be examined carefully in any patient with suspected abdominal disease. The position of the abdominal organs can be quite variable, but there are important surface markings which should be kept in mind during the examination.



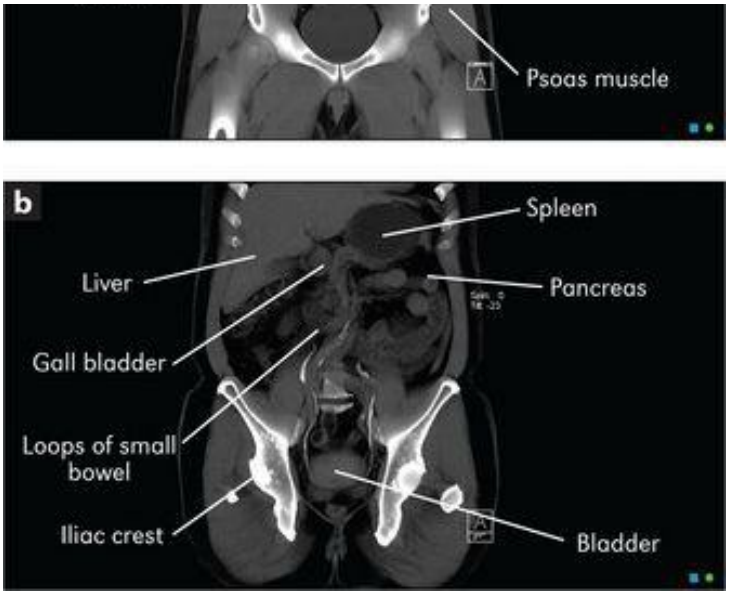


Figure 6.1 MRI scans of the abdomen
(a) Front. (b) Back.

Courtesy M Thomson, National Capital Diagnostic Imaging, Canberra.

The **liver** is the largest organ in the abdomen; it comprises a large right lobe and smaller left lobe divided into eight segments, including the caudate lobe (segment I) squeezed in between. The lower border of the liver extends from the tip of the right tenth rib to just below the left nipple. Normally the liver is not palpable but it may just be possible to feel the lower edge in healthy people.

The **spleen** is a lymphoid organ that underlies the ninth, tenth and eleventh ribs posteriorly on the left. It is usually not palpable in health.

The **kidneys** lie anteriorly 4 finger-breadths from the midline and posteriorly under the twelfth rib. Normally, the right kidney extends 2.5 cm lower than the left. In thin patients, the lower pole of the right kidney may be palpable in health.

The **gallbladder** is a pear-shaped organ and the fundus (top) is at the tip of the right ninth costal cartilage; it cannot be felt in health. The **pancreas** is situated in the retroperitoneum (behind the peritoneum), with the head tucked into the C-shaped duodenum and the tail snuggling into the spleen. A huge pancreatic mass may rarely be large enough to be palpable.

The **aorta** lies in the midline and terminates just to the left of the midline at the level of the iliac crest. A pulsatile mass in the middle of the abdomen is likely to be arising from the aorta and may indicate an aneurysm.

The **stomach** is usually J-shaped and lies in the left upper part of the abdomen over the spleen and pancreas; it connects with the duodenum. The **small intestine** ranges from 3 to 10 metres in length and comprises the upper half (duodenum and jejunum) and the lower half (ileum). The small intestine lies over the middle section of the abdomen but is usually impalpable.

lies over the middle section of the abdomen but is usually impalpable.

The **colon** is approximately 1.5 metres in length, and from right to left consists of the caecum, ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon, sigmoid colon, rectum and anal canal (anorectum). The **appendix** usually lies in the right lower abdominal area, arising posterior-medially from the caecum. The caecum and ascending colon lie on the right side of the abdomen, the transverse colon runs across the upper abdomen from right to left, and then the descending colon and sigmoid and rectum lie on the left side of the abdomen. Rarely, masses arising from the colon will be felt in the abdomen.

Other important anatomical areas include the **inguinal canal** and the **anorectum** which will be described later in this chapter in relation to examination of hernias and the rectal examination.

Positioning the patient

For the proper examination of the abdomen it is important that the patient be lying flat with the head resting on a single pillow (Figure 6.2). This relaxes the abdominal muscles and facilitates abdominal palpation. Helping the patient into this position affords the opportunity to make a general inspection.

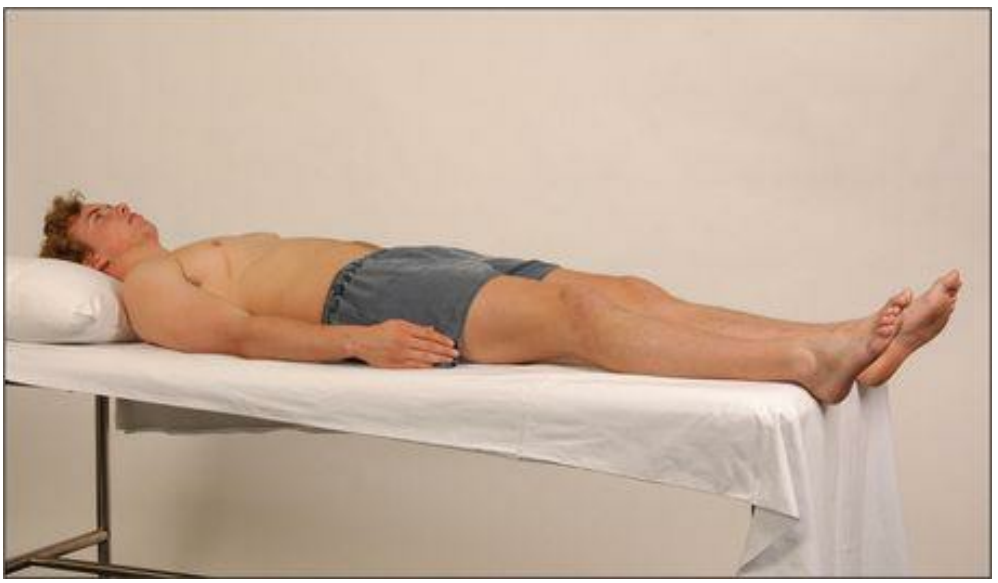


Figure 6.2 Gastrointestinal examination: positioning the patient

General appearance

Jaundice

Jaundice

The yellow discoloration of the sclerae (conjunctivae) and the skin that results from hyperbilirubinaemia is best observed in natural daylight (page 185). Whatever the underlying cause, the depth of jaundice can be quite variable.

Weight and wasting

The patient's weight must be recorded. Failure of the gastrointestinal tract to absorb food normally may lead to loss of weight and cachexia. This may also be the result of gastrointestinal malignancy or alcoholic cirrhosis. Folds of loose skin may be visible hanging from the abdomen and limbs. These suggest recent weight loss. Obesity can cause fatty infiltration of the liver (non-alcoholic steatohepatitis) and result in abnormal liver function tests. Anabolic steroid use can induce increase in muscle bulk (sometimes considered desirable) and various liver tumours, including adenomas or hepatocellular carcinomas.

Skin

The gastrointestinal tract and the skin have a common origin from the embryoblast. A number of diseases can present with both skin and gut involvement (Figures 6.3-6.8, Table 6.4).[8]

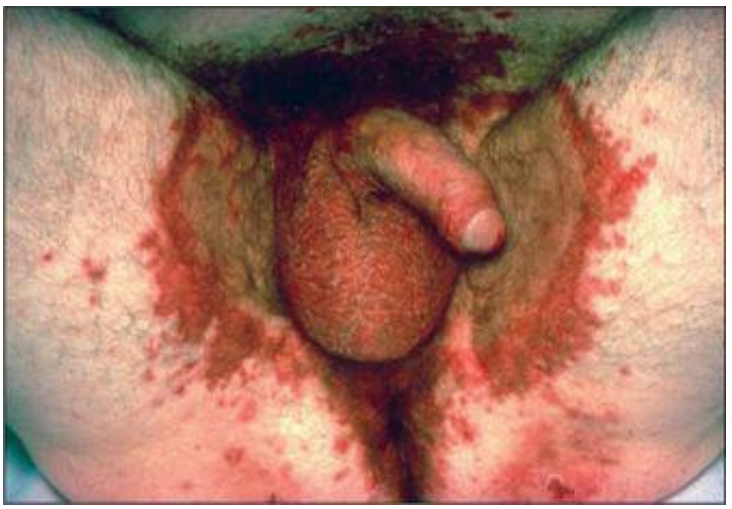


Figure 6.3 (above) Glucagonoma: migratory rash involving the groin

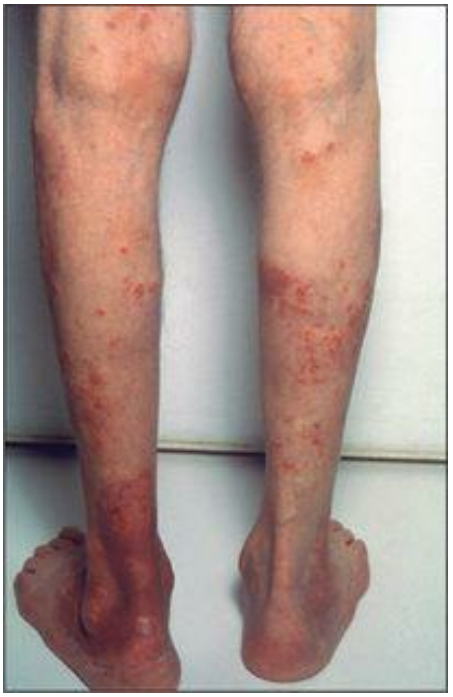


Figure 6.4 (right) Dermatitis herpetiformis

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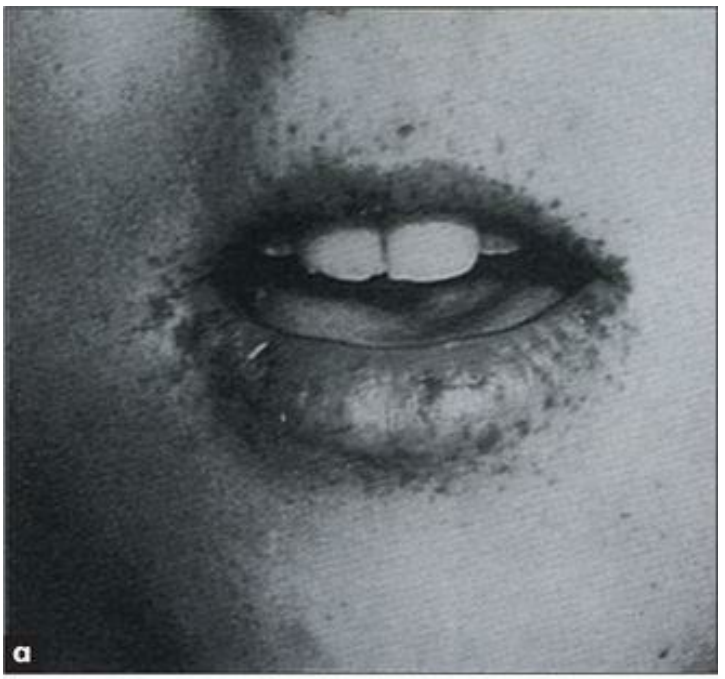


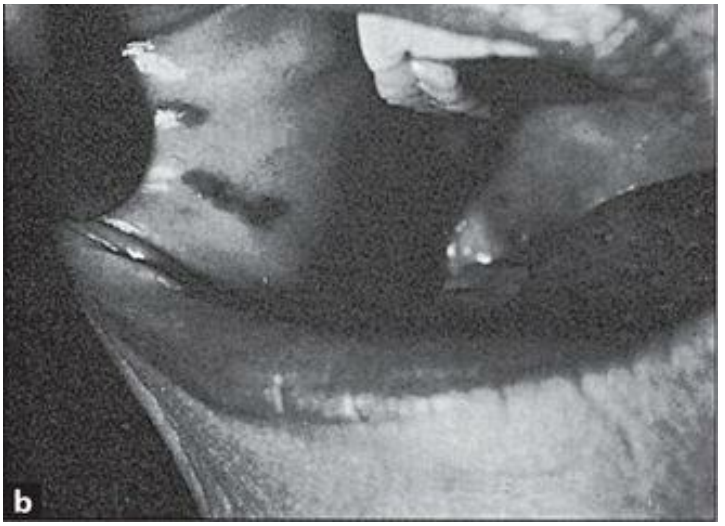
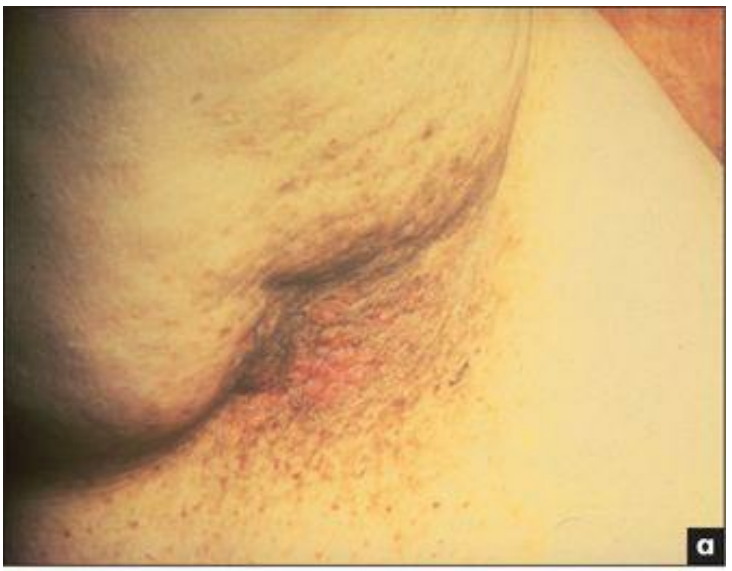


Figure 6.5 Peutz-Jeghers syndrome, with discrete brown-black lesions of the lips

Figure (a) from Jones DV et al, in Feldman M et al, Sleisenger & Fordtran's gastrointestinal disease, 6th edn, Chapter 112. Philadelphia: WB Saunders, 1998, with permission. Figure (b) from McDonald FS, ed., Mayo Clinic images in internal medicine, with permission. ©Mayo Clinic Scientific Press and CRC Press.



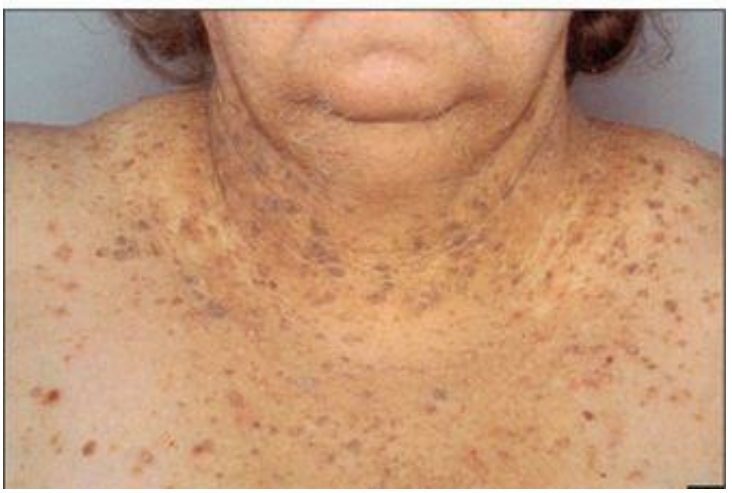


Figure 6.6 Acanthosis nigricans: (a) axilla; (b) chest wall

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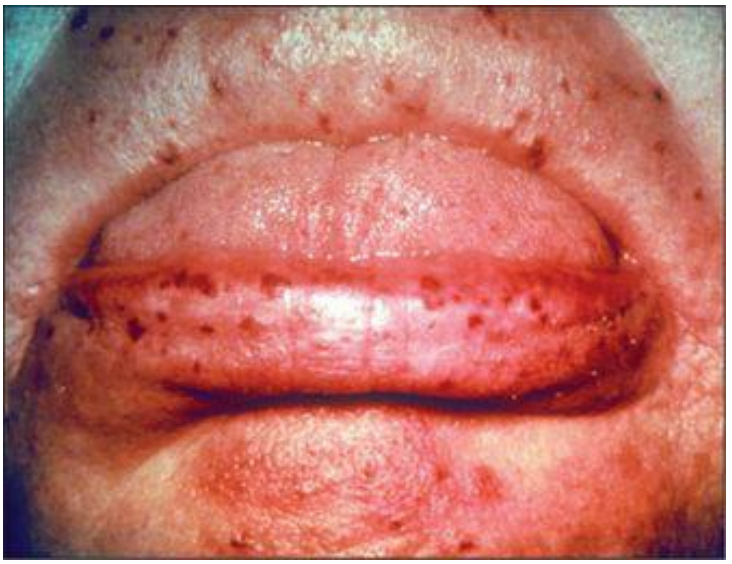

Figure 6.7 (right) Hereditary haemorrhagic telangiectasia involving the lips

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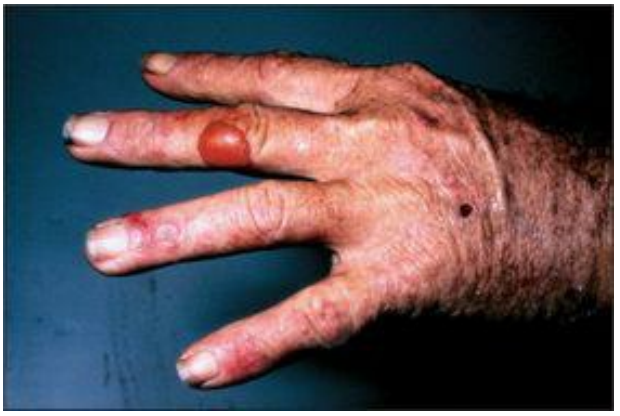

Figure 6.8 Porphyria cutanea tarda—scarring from photosensitivity

TABLE 6.4 The skin and the gut

Disease	Skin	Gut	Other associations

Disease	Skin	Gut	Other associations
Gastrointestinal polyposis syndromes			
Peutz-Jeghers syndrome (autosomal dominant)	Pigmented macules on hands, feet, lips	Hamartomatous polyps (rarely adenocarcinoma) in stomach, small bowel, large bowel	
Gardner's* syndrome (autosomal dominant)	Cysts, fibromas, lipomas (multiple)	Polyps, adenocarcinoma in large bowel	Bone osteomata
Cronkhite-Canada syndrome	Alopecia, hyperpigmentation, glossitis, dystrophic nails	Hamartomatous polyps, diarrhoea, exocrine pancreatic insufficiency	
Hormone-secreting tumours			
Carcinoid syndrome	Flushing, telangiectases	Watery diarrhoea, hepatomegaly	Wheeze, right heart murmurs
Systemic mastocytosis (due to mast cell proliferation and histamine release)	Telangiectases, flushing, pigmented papules, pruritus, dermographism, Darier's† sign (rub skin lesion with the blunt end of a pen: a palpable red wheal occurs minutes later)	Peptic ulcer, diarrhoea, malabsorption	Asthma, headache, tachycardia
Glucagonoma (glucagon-secreting tumour)	Migratory necrolytic rash (on flexural and friction areas)	Glossitis, weight loss, diabetes mellitus	
Vascular disorders			
Hereditary haemorrhagic telangiectasia (autosomal dominant)	Telangiectases (especially nail beds, palms, feet)	Gastrointestinal bleeding	Nasopharyngeal bleeding, pulmonary arteriovenous fistulas, high output cardiac failure
Pseudoxanthoma elasticum (autosomal recessive)	Yellow plaques/papules in flexural areas	Bowel bleeding, ischaemia	Angioid streaks in fundus
Blue rubber bleb syndrome	Hemangiomas (e.g. tongue)	Bleeding into bowel or liver	
Degos' disease (malignant atrophic papulosis)	Dome-shaped red papules (early), small porcelain white atrophic scars (late)	Intestinal perforation, infarction (primarily in young men—very rare)	
Disease	**Skin**	**Gut**	**Other associations**
Vascular disorders (continued)			
Acanthosis nigricans	Brown to black skin papillomas (usually axillae)	Carcinoma	Acromegaly, diabetes mellitus
Dermatitis herpetiformis	Pruritic vesicles—typically on knees, elbows, buttocks	Coeliac disease	
Zinc deficiency	Red, scaly, crusting lesions around mouth, eyes, genitalia; white patches on tongue	Diarrhoea (zinc deficiency occurs particularly in setting of Crohn's disease with fistulas, cirrhosis, parenteral nutrition, pancreatitis)	
Porphyria cutanea tarda	Vesicles on exposed skin (e.g. hands)	Alcoholic liver disease	
Inflammatory bowel disease	Pyoderma gangrenosum		
	Erythema nodosum		
	Clubbing		
	Mouth ulcers		
Haemochromatosis (autosomal recessive)	Skin pigmentation (bronze)	Hepatomegaly, signs of chronic liver disease	Diabetes mellitus, heart failure (cardiomyopathy), arthropathy, testicular atrophy
Systemic sclerosis	Skin that is thick and bound down, calcinosis, Raynaud's‡ phenomenon, sclerodactyly, telangiectases	Gastro-oesophageal reflux, oesophageal dysmotility, small bowel bacterial overgrowth with malabsorption	

* Eldon John Gardner (b. 1909), American geneticist.
† Ferdinand Jean Darier (1856–1938), Paris dermatologist.
‡ Maurice Raynaud (1834–1881), Paris physician.

Pigmentation

Generalised skin pigmentation can result from chronic liver disease, especially in haemochromatosis (due to haemosiderin-stimulating melanocytes, to produce melanin). Malabsorption may result in Addisonian-type pigmentation ('sunkissed' pigmentation) of the nipples, palmar creases, pressure areas and

mouth (page 311).

Peutz-Jeghers[c] syndrome

Freckle-like spots (discrete, brown-black lesions) around the mouth and on the buccal mucosa (Figure 6.5) and on the fingers and toes, are associated with hamartomas of the small bowel (50%) and colon (30%), which can present with bleeding or intussusception. In this autosomal dominant condition the incidence of gastrointestinal adenocarcinoma is increased.

Acanthosis nigricans

These are brown-to-black velvety elevations of the epidermis due to confluent papillomas and are usually found in the axillae and nape of the neck (Figure 6.6). Acanthosis nigricans is associated rarely with gastrointestinal carcinoma (particularly stomach) and lymphoma, as well as with acromegaly, diabetes mellitus and other endocrinopathies.

Hereditary haemorrhagic telangiectasia (Rendu-Osler-Weber syndrome[d])

Multiple small telangiectasia occur in this disease. They are often present on the lips and tongue (Figure 6.7), but may be found anywhere on the skin. When they are present in the gastrointestinal tract they can cause chronic blood loss or even, occasionally, torrential bleeding. An associated arteriovenous malformation in the liver may be present. This is an autosomal dominant condition and is uncommon.

Porphyria cutanea tarda

Fragile vesicles appear on exposed areas of the skin and heal with scarring (Figure 6.8). The urine is dark in this chronic disorder of porphyrin metabolism associated with alcoholism, liver disease and hepatitis C.

Systemic sclerosis

Tense tethering of the skin in systemic sclerosis is often associated with gastro-oesophageal reflux and gastrointestinal motility disorders (page 285).

Mental state

Assess orientation (page 380). The syndrome of hepatic encephalopathy, due to decompensated advanced cirrhosis (chronic liver failure) or fulminant hepatitis (acute liver failure), is an organic neurological disturbance. The features depend on the aetiology and the precipitating factors (page 188). Patients eventually become stuporous and then comatose. The combination of hepatocellular damage and portosystemic shunting due to disturbed hepatic structure (both extrahepatic and intrahepatic) causes this syndrome. It is probably related to the liver's failure to remove toxic metabolites from the portal blood. These toxic metabolites may include ammonia, mercaptans, short-chain fatty acids and amines.

The hands

Even the experienced gastroenterologist must restrain his or her excitement and begin the examination of the gastrointestinal tract with the hands. The signs that may be elicited here give a clue to the presence of chronic liver disease. Whatever its aetiology, permanent diffuse liver damage results in similar peripheral signs. However, none of these signs alone is specific for chronic liver disease.

Nails

Leuconychia

When chronic liver or other disease results in hypoalbuminaemia, the nail beds opacify (the abnormality is of the nail bed and not of the nail), often leaving only a rim of pink nail bed at the top of the nail (Terry's nails; eFigure 6.9). The thumb and index nails are most often involved. The exact mechanism is uncertain. It may be that the explanation is compression of capillary flow by extracellular fluid.

Figure 6.9 Leuconychia—Terry's nails

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Muehrcke's lines (transverse white lines) can also occur in hypoalbuminaemic states, including cirrhosis. Blue lunulae may be seen in patients with Wilson's disease (hepatolenticular degeneration).

Clubbing

Up to one-third of patients with cirrhosis may have finger clubbing. In at least some cases, this may be related to arteriovenous (AV) shunting in the lungs, resulting in arterial oxygen desaturation. Cyanosis may be associated with severe long-standing chronic liver disease. The cause of this pulmonary AV shunting is unknown. Conditions such as inflammatory bowel disease and coeliac disease, which cause long-standing nutritional depletion, can also cause clubbing.

The palms

Palmar erythema ('liver palms')

This is reddening of the palms of the hands affecting the thenar and hypothenar eminences. Often the soles of the feet are also affected. This can be a feature of chronic liver disease. While the finding has been attributed to raised oestrogen levels, it has not been shown to be related to plasma oestradiol levels, so the aetiology remains uncertain. Palmar erythema can also occur with pregnancy, thyrotoxicosis, rheumatoid arthritis,

polycythaemia and rarely with chronic febrile diseases or chronic leukaemia. It may also be a normal finding, especially in women, and like spider naevi can occur in pregnancy.

Anaemia

Inspect the palmar creases for pallor suggesting anaemia, which may result from gastrointestinal blood loss, malabsorption (folate, vitamin B_{12}), haemolysis (e.g. hypersplenism) or chronic disease.

Dupuytren's contracture[f]

This is a visible and palpable thickening and contraction of the palmar fascia causing permanent flexion, most often of the ring finger. It is often bilateral and occasionally affects the feet. It is associated with alcoholism (not liver disease), but is also found in some manual workers; it may be familial. The palmar fascia of these patients contains abnormally large amounts of xanthine, and this may be related to the pathogenesis.

Hepatic flap (asterixis[g])

Before leaving the hands one should ask the patient to stretch out the arms in front, separate the fingers and extend the wrists for 15 seconds. Jerky, irregular flexion–extension movement at the wrist and metacarpophalangeal joints, often accompanied by lateral movements of the fingers, constitute the flapping of hepatic encephalopathy. It is thought to be due to interference with the inflow of joint position sense information to the reticular formation in the brainstem. This results in rhythmical lapses of postural muscle tone. Occasionally the arms, neck, tongue, jaws and eyelids can also be involved. It can be demonstrated if the patient is asked to close the eyes forcefully or to protrude the tongue. The flap is usually bilateral, tends to be absent at rest, and is brought on by sustained posture. The rhythmic movements are not synchronous on each side and the flap is absent when coma supervenes.

Although this flap is a characteristic and early sign of liver failure, it is not diagnostic: it can also occur in cardiac, respiratory and renal failure, as well as in hypoglycaemia, hypokalaemia, hypomagnesaemia or barbiturate intoxication.

An apparent tremor (really a form of choreoathetosis, page 399) may occur in Wilson's disease. A fine resting tremor is common in alcoholism.

The arms

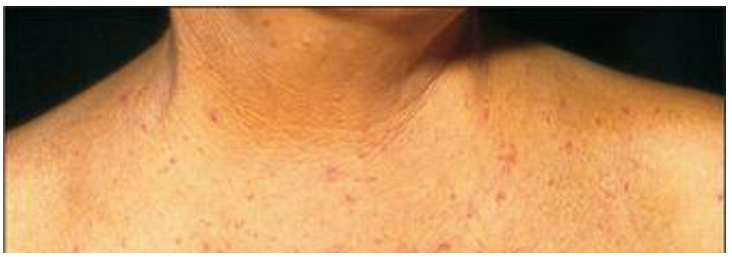
Inspect the upper limbs for *bruising*. Large bruises (ecchymoses) may be due to clotting abnormalities. Hepatocellular damage can interfere with protein synthesis and therefore the production of all the clotting factors (except factor VIII, which is made elsewhere in the reticuloendothelial system). Obstructive jaundice results in a shortage of bile acids in the intestine, and therefore may reduce absorption of vitamin K (a fat-soluble vitamin), which is essential for the production of clotting factors II (prothrombin), VII, IX and X.

Petechiae (pinhead-sized bruises) may also be present. Chronic excessive alcohol consumption can sometimes result in bone marrow depression, causing thrombocytopenia, which may be responsible for petechiae. In addition, splenomegaly secondary to portal hypertension can cause hypersplenism, with resultant excessive destruction of platelets in the spleen; in severe liver disease (especially acute hepatic necrosis), diffuse intravascular coagulation can occur.

Look for muscle wasting, which is often a late manifestation of malnutrition in alcoholic patients. Alcohol can also cause a proximal myopathy (page 391).

Scratch marks due to severe itch (pruritus) are often prominent in patients with obstructive or cholestatic jaundice. This is commonly the presenting feature of primary biliary cirrhosis[h] before other signs are apparent. The mechanism of pruritus is thought to be retention of an unknown substance normally excreted in the bile, rather than bile salt deposition in the skin as was earlier thought.

Spider naevi[i] (Figure 6.10) consist of a central arteriole from which radiate numerous small vessels which look like spiders' legs. They range in size from just visible to half a centimetre in diameter. Their usual distribution is in the area drained by the superior vena cava, so they are found on the arms, neck and chest wall. They can occasionally bleed profusely. Pressure applied with a pointed object to the central arteriole causes blanching of the whole lesion. Rapid refilling from the centre to the legs occurs on release of the pressure.



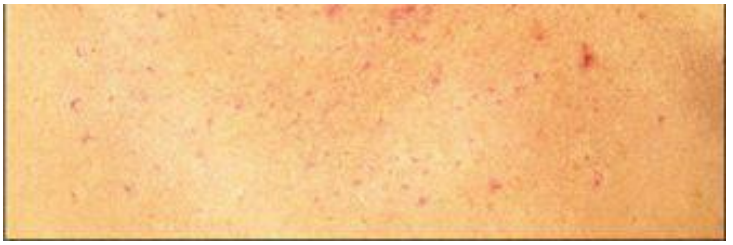


Figure 6.10 A large crop of spider naevi

The finding of more than two or three spider naevi anywhere on the body is likely to be abnormal. Spider naevi can be caused by cirrhosis, most frequently due to alcohol. In patients with cirrhosis the number of spider naevi may increase or decrease as the patient's condition changes, as does palmar erythema. They may occur transiently with viral hepatitis. During the second to fifth months of pregnancy, spider naevi frequently appear, only to disappear again within days of delivery. It is not known why they occur only in the upper part of the body, but it may be related to the fact that this is the part of the body where flushing usually occurs. Like palmar erythema they are traditionally attributed to oestrogen excess. Part of the normal hepatic function is the inactivation of oestrogens, which is impaired in chronic liver disease. Oestrogens are known to have a dilating effect on the spiral arterioles of the endometrium, and this has been used to explain the presence of spider naevi, but changes in plasma oestradiol levels have not been found to correlate with the appearance and disappearance of spider naevi.

The differential diagnosis of spider naevi includes *Campbell de Morgan spots,* venous stars and hereditary haemorrhagic telangiectasia. Campbell de Morgan spots are flat or slightly elevated red circular lesions that occur on the abdomen or the front of the chest. They do not blanch on pressure and are very common. *Venous stars* are 2- to 3-cm lesions that can occur on the dorsum of the feet, legs, back and the lower chest. They are due to elevated venous pressure and are found overlying the main tributary to a large vein. They are not obliterated by pressure. The blood flow is from the periphery to the centre of the lesion, which is the opposite of the flow in the spider naevus. Lesions of *hereditary haemorrhagic telangiectasia* (page 228) occasionally resemble spider naevi.

Palpate the axillae for *lymphadenopathy* (page 229). Look in the axillae for acanthosis nigricans.

The face

Eyes

Look first at the sclerae for signs of *jaundice* (Figure 6.11) or *anaemia*. Bitot's[k] spots are yellow keratinised areas on the sclera (Figure 6.12). They are the result of severe vitamin A deficiency due to malabsorption or malnutrition. Retinal damage and blindness may occur as a later development. *Kayser-Fleischer rings*[l] (Figure 6.13) are brownish green rings occurring at the periphery of the cornea, affecting the upper pole more than the lower. They are due to deposits of excess copper in Descemet's membrane[m] of the cornea. Slit-lamp examination is often necessary to show them. They are typically found in Wilson's disease,[n] a copper storage disease that causes cirrhosis and neurological disturbances. The Kayser-Fleischer rings are usually present by the time neurological signs have appeared. Patients with other cholestatic liver diseases, however, can also have these rings. *Iritis* may be seen in inflammatory bowel disease (page 191).

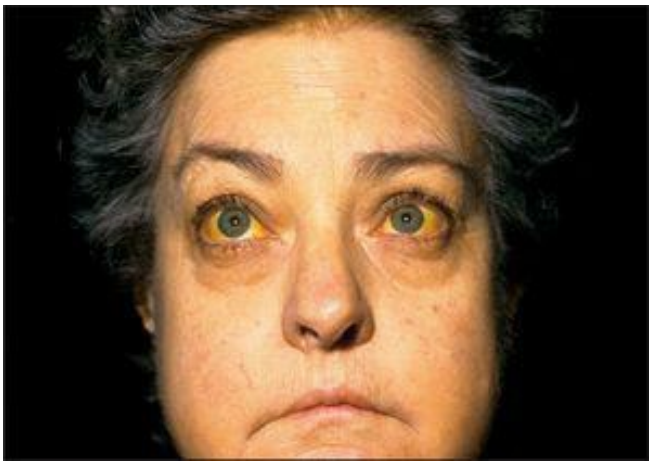


Figure 6.11 Scleral icterus

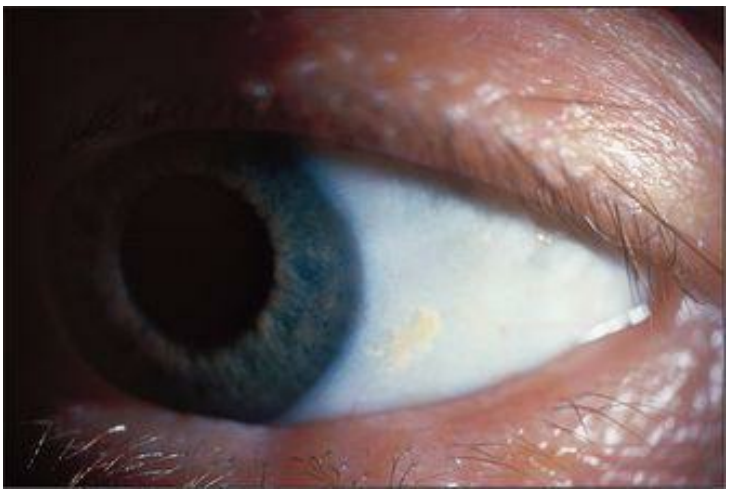


Figure 6.12 Bitot spot: focal area of conjunctival xerosis with a foamy appearance

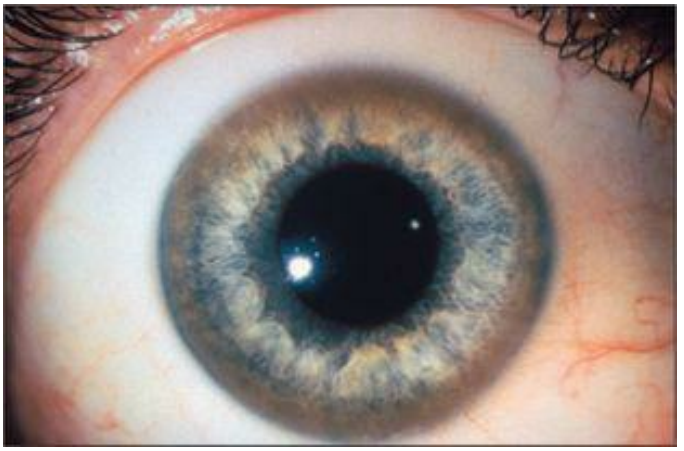


Figure 6.13 Kayser-Fleischer rings

Xanthelasma are yellowish plaques in the subcutaneous tissues in the periorbital region and are due to deposits of lipids (see Figure 4.19, page 57). They may indicate protracted elevation of the serum cholesterol. In patients with cholestasis, an abnormal lipoprotein (lipoprotein X) is found in the plasma and is associated with elevation of the serum cholesterol. Xanthelasma are common in patients with primary biliary cirrhosis.

Periorbital purpura following proctosigmoidoscopy ('black eye syndrome') is a characteristic sign of amyloidosis (perhaps related to factor X deficiency) but is exceedingly rare (Figure 6.14).

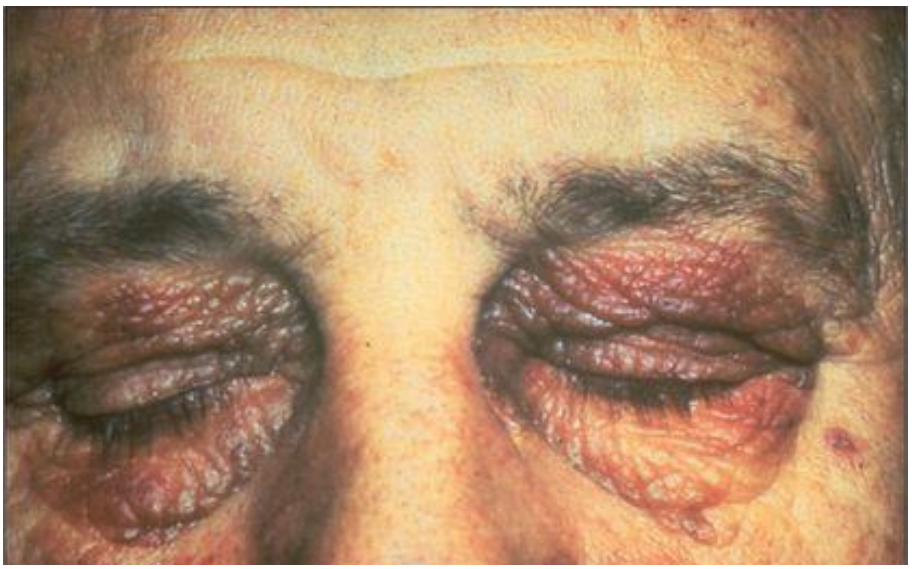


Figure 6.14 Amyloidosis causing periorbital purpura
Note the periorbital purpura that followed a proctoscopic examination, a characteristic (albeit rare) sign.

Salivary glands

Next inspect and palpate the cheeks over the parotid area for *parotid enlargement* (Table 6.5). Ask the patient to clench the teeth so that the masseter muscle is palpable; the normal parotid gland is impalpable but the enlarged gland is best felt behind the masseter muscle and in front of the ear. Parotidomegaly that is bilateral is associated with alcoholism rather than liver disease per se. It is due to fatty infiltration, perhaps secondary to alcohol toxicity with or without malnutrition. A tender, warm, swollen parotid suggests the diagnosis of parotiditis following an acute illness or surgery. A mixed parotid tumour (a pleomorphic adenoma) is the commonest cause of a lump. Parotid carcinoma may cause a facial nerve palsy (page 343). Feel in the mouth for a parotid calculus, which may be present at the parotid duct orifice (opposite the upper second molar). Mumps also causes acute parotid enlargement which is usually bilateral.

TABLE 6.5 Causes of parotid enlargement

Bilateral
1. Mumps (can be unilateral)
2. Sarcoidosis or lymphoma, which may cause painless bilateral enlargement
3. Mikulicz* syndrome: bilateral painless enlargement of all three salivary glands. This disease is probably an early stage of Sjögren's syndrome

5. Alcohol-associated parotitis

6. Malnutrition

7. Severe dehydration: as occurs in renal failure, terminal carcinomatosis and severe infections

Unilateral

1. Mixed parotid tumour (occasionally bilateral)

2. Tumour infiltration, which usually causes painless unilateral enlargement and may cause facial nerve palsy

3. Duct blockage, e.g. salivary calculus

* Johann von Mikulicz-Radecki (1850–1905), professor of surgery, Breslau. He described this condition in 1892.

Submandibular gland enlargement is most often due to a calculus. This may be palpable bimanually (Figure 6.15). The examiner's gloved index finger is placed on the floor of the mouth beside the tongue, feeling between it and fingers placed behind the body of the mandible. It may also be enlarged in chronic liver disease.

Figure 6.15 Examination of the submandibular gland

The mouth

The teeth and breath

The very beginning of the gastrointestinal tract is, like the very end of the tract, accessible to inspection without elaborate equipment.[9] Look first briefly at the state of the teeth and note whether they are real or false. False teeth will have to be removed for a complete examination of the mouth. Note whether there is gum hypertrophy (Table 6.6) or pigmentation (Table 6.7). Loose-fitting false teeth may be responsible for ulcers and decayed teeth may be responsible for fetor (bad breath).

TABLE 6.6 Causes of gum hypertrophy

1 Phenytoin
2 Pregnancy
3 Scurvy (vitamin C deficiency: the gums become spongy, red, bleed easily and are swollen and irregular)
4 Gingivitis, e.g. from smoking, calculus, plaque, Vincent's* angina (fusobacterial membranous tonsillitis)
5 Leukaemia (usually monocytic)

* Jean Hyacinthe Vincent (1862–1950), professor of forensic medicine and French Army bacteriologist, described this in 1898.

TABLE 6.7 Causes of pigmented lesions in the mouth

1 Heavy metals: lead or bismuth (blue-black line on the gingival

margin), iron (haemochromatosis—blue-grey pigmentation of the hard palate)
2 Drugs: antimalarials, the oral contraceptive pill (brown or black areas of pigmentation anywhere in the mouth)
3 Addison's disease (blotches of dark brown pigment anywhere in the mouth)
4 Peutz-Jeghers syndrome (lips, buccal mucosa or palate)
5 Malignant melanoma (raised, painless black lesions anywhere in the mouth)

Other causes of fetor are listed in Table 6.8. These must be distinguished from *fetor hepaticus* which is a rather sweet smell of the breath. It is an indication of severe hepatocellular disease and may be due to methylmercaptans. These substances are known to be exhaled in the breath and may be derived from methionine when this amino acid is not demethylated by a diseased liver. Severe fetor hepaticus that fills the patient's room is a bad sign and indicates a precomatose condition in many cases. The presence of fetor hepaticus in a patient with a coma of unknown cause may be a helpful clue to the diagnosis.

TABLE 6.8 Causes of fetor (bad breath)

1 Faulty oral hygiene
2 Fetor hepaticus (a sweet smell)
3 Ketosis (diabetic ketoacidosis results in excretion of ketones in exhaled air, causing a sickly sweet smell)
4 Uraemia (fish breath: an ammoniacal odour)
5 Alcohol (distinctive)
6 Paraldehyde

7 Putrid (due to anaerobic chest infections with large amounts of sputum)
8 Cigarettes

Unless the smell is obvious one should get a patient to exhale through the mouth while one sniffs a little of the exhaled air.

The tongue

Thickened epithelium with bacterial debris and food particles commonly causes a *coating* over the tongue, especially in smokers. It is rarely a sign of disease and is more marked on the posterior part of the tongue where there is less mobility and the papillae desquamate more slowly. It occurs frequently in respiratory tract infections, but is in no way related to constipation or any serious abdominal disorder.

Lingua nigra (black tongue) is due to elongation of papillae over the posterior part of the tongue, which appears dark brown because of the accumulation of keratin. There is no known cause and apart from its aesthetic problems it is symptomless. Bismuth compounds may also cause a black tongue.

Geographical tongue is a term used to describe slowly changing red rings and lines that occur on the surface of the tongue. It is not painful, and the condition tends to come and go. It is not usually of any significance, but can be a sign of riboflavin (vitamin B_2) deficiency.

Leucoplakia is white-coloured thickening of the mucosa of the tongue and mouth; the condition is premalignant. Most of the causes of leucoplakia begin with 'S': sore teeth (poor dental hygiene), smoking, spirits, sepsis or syphilis, but often no cause is apparent. Leucoplakia may also occur on the larynx, anus and vulva.

The term *glossitis* is generally used to describe a smooth appearance of the tongue which may also be erythematous. The appearance is due to atrophy of the papillae, and in later stages there may be shallow ulceration. These changes occur in the tongue often as a result of nutritional deficiencies to which the tongue is sensitive because of the rapid turnover of mucosal cells. Deficiencies of iron, folate and the vitamin B group, especially vitamin B_{12}, are common causes. Glossitis is common in alcoholics and can also occur in the rare carcinoid syndrome. However, many cases, especially those in elderly people, are impossible to explain.

Enlargement of the tongue (*macroglossia*) may occur in congenital conditions such as Down syndrome (page 314) or in endocrine disease, including acromegaly (page 307). Tumour infiltration (e.g. haemangioma or

lymphangioma) or infiltration of the tongue with amyloid material in amyloidosis can also be responsible for macroglossia.

Mouth ulcers

This is an important topic because a number of systemic diseases can present with ulcers in the mouth (Table 6.9). *Aphthous ulceration* is the commonest type seen (Figure 6.16). This begins as a small painful vesicle on the tongue or mucosal surface of the mouth, which may break down to form a painful, shallow ulcer. These ulcers heal without scarring. The cause is completely unknown. They usually do not indicate any serious underlying systemic disease, but may occur in Crohn's[o] disease or coeliac disease. HIV infection may be associated with a number of mouth lesions (page 457) . *Angular stomatitis* refers to cracks at the corners of the mouth; causes include deficiencies in vitamin B_6, vitamin B_{12}, folate and iron.

TABLE 6.9 Causes of mouth ulcers

Common Aphthous Drugs (e.g. gold, steroids) Trauma
Uncommon Gastrointestinal disease: Crohn's disease, ulcerative colitis, coeliac disease Rheumatological disease: Behçet's[*] syndrome, Reiter's[†] syndrome Erythema multiforme Infection: viral—herpes zoster, herpes simplex; bacterial—

syphilis (primary chancre, secondary snail track ulcers, mucous patches), tuberculosis Self-inflicted

* Halusi Behçet (1889–1948), Turkish dermatologist. He described the disease in 1937.

† Hans Reiter (1881–1969), Berlin bacteriologist, described this in 1916.

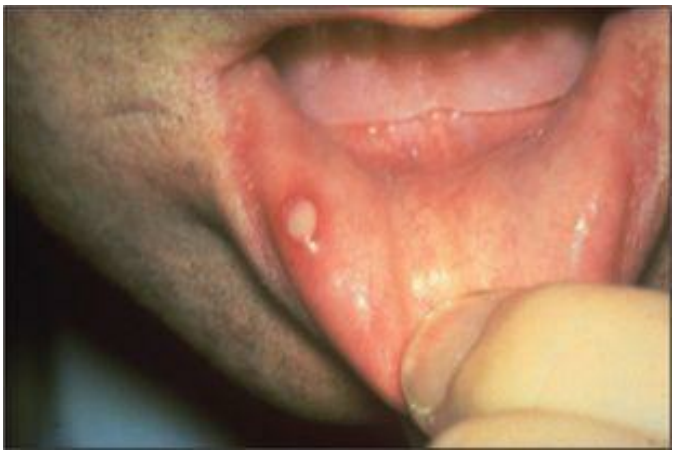


Figure 6.16 Aphthous ulceration

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Candidiasis (moniliasis)

Fungal infection with *Candida albicans* (thrush) causes creamy white curd-like patches in the mouth which are removed only with difficulty and leave a bleeding surface. The infection may spread to involve the oesophagus, causing dysphagia or odynophagia. Moniliasis is associated with immunosuppression (steroids, tumour chemotherapy, alcoholism or an underlying immunological abnormality such as HIV infection, or haematological malignancy), where it is due to decreased host resistance. Broad-spectrum antibiotics, which inhibit the normal oral flora, are also a common cause, because fungal overgrowth is permitted. Faulty oral hygiene, iron deficiency and diabetes mellitus can also be responsible. Rarely, chronic mucocutaneous candidiasis, a distinct syndrome comprising recurrent or persistent oral thrush, fingernail or toenail bed infection and skin involvement, occurs; in some of these patients, endocrine diseases such as hypoparathyroidism, hypothyroidism or Addison's disease are associated (page 305).

The neck and chest

Palpate the cervical lymph nodes. It is particularly important to feel for the supraclavicular nodes, especially on the left side. These may be involved with advanced gastric or other gastrointestinal malignancy, or with lung cancer. The presence of a large left supraclavicular node (Virchow's node) in combination with carcinoma of the stomach is called Troisier's sign.[p] Look for spider naevi.

In males, *gynaecomastia* may be a sign of chronic liver disease. Gynaecomastia may be unilateral or bilateral and the breasts may be tender (Figure 6.17). This may be a sign of cirrhosis, particularly alcoholic cirrhosis, or of chronic autoimmune hepatitis. In chronic liver disease, changes in the oestradiol-to-testosterone ratio may be responsible. In cirrhotic patients, spironolactone, used to treat ascites, is also a common cause. Gynaecomastia may also occur in alcoholics without liver disease because of damage to the Leydig cells[q] of the testis from alcohol. A number of drugs may rarely cause gynaecomastia (e.g. digoxin, cimetidine).

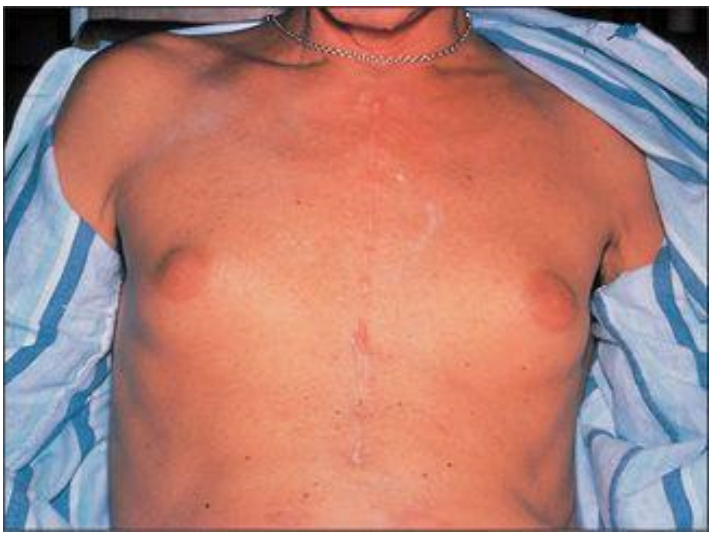


Figure 6.17 Gynaecomastia with prominent breasts and unassociated with confounding obesity

From Mir MA, Atlas of Clinical Diagnosis, 2nd edn. Edinburgh: Saunders, 2003, with permission.

The abdomen

Self-restraint is no longer required and it is now time to examine the abdomen

itself.

Inspection

The patient should lie flat, with one pillow under the head and the abdomen exposed from the nipples to the pubic symphysis (see Figure 6.2). It may be preferable to expose this area in stages to preserve the patient's dignity.

Does the patient appear unwell? The patient with an *acute abdomen* may be lying very still and have shallow breathing (page 186).

Inspection begins with a careful look for abdominal *scars,* which may indicate previous surgery or trauma (Figure 6.18). Look in the area around the umbilicus for laparoscopic surgical scars. Older scars are white and recent scars are pink because the tissue remains vascular. Note the presence of stomata (end-colostomy, loop colostomy, ileostomy or ileal conduit) or fistulae. There may be visible abdominal striae following weight loss.

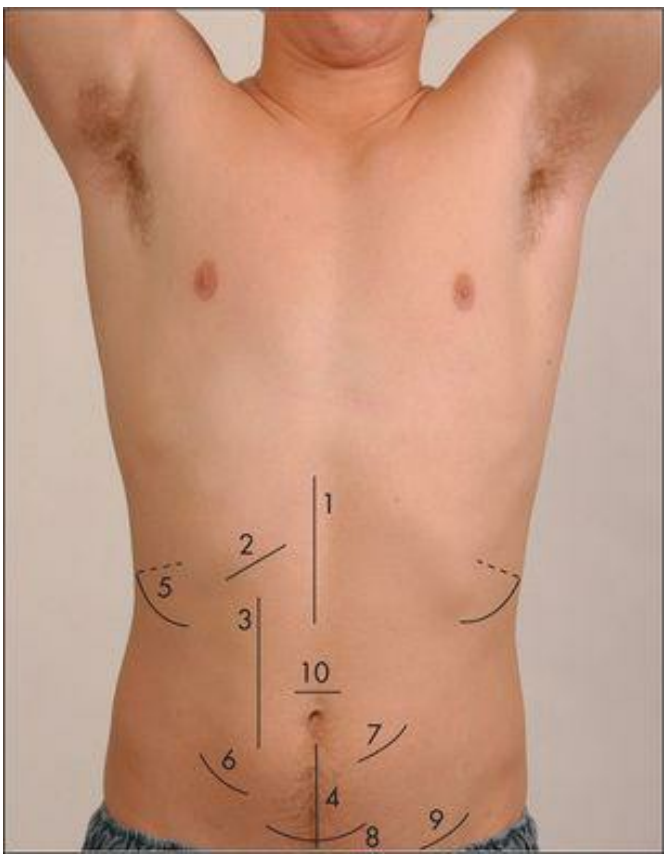


Figure 6.18 Abdominal scars
Note: Laparoscopic surgical scars are now common. Most of these procedures include a port about 2 cm in length, just above the umbilicus.

1. Upper midline

2. Right subcostal (Kocher's)

3. Right paramedian
4. Lower midline
5. Nephrectomy
6. Appendicectomy (Gridiron)
7. Transplanted kidney
8. Suprapubic (Pfannensteil)
9. Left inguinal
10. Umbilical port—laparoscopic surgery

Generalised abdominal *distension* (Figure 6.19) may be present. All the causes of this sound as if they begin with the letter 'F': fat (gross obesity), fluid (ascites), fetus, flatus (gaseous distension due to bowel obstruction), faeces, 'filthy' big tumour (e.g. ovarian tumour or hydatid cyst) or 'phantom' pregnancy. Look at the shape of the umbilicus, which may give a clue to the underlying cause. An umbilicus buried in fat suggests that the patient eats too much. However, when the peritoneal cavity is filled with large volumes of fluid (ascites) from whatever cause, the abdominal flanks and wall appear tense and the umbilicus is shallow or everted and points downwards. In pregnancy the umbilicus is pushed upwards by the uterus enlarging from the pelvis. This appearance may also result from a huge ovarian cyst.

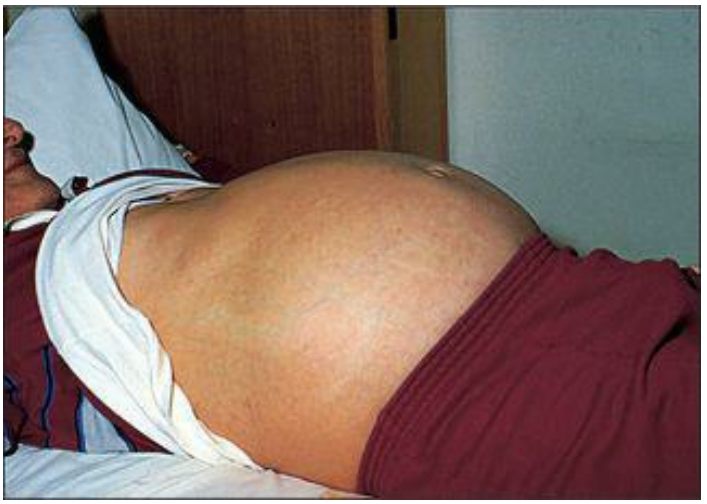


Figure 6.19 Abdomen distended with ascites: umbilicus points downwards, unlike cases of distension due to a pelvic mass

Local *swellings* may indicate enlargement of one of the abdominal or pelvic organs. A hernia is a protrusion of an intra-abdominal structure through an abnormal opening; this may occur because of weakening of the abdominal wall by previous surgery (incisional hernia), a congenital abdominal wall defect, or chronically increased intra-abdominal pressure.

Prominent *veins* may be obvious on the abdominal wall. If these are present, the direction of venous flow should be elicited at this stage. A finger is used to occlude the vein and blood is then emptied from the vein below the occluding finger with a second finger. The second finger is removed and, if the vein refills, flow is occurring towards the occluding finger (Figure 6.20). Flow should be tested separately in veins above and below the umbilicus. In patients with severe portal hypertension, portal to systemic flow occurs through the umbilical veins, which may become engorged and distended (Figure 6.21). The direction of flow then is away from the umbilicus. Because of their engorged appearance they have been likened to the mythical Medusa's hair after Minerva had turned it into snakes; this sign is called a *caput Medusae* (head of Medusa) but is very rare (Figure 6.22). Usually only one or two veins (often epigastric) are visible. Engorgement can also occur because of inferior vena caval obstruction, usually due to a tumour or thrombosis but sometimes because of tense ascites. In this case the abdominal veins enlarge to provide collateral blood flow from the legs, avoiding the blocked inferior vena cava. The direction of flow is then upwards towards the heart. Therefore, to distinguish caput Medusae from inferior vena caval obstruction, determine the direction of flow *below* the umbilicus; it will be towards the legs in the former and towards the head in the latter. Prominent superficial veins can occasionally be congenital.

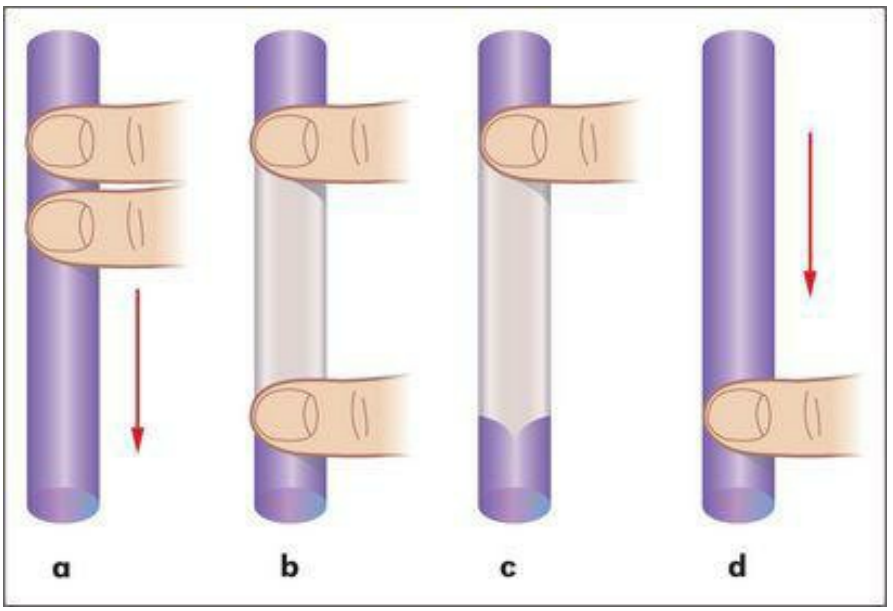


Figure 6.20 Detecting the direction of flow of a vein
(a) Place two fingers firmly on the vein. (b) The second finger is moved along the vein to empty it of blood and keep it occluded. (c) The second finger is removed but the vein does not refill. (d) At repeat testing and removing the first finger, filling occurs, indicating the direction of flow.

Adapted from Swash M, ed. Hutchison's clinical methods, 20th edn. Philadelphia: Baillière Tindall, 1995, with permission.

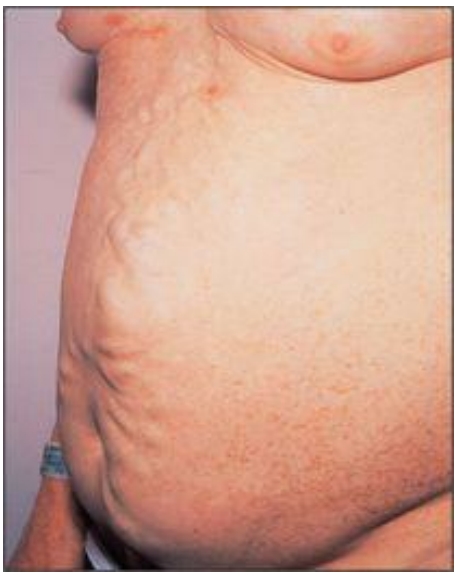
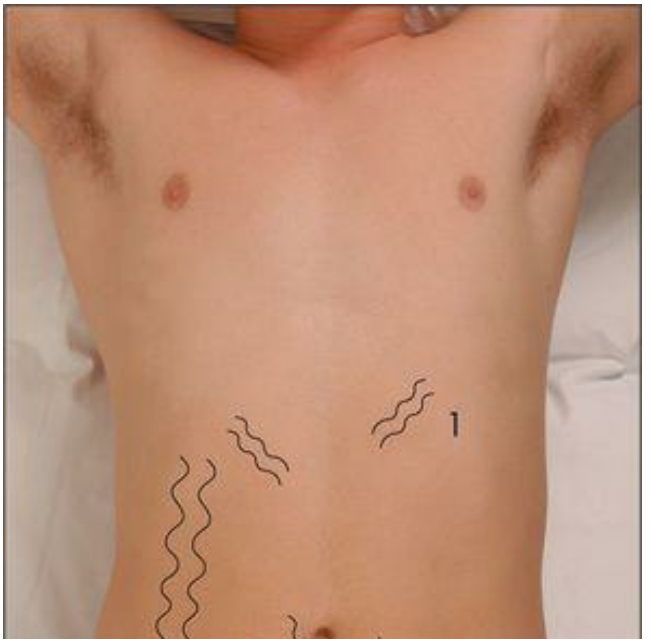


Figure 6.21 Distended abdominal veins in a patient with portal hypertension

From Mir MA, Atlas of Clinical Diagnosis, 2nd edn. Edinburgh: Saunders, 2003, with permission.



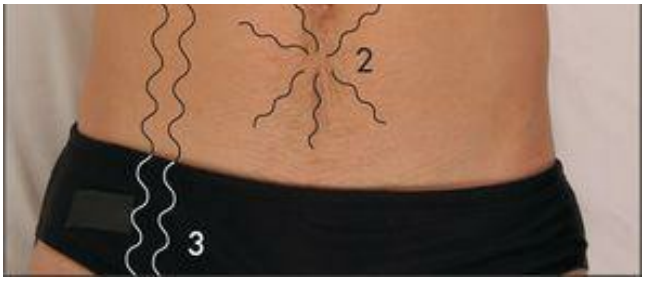


Figure 6.22 Prominent veins on the abdominal wall
1 = thin veins over the costal margin—not of clinical relevance. 2 = caput Medusae. 3 = inferior vena caval obstruction.

Based on Swash M, ed. Hutchison's clinical methods, 20th edn. Philadelphia: Baillière Tindall, 1995.

Pulsations may be visible. An expanding central pulsation in the epigastrium suggests an abdominal aortic aneurysm. However, the abdominal aorta can often be seen to pulsate in normal thin people.

Visible peristalsis may occur occasionally in very thin normal people; however, it usually suggests intestinal obstruction. Pyloric obstruction due to peptic ulceration or tumour may cause visible peristalsis, seen as a slow wave of movement passing across the upper abdomen from left to right. Obstruction of the distal small bowel can cause similar movements in a ladder pattern in the centre of the abdomen.

Skin lesions should also be noted on the abdominal wall. These include the vesicles of herpes zoster, which occur in a radicular pattern (they are localised to only one side of the abdomen in the distribution of a single nerve root). Herpes zoster may be responsible for severe abdominal pain that is of mysterious origin until the rash appears. The Sister Joseph[r] nodule is a metastatic tumour deposit in the umbilicus, the anatomical region where the peritoneum is closest to the skin. Discoloration of the umbilicus where a faintly bluish hue is present is found rarely, in cases of extensive haemoperitoneum and acute pancreatitis (Cullen's sign[s]—the umbilical 'black eye'). Skin discoloration may also rarely occur in the flanks in severe cases of acute pancreatitis (Grey-Turner's sign[t]).

Stretching of the abdominal wall severe enough to cause rupture of the elastic fibres in the skin produces pink linear marks with a wrinkled appearance, which are called *striae*. When these are wide and purple-coloured, Cushing's syndrome may be the cause (page 309). Ascites, pregnancy or recent weight gain are much more common causes of striae.

Next, squat down beside the bed so that the patient's abdomen is at eye level. Ask him or her to take slow deep breaths through the mouth and watch for evidence of asymmetrical movement, indicating the presence of a mass. In particular a large liver may be seen to move below the right costal margin or a large spleen below the left costal margin.

Palpation

This part of the examination often reveals the most information. Successful palpation requires relaxed abdominal muscles. To this end, reassure the patient that the examination will not be painful and use warm hands. Ask the patient if any particular area is tender and examine this area last. Encourage the patient to breathe gently through the mouth. If necessary, ask the patient to bend the knees to relax the abdominal wall muscles.

For descriptive purposes the abdomen has been divided into nine areas or regions (Figure 6.23). Palpation in each region is performed with the palmar surface of the fingers acting together. For the palpation of the edges of organs or masses, the lateral surface of the forefinger is the most sensitive part of the hand.

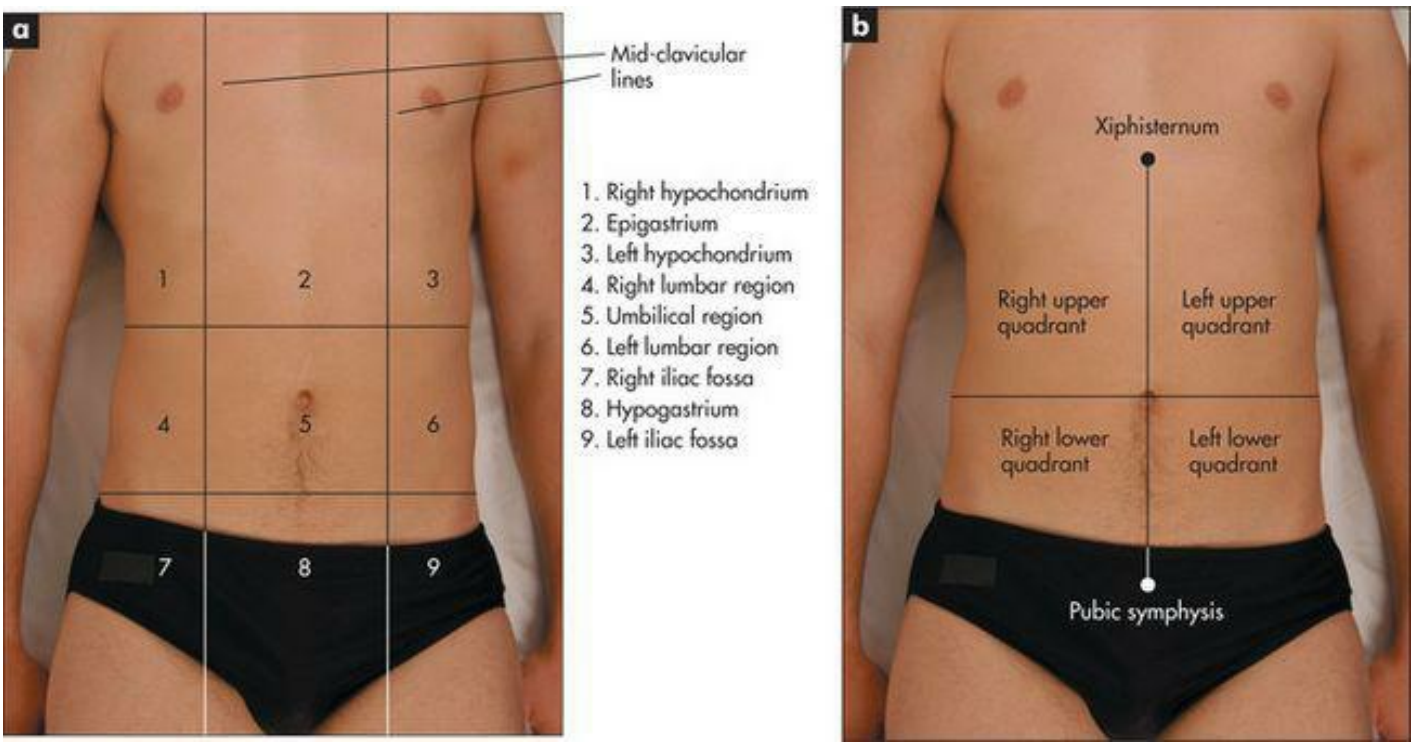


Figure 6.23 (a) Regions of the abdomen (b) Quadrants of the abdomen

Palpation should begin with *light pressure* in each region. All the movements of the hand should occur at the metacarpophalangeal joints and the hand should be moulded to the shape of the abdominal wall. Note the presence of any tenderness or lumps in each region. As the hand moves over each region, the mind should be considering the anatomical structures that underlie it. *Deep palpation* of the abdomen is performed next, though care should be taken to avoid the tender areas until the end of the examination.

Deep palpation is used to detect deeper masses and to define those already discovered. Any mass must be carefully characterised and described (Table 6.10).

TABLE 6.10 Descriptive features of intra-abdominal masses

For any abdominal mass all the following should be determined:
1 Site: the region involved
2 Tenderness
3 Size (which must be measured) and shape
4 Surface, which may be regular or irregular
5 Edge, which may be regular or irregular
6 Consistency, which may be hard or soft
7 Mobility and movement with inspiration
8 Whether it is pulsatile or not
9 Whether one can get above the mass

Guarding of the abdomen (when resistance to palpation occurs due to contraction of the abdominal muscles) may result from tenderness or anxiety, and may be voluntary or involuntary. The latter suggests peritonitis. *Rigidity* is a constant involuntary contraction of the abdominal muscles always associated with tenderness and indicates peritoneal irritation. *Rebound tenderness* is said to be present when the abdominal wall, having been compressed slowly, is released rapidly and a sudden stab of pain results. This may make the patient wince, so the face should be watched while this manoeuvre is performed. It strongly suggests the presence of peritonitis and should be performed if there is doubt about the presence of localised or generalised peritonitis. The patient with a confirmed acute abdomen should

generalised peritonitis. The patient with a confirmed acute abdomen should not be subjected to repeated testing of rebound tenderness because of the distress this can cause. Be careful not to surprise your patient by a sudden jabbing and release movement: rebound tenderness should be elicited slowly. If you suspect the patient may be feigning a tender abdomen, test for rebound with your stethoscope after telling the patient to lie still and quiet so that you can hear.

The liver

Feel for hepatomegaly (Figure 6.24).[10] With the examining hand aligned parallel to the right costal margin, and beginning in the right iliac fossa, ask the patient to breathe in and out slowly through the mouth. With each expiration the hand is advanced by 1 or 2 cm closer to the right costal margin. During inspiration the hand is kept still and the lateral margin of the forefinger waits expectantly for the liver edge to strike it.

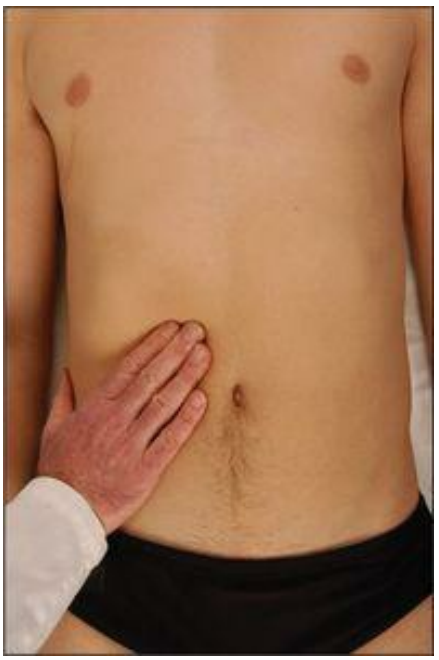


Figure 6.24 Abdominal examination: the liver

If the liver edge has been identified, an attempt should be made to feel the surface of the liver. The edge of the liver and the surface itself may be hard or soft, tender or non-tender, regular or irregular, and pulsatile or non-pulsatile. The normal liver edge may be just palpable below the right costal margin on deep inspiration, especially in thin people. The edge is then felt to be soft and regular with a fairly sharply defined border and the surface of the liver itself is smooth. Sometimes only the left lobe of the liver may be

palpable (to the left of the midline) in patients with cirrhosis.

If the liver edge is palpable the total *liver span* can be measured. Remember that the liver span varies with height and is greater in men than women, and that inter-observer error is quite large for this measurement. The normal upper border of the liver is level with the sixth rib in the midclavicular line. At this point the percussion note over the chest changes from resonant to dull (Figure 6.25a). To estimate the liver span (Figure 6.25b), percuss down along the right midclavicular line until the liver dullness is encountered and measure from here to the palpable liver edge. Careful assessment of the position of the midclavicular line will improve the accuracy of this measurement. The normal span is less than 13 cm. Note that the clinical estimate of the liver span usually underestimates its actual size by 2 to 5 cm.

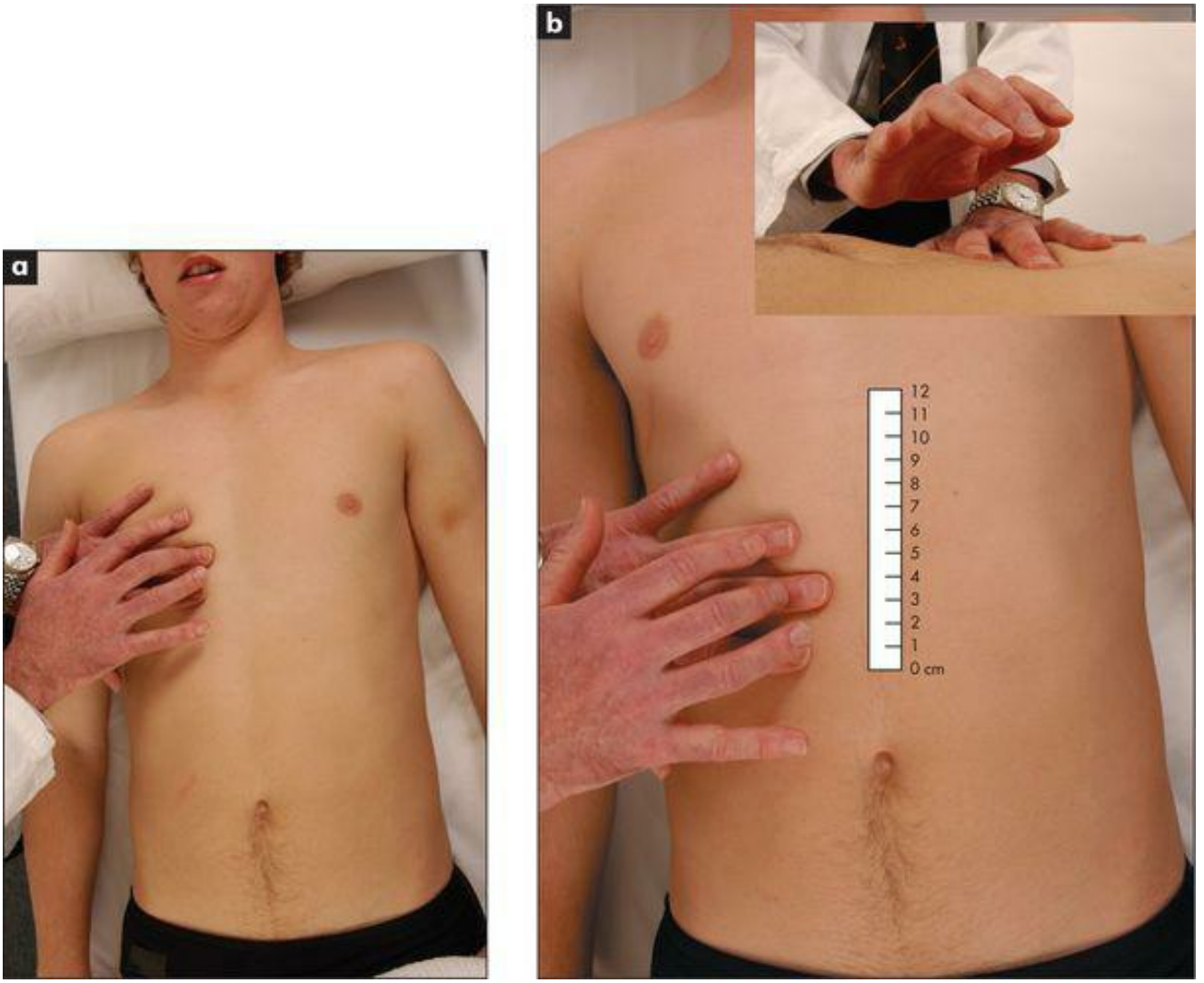


Figure 6.25 Percussing the liver span: (a) upper border; (b) lower border

Other causes of a normal but palpable liver include ptosis due to emphysema, asthma or a subdiaphragmatic collection, or a Riedel's lobe.[u] The Riedel's lobe is a tongue-like projection of the liver from the right lobe's

The Riedel's lobe is a tongue-like projection of the liver from the right lobe's inferior surface; it can be quite large and rarely extends as far as the right iliac fossa. It can be confused with an enlarged gallbladder or right kidney.

Many diseases cause hepatic enlargement and these are listed in Table 6.11. Detecting the liver edge below the costal margin clinically is highly specific (100%) but insensitive (48%)—positive LR 2.5, negative LR 0.5.[10,11] Remember, the diseased liver is not always enlarged; a small liver is common in advanced cirrhosis, and the liver shrinks rapidly with acute hepatic necrosis (due to liver cell death and collapse of the reticulin framework).

TABLE 6.11 Differential diagnosis in liver palpation

Hepatomegaly

1. Massive
    - Metastases
    - Alcoholic liver disease with fatty infiltration
    - Myeloproliferative disease
    - Right heart failure
    - Hepatocellular cancer

2. Moderate
    - The above causes
    - Haemochromatosis
    - Haematological disease—e.g. chronic leukaemia, lymphoma
    - Fatty liver—secondary to e.g. diabetes mellitus, obesity, toxins
    - Infiltration—e.g. amyloid

3. Mild
    - The above causes
    - Hepatitis
    - Biliary obstruction
    - Hydatid disease

• Human immunodeficiency virus (HIV) infection
Firm and irregular liver Hepatocellular carcinoma Metastatic disease Cirrhosis Hydatid disease, granuloma (e.g. sarcoid), amyloid, cysts, lipoidoses
Tender liver Hepatitis Rapid liver enlargement—e.g. right heart failure, Budd-Chiari* syndrome (hepatic vein thrombosis) Hepatocellular cancer Hepatic abscess Biliary obstruction/cholangitis
Pulsatile liver Tricuspid regurgitation Hepatocellular cancer Vascular abnormalities

* George Budd (1808–1882), professor of medicine, King's College Hospital, London, described this in 1845. Hans Chiari (1851–1916), professor of pathology, Prague, described it in 1898.

The gallbladder

The gallbladder is occasionally palpable below the right costal margin where

this crosses the lateral border of the rectus muscles. If biliary obstruction or acute cholecystitis is suspected, the examining hand should be oriented perpendicular to the costal margin, feeling from medial to lateral. Unlike the liver edge, the gallbladder, if palpable, will be a bulbous, focal, rounded mass which moves downwards on inspiration. The causes of an enlarged gallbladder are listed in Table 6.12.

TABLE 6.12 Gallbladder enlargement

With jaundice
1. Carcinoma of the head of the pancreas
2. Carcinoma of the ampulla of Vater*
3. In-situ gallstone formation in the common bile duct
4. Mucocele of the gallbladder due to a stone in Hartmann's† pouch and a stone in the common bile duct (very rare)
Without jaundice
1. Mucocele or empyema of the gallbladder.
2. Carcinoma of the gallbladder (stone hard, irregular swelling)
3. Acute cholecystitis

* Abraham Vater (1684–1751), Wittenberg anatomist and botanist.

† Henri Hartmann (1860–1952), professor of surgery, Paris.

Murphy's sign[v] should be sought if cholecystitis is suspected (*Good signs guide 6.1*). On taking a deep breath, the patient catches his or her breath when an inflamed gallbladder presses on the examiner's hand, which is lying at the costal margin. Other signs are less helpful.

GOOD SIGNS GUIDE 6.1 Cholecystitis

Sign	Positive LR	Negative LR
Murphy's sign	2.0	NS
Back tenderness	0.4	2.0
Right upper quadrant mass	NS	NS

NS = not significant.

From McGee S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.

The clinician examining for an enlarged gallbladder must always be mindful of *Courvoisier's law*,[w] which states that, if the gallbladder is enlarged and the patient is jaundiced, the cause is unlikely to be gallstones. Rather, carcinoma of the pancreas or lower biliary tree resulting in obstructive jaundice is likely to be present. This is because the gallbladder with stones is usually chronically fibrosed and therefore incapable of enlargement. Note that if the gallbladder is not palpable, and the patient is jaundiced, some cause other than gallstones is still possible, as at least 50% of dilated gallbladders are impalpable (*Good signs guide 6.2*).

GOOD SIGNS GUIDE 6.2 Gallbladder

Sign	Positive LR	Negative LR
Detecting obstructed bile duct in jaundiced patient		
Palpable gallbladder	26.0	0.7
Malignant obstruction in patient with obstructive jaundice	2.6	0.7

From McGee S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.

The spleen

The spleen enlarges inferiorly and medially (Figure 6.26). Its edge should be sought below the umbilicus in the midline initially. A two-handed technique is recommended. The left hand is placed posterolaterally over the left lower ribs and the right hand is placed on the abdomen below the umbilicus, parallel to the left costal margin (Figure 6.27a). Don't start palpation too near the costal margin or a large spleen will be missed. As the right hand is advanced closer to the left costal margin, the left hand compresses firmly over the rib cage so as to produce a loose fold of skin (Figure 6.27b); this removes tension from the abdominal wall and enables a slightly enlarged soft spleen to be felt as it moves down towards the right iliac fossa at the end of inspiration (Figure 6.27c).