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3

General aspects of examination

General principles of physical examination

The process of taking a history and conducting a physical examination is artificially separated in classical medical teaching, to encourage learners to develop a structured approach to information gathering. However, your physical assessment of patients undoubtedly begins as soon as you see them, and the astute clinician may notice signs of disease, such as subtle abnormalities of demeanour, gait or appearance, even before the formal consultation begins. The clinician can be likened to a detective, gathering clues, and the physical assessment of a patient can then be seen as the investigation itself!

Historically, great importance has been placed on the value of empirical observation of patients in the formulation of a differential diagnosis. Modern technological advances have increased the reliance on radiological and laboratory testing for diagnosis, sometimes even at the bedside (such as portable ultrasound or near-patient capillary blood ketone testing), and this has called into question the utility of systematic physical examination in modern practice. Nevertheless, the importance of performing a methodical and accurate physical examination cannot be overstated. The inconstancy of physical signs and the need to monitor patient progress by repeated bedside assessment, often conducted by different clinicians, mean that a standardised approach to physical examination resulting in reproducible findings is crucial. Additionally, the interpretation of many diagnostic investigations (such as detection of interstitial oedema on a chest X-ray in heart failure) is subject to variation between clinicians, as is the detection of physical signs (such as audible crackles on auscultating the lungs). Furthermore, the utility of many diagnostic investigations relies heavily on the pre-test probability (the likelihood of the disease being present prior to the test being performed; p. 362), which depends on information gathered during the history and examination. Finally, there are a number of conditions, or syndromes, that can be diagnosed only by the detection of a characteristic pattern of physical signs. Thus by mastering structured skills in physical examination, clinicians can improve the reliability and precision of their clinical assessment, which, together with the appropriate diagnostic investigations, lead to accurate diagnosis.

3.1 Information gleaned from a handshake

Features	Diagnosis
Cold, sweaty hands	Anxiety
Cold, dry hands	Raynaud's phenomenon
Hot, sweaty hands	Hyperthyroidism
Large, fleshy, sweaty hands	Acromegaly
Dry, coarse skin	Regular water exposure Manual occupation Hypothyroidism
Delayed relaxation of grip	Myotonic dystrophy
Deformed hands/fingers	Trauma Rheumatoid arthritis Dupuytren's contracture

3.2 Equipment required for a full examination

- Stethoscope
- Pen torch
- Measuring tape
- Ophthalmoscope
- Otoscope
- Sphygmomanometer
- Tendon hammer
- Tuning fork
- Cotton wool
- Disposable Neurotips
- Wooden spatula
- Thermometer
- Magnifying glass
- Accurate weighing scales and a height-measuring device (preferably a calibrated, wall-mounted stadiometer)
- Personal protective equipment (disposable gloves and apron)
- Facilities for obtaining blood samples and urinalysis

Preparing for physical examination

It is important to prepare both yourself and your patient for the physical examination. As a clinician, you must take reasonable steps to ensure you can give the patient your undivided attention, in an environment free from interruption, noise or distraction. Always introduce yourself to the patient, shake hands (which may provide diagnostic clues; Box 3.1 and see later) and seek permission to conduct the consultation. Make sure you have the relevant equipment available (Box 3.2) and that you have observed local hand hygiene policies (Fig. 3.1). As discussed on page 4, privacy is essential when assessing a patient. At the very least, ensure screens or curtains are fully closed around a ward bed; where possible, use a separate private room to avoid being overheard. Seek permission from the patient to proceed to examination, and offer a chaperone where appropriate to prevent misunderstandings and to provide support and encouragement for the patient. Regardless of whether the patient is the same gender as the doctor or not, chaperones are always appropriate for intimate (breast, genital or rectal) examination. Chaperones are also advised if the patient is especially anxious or vulnerable, if there have been misunderstandings in the past, or if religious or cultural factors require a different approach to physical examination. Record the chaperone's name and presence. If patients decline the offer, respect their wishes and record this in the notes. Tactfully invite relatives to leave the room before physical examination unless the patient is very apprehensive and requests that they stay. A parent or guardian should always be present when you examine children (p. 307).

The room should be warm and well lit; subtle abnormalities of complexion, such as mild jaundice, are easier to detect in natural light. The height of the examination couch or bed should be adjustable, with a step to enable patients to get up easily. An adjustable backrest is essential, particularly for breathless patients who cannot lie flat. It is usual practice to examine a recumbent patient from the right-hand side of the bed. Ensure the patient is comfortably positioned before commencing the physical examination.

Seek permission and sensitively, but adequately, expose the areas to be examined; cover the rest of the patient with a blanket or sheet to ensure that they do not become cold. Avoid unnecessary exposure and embarrassment; a patient may appreciate the opportunity to replace their top after examination of the chest before exposing the abdomen. Remain gentle towards the patient at all times, and be vigilant for aspects of the examination that may cause distress or discomfort. Acknowledge any anxiety or concerns raised by the patient during the consultation.

3.3 Facial expression as a guide to diagnosis

Features	Diagnosis
Poverty of expression	Parkinsonism
Startled expression	Hyperthyroidism
Apathy, with poverty of expression and poor eye contact	Depression
Apathy, with pale and puffy skin	Hypothyroidism
Agitated expression	Anxiety, hyperthyroidism, hypomania

cerebrovascular disease or Parkinsonism (see Fig. 7.17D). Notice any abnormal movements such as tremor (in alcohol withdrawal, for example), dystonia (perhaps as a side effect of neuroleptic therapy) or chorea (jerky, involuntary movements, characteristic of Huntington's disease). Abnormalities of posture and movement can also be a clue to the patient's overall wellbeing, and may represent pain, weakness or psychological or emotional disturbance.

Facial expression and speech

As with gait and posture, a patient's facial expression and how they interact with you can provide clues to their physical and psychological wellbeing (Box 3.3). Reluctance to engage in the consultation may indicate underlying depression, anxiety, fear, anger or grief, and it is important to recognise these emotions to ensure that both the physical and the emotional needs of the patient are addressed effectively. Some people conceal anxieties and depression with inappropriate cheerfulness. Illness itself may alter demeanour: frontal lobe disease or bipolar disorders may lead to animated disinhibition, whereas poverty of expression may occur in depression or Parkinson's disease. Physical signs in the face that are associated with specific diagnoses are covered later (see Box 3.9).

Be vigilant for abnormalities in the character of speech, such as slurring (due to alcohol, for example, or dysarthria caused by motor neurone disease; p. 125), hoarseness (which can represent recurrent laryngeal nerve damage; p. 186) or abnormality of speech cadence (which could be caused by pressure of speech in hyperthyroidism or slowing of speech in myxoedema; p. 197).

Hands

Starting your physical contact with a patient with a handshake not only is polite but also may reveal relevant signs (see Box 3.1). The rare disease myotonic dystrophy (which is over-represented in candidate assessments) causes a patient to fail to release the handgrip (due to delayed muscle relaxation). A patient with neurological disease may be unable to shake your hand, or may have signs of muscle wasting or tremor. Detailed examination of the hands is described on page 265 but even a brief inspection and palpation may be very revealing.

Deformity

Deformity may indicate nerve palsies or arthritic changes (such as ulnar deviation at the metacarpophalangeal joints in longstanding rheumatoid arthritis; see Fig. 13.22). Arthritis frequently involves the small joints of the hands. Rheumatoid arthritis typically affects metacarpophalangeal and proximal interphalangeal joints (see Fig. 13.22), and osteoarthritis and psoriatic arthropathy affect the distal interphalangeal joints (see Fig. 13.8). Small-muscle wasting of the hands is common in rheumatoid arthritis, producing 'dorsal guttering' of the hands, and also occurs in cervical spondylosis with nerve root entrapment. In carpal tunnel syndrome, median nerve compression leads to wasting of the thenar muscles, also seen in damage affecting the T1 nerve root (see Fig. 13.23).

Dupuytren's contracture is a thickening of the palmar fascia causing fixed flexion deformity, and usually affects the little and ring fingers (Fig. 3.5). Arachnodactyly (long, thin fingers) is typical of Marfan's syndrome (see Fig. 3.21B). Trauma is the most common cause of hand deformity.

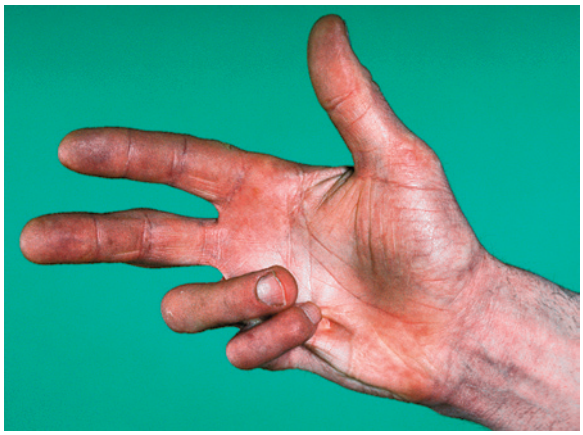


Fig. 3.5 Dupuytren's contracture.

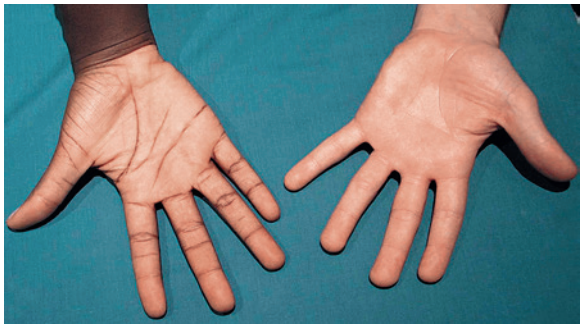


Fig. 3.6 Normal palms. African (left) and European (right).

Colour

Colour changes in the hands may also be revealing. Look for peripheral cyanosis in the nail bed and tobacco staining of the fingers (see Fig. 5.8). Examine the skin creases for pigmentation, although pigmentation is normal in many non-Caucasian races (Fig. 3.6).

Temperature

The temperature of the patient's hand is a good guide to peripheral perfusion. In chronic obstructive pulmonary disease the hands may be cyanosed due to reduced arterial oxygen saturation but warm due to vasodilatation from elevated arterial carbon dioxide levels. In heart failure the hands are often cold and cyanosed because of vasoconstriction in response to a low cardiac output. If they are warm, heart failure may be due to a high-output state, such as hyperthyroidism.

3.4 The nails in systemic disease

Nail changes	Description of nail	Differential diagnosis
Beau's lines	Transverse grooves (see Fig. 3.7B)	Sequella of any severe systemic illness that affects growth of the nail matrix
Clubbing	Loss of angle between nail fold and nail plate (see Fig. 3.8)	Serious cardiac, respiratory or gastrointestinal disease (see Box 3.5)
Leuconychia	White spots, ridges or complete discoloration of nail (see Fig. 3.7C)	Trauma, infection, poisoning, chemotherapy, vitamin deficiency
Lindsay's nails	White/brown 'half-and-half' nails (see Fig. 12.7)	Chronic kidney disease
Koilonychia	Spoon-shaped depression of nail plate (see Fig. 3.7D)	Iron deficiency anaemia, lichen planus, repeated exposure to detergents
Muehrcke's lines	Narrow, white transverse lines (see Fig. 12.6)	Decreased protein synthesis or protein loss
Nail-fold telangiectasia	Dilated capillaries and erythema at nail fold (see Fig. 14.13B)	Connective tissue disorders, including systemic sclerosis, systemic lupus erythematosus, dermatomyositis
Onycholysis	Nail separates from nail bed (see Fig. 3.7A)	Psoriasis, fungal infection, trauma, thyrotoxicosis, tetracyclines (photo-onycholysis)
Onychomycosis	Thickening of nail plate with white, yellow or brown discoloration	Fungal infection
Pitting	Fine or coarse pits in nail (see Fig. 3.7A)	Psoriasis (onycholysis, thickening and ridging may also be present), eczema, alopecia areata, lichen planus
Splinter haemorrhages	Small red streaks that lie longitudinally in nail plate (see Fig. 4.5B)	Trauma, infective endocarditis
Yellow nails	Yellow discoloration and thickening (see Fig. 14.13C)	Yellow nail syndrome

Skin

Skin changes in the hands can indicate systemic disease, as in the coarse skin and broad hands of a patient with acromegaly (see Fig. 10.8), or the tight, contracted skin (scleroderma) and calcium deposits associated with systemic sclerosis (see Figs 3.30C and 13.6). Clues about lifestyle can also be seen in the hands: manual workers may have specific callosities due to pressure at characteristic sites, while disuse results in soft, smooth skin.

Nails

Nail changes occur in a wide variety of systemic diseases. Box 3.4 and Fig. 3.7 summarise nail changes seen on general examination that may indicate underlying systemic disease.

Finger clubbing describes painless soft tissue swelling of the terminal phalanges and increased convexity of the nail (Fig. 3.8). Clubbing usually affects the fingers symmetrically. It may also involve the toes and can be unilateral if caused by a proximal vascular condition, such as arteriovenous shunts for dialysis. It is sometimes congenital but in over 90% of patients it heralds a serious underlying disorder (Box 3.5). Clubbing may recede if the underlying condition resolves.

3.5 Causes of clubbing

Congenital or familial (5–10%)

Acquired

- Thoracic (~70%):
  - Lung cancer
  - Chronic suppurative conditions: pulmonary tuberculosis, bronchiectasis, lung abscess, empyema, cystic fibrosis
  - Mesothelioma
  - Fibroma
  - Pulmonary fibrosis
- Cardiovascular:
  - Cyanotic congenital heart disease
  - Infective endocarditis
  - Arteriovenous shunts and aneurysms
- Gastrointestinal:
  - Cirrhosis
  - Inflammatory bowel disease
  - Coeliac disease
- Others:
  - Thyrotoxicosis (thyroid acropachy)
  - Primary hypertrophic osteoarthropathy

Examination sequence

- Look across the nail bed from the side of each finger. Observe the distal phalanges, nail and nail bed:
  - Estimate the interphalangeal depth at the level of the distal interphalangeal joint (this is the anteroposterior thickness of the digit rather than the width). Repeat at the level of the nail bed.
  - Assess the nail-bed (hyponychial) angle (Fig. 3.9A).
- Ask the patient to place the nails of corresponding (ring) fingers back to back and look for the normal 'diamond-shaped' gap between the nail beds (Schamroth's window sign; Fig. 3.9B).
- Place your thumbs under the pulp of the distal phalanx and use your index fingers alternately to see if there is fluctuant movement of the nail on the nail bed (Fig. 3.9C).

Finger clubbing is likely if:

- the interphalangeal depth ratio is >1 (that is, the digit is thicker at the level of the nail bed than the level of the distal interphalangeal joint; Fig. 3.9A)
- the nail fold angle is >190 degrees (Fig. 3.9A)
- Schamroth's window sign is absent (Fig. 3.9B).

Increased nail-bed fluctuation may be present and may support the finding of clubbing, but its presence is subjective and less discriminatory than the above features.

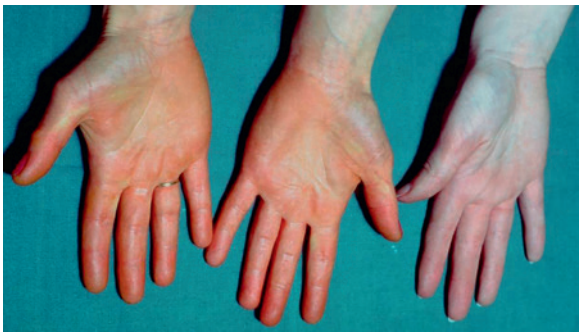


Fig. 3.13 Hypercarotenaemia. A control normal hand is shown on the right for comparison.

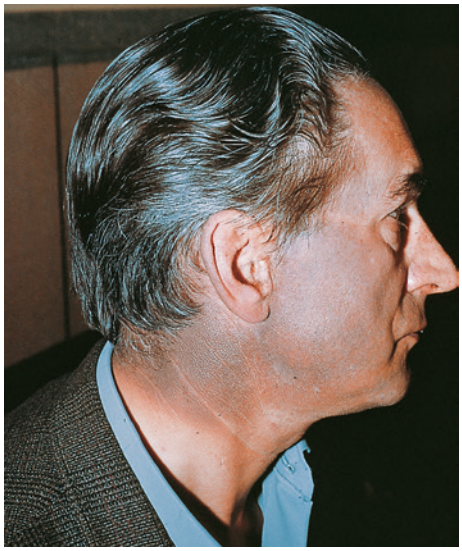


Fig. 3.14 Phenothiazine-induced pigmentation.

strikingly abnormal coloration of the skin, particularly in areas exposed to light: for example, mepacrine (yellow), amiodarone (bluish-grey) and phenothiazines (slate-grey; Fig. 3.14).

Jaundice

Jaundice is an abnormal yellow discoloration of the skin, sclera and mucous membranes. It is usually detectable when serum bilirubin concentration rises above 50 μmol/L (3 mg/dL) as a result of parenchymal liver disease, biliary obstruction or haemolysis (see Fig. 6.8).

Pallor

Pallor can result from anaemia, in which there is a reduction in circulating oxyhaemoglobin in the dermal and subconjunctival capillaries, or from vasoconstriction due to cold exposure or sympathetic activation. The best sites to assess for the pallor of anaemia are the conjunctiva (specifically the anterior rim; Fig. 3.15), the palmar skin creases and the face in general, although absence of pallor does not exclude anaemia. Nail-bed pallor lacks diagnostic value for predicting anaemia but is still often assessed by clinicians. In significant iron deficiency anaemia there may be additional findings of angular stomatitis, glossitis (Fig. 3.16), koilonychia (spoon-shaped nails) and blue sclerae.

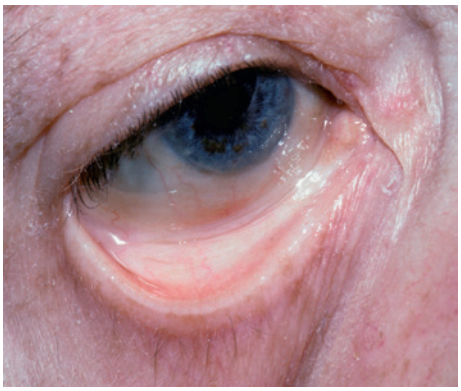


Fig. 3.15 Conjunctival pallor.

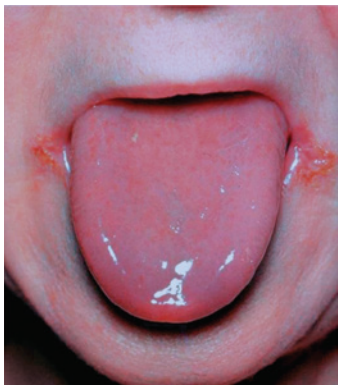


Fig. 3.16 Smooth red tongue (glossitis) and angular stomatitis of iron deficiency.

3.6 Conditions associated with facial flushing

Physiological
- Fever
- Exercise
- Heat exposure
- Emotional

Drugs (e.g. glyceryl trinitrate, calcium channel blockers, nicotinic acid)

Anaphylaxis

Endocrine
- Menopause
- Androgen deficiency (in men)
- Carcinoid syndrome
- Medullary thyroid cancer

Others
- Serotonin syndrome
- Food/alcohol ingestion
- Neurological (e.g. Frey's syndrome)
- Rosacea
- Mastocytoses

Conversely, vasodilatation, or flushing, may produce a pink complexion, even in anaemia, and may be due to fever, heat, exercise, food, drugs and other neurological or hormonal disturbances (Fig. 3.17 and Box 3.6). Facial plethora is caused by raised haemoglobin concentration with elevated haematocrit (polycythaemia); it may be primary or may indicate an underlying

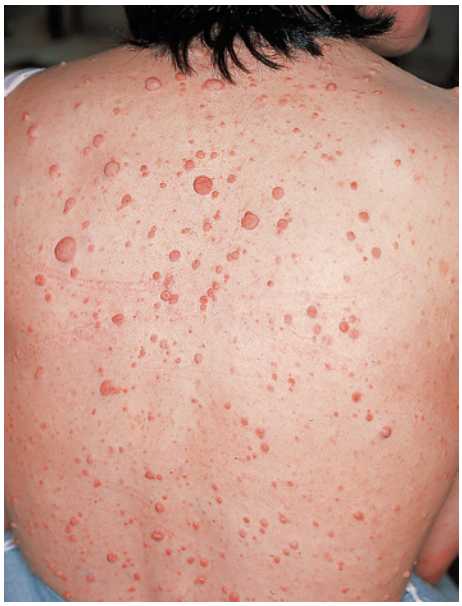


Fig. 3.20 Neurofibromatosis.

Tongue

In addition to revealing central cyanosis, examination may uncover the smooth tongue of iron deficiency (see Fig. 3.16), enlargement in acromegaly, or wasting and fasciculation in motor neurone disease.

Odours

Odours can provide clues to a patient's social or behavioural habits; the smell of alcohol, tobacco or cannabis may be readily apparent. Stale urine and anaerobic skin infections also produce distinctive smells. Halitosis (bad breath) can be due to poor dental hygiene, gingivitis, stomatitis, atrophic rhinitis, tumours of the nasal passages or suppurative lung conditions such as lung abscess or bronchiectasis.

Other characteristic odours include:

- ketones: a sweet smell (like nail varnish remover) due to acetone in diabetic ketoacidosis or starvation
- fetor hepaticus: the stale, 'mousy' smell of the volatile amine dimethylsulphide in patients with liver failure
- uraemic fetor: a fishy or ammoniacal smell on the breath in uraemia
- foul-smelling belching in patients with gastric outlet obstruction
- a faecal smell in patients with gastrocolic fistula.

Body habitus and nutrition

Weight

Weight is an important indicator of general health and nutrition, and serial weight measurements can be useful in monitoring both acute and chronic disease. The body mass index (BMI; calculated from the formula weight(kg)/height(m)^2) is more useful than weight alone, as it allows for differing height. Normal values for different ethnicities are available (Box 3.7).

3.7 The relationship between body mass index (BMI), nutritional status and ethnic group

Nutritional status	BMI non-Asian	BMI Asian
Underweight	<18.5	<18.5
Normal	18.5–24.9	18.5–22.9
Overweight	25–29.9	23–24.9
Obese	30–39.9	25–29.9
Morbidly obese	≥40	≥30

Obesity

Obesity is associated with an increased risk of malignancy, particularly oesophageal and renal cancer in both sexes, thyroid and colon cancer in men, and endometrial and gallbladder cancer in women, as well as hypertension, hyperlipidaemia, type 2 diabetes mellitus, gastro-oesophageal reflux, gallbladder disease, osteoarthritis and sleep apnoea. While it is usually the result of excessive calorie intake relative to calories expended, it can rarely be secondary to hypothyroidism, Cushing's syndrome, hypothalamic disease or drugs such as oral hypoglycaemic agents, insulin and antipsychotics.

Note the distribution of fat, since central obesity (as judged by the waist circumference: the maximum abdominal girth at the midpoint between the lower costal margin and the iliac crest) correlates with increased visceral adiposity and has worse health outcomes due to its association with hypertension, insulin resistance, type 2 diabetes mellitus and coronary artery disease. Waist-to-hip ratio can also be a useful assessment of adipose distribution: gluteal–femoral obesity or the 'pear shape' (waist : hip ratio of ≤0.8 in females or <0.9 in males) has a better prognosis, whereas 'apple-shaped' patients with a greater waist : hip ratio have an increased risk of coronary artery disease and the 'metabolic syndrome'.

Weight loss

Weight loss or malnutrition (p. 94) may be due to inadequate energy consumption or utilisation (such as malabsorption, anorexia, glycosuria) or to conditions in which nutritional demand is increased (such as fever, infection, thyrotoxicosis, malignancy, surgery). Psychiatric disease and alcohol or drug dependency may also result in weight loss. Useful markers of malnutrition include arm muscle circumference and grip strength. Malnutrition may also be associated with biochemical and physical evidence of hypoproteinaemia and/or vitamin deficiencies. Malnutrition lengthens recovery time from illness and surgery, and delays wound healing.

Stature

Short stature

Short stature may reflect general nutritional state or significant illness during childhood, although it may be familial (ask about the height of the patient's parents and siblings; p. 310). Loss of height is part of normal ageing but is accentuated by compression

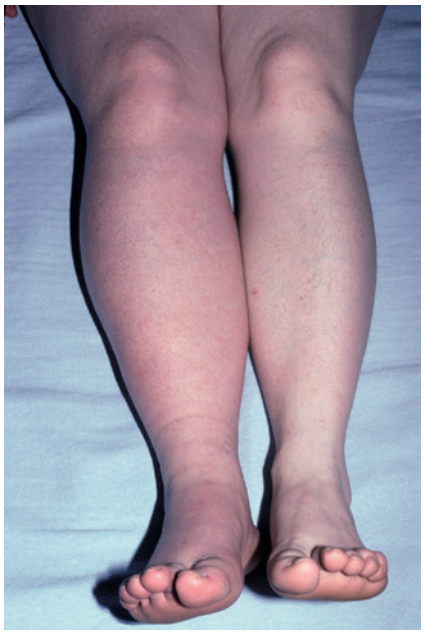


Fig. 3.22 Swollen right leg, suggesting deep vein thrombosis or inflammation. Causes include soft tissue infection or a ruptured Baker's cyst.

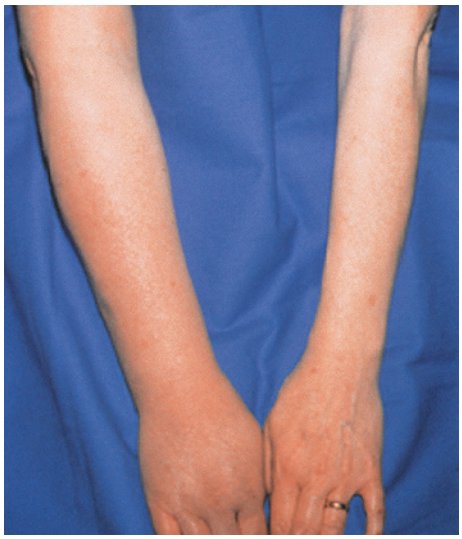


Fig. 3.23 Lymphoedema of the right arm following right-sided mastectomy and radiotherapy.

Lymphatic causes

Normally, interstitial fluid returns to the central circulation via the lymphatic system. Any obstruction to lymphatic flow may produce localised oedema (lymphoedema; Fig. 3.23). If the condition persists, fibrous tissue proliferates in the interstitial space and the affected area becomes hard and no longer pits on pressure. In the UK the most common cause of chronic leg lymphoedema is congenital hypoplasia of leg lymphatics (Milroy's disease); in the arm, lymphoedema usually follows radical mastectomy and/or irradiation for breast cancer. Lymphoedema is common in some tropical countries because of lymphatic obstruction by filarial worms (elephantiasis).

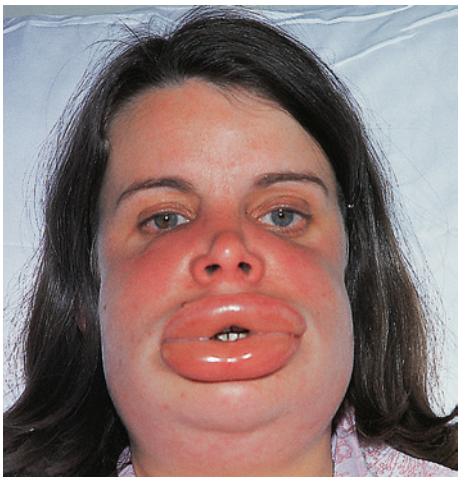


Fig. 3.24 Angio-oedema following a wasp sting.

3.8 Features to note in any lump or swelling (SPACESPIT)

- Size
- Position
- Attachments
- Consistency
- Edge
- Surface and shape
- Pulsation, thrills and bruits
- Inflammation:
  - Redness
  - Tenderness
  - Warmth
- Transillumination

Inflammatory causes

Any cause of tissue inflammation, including infection or injury, liberates mediators such as histamine, bradykinin and cytokines, which cause vasodilatation and increase capillary permeability. Inflammatory oedema is accompanied by the other features of inflammation (redness, tenderness and warmth) and is therefore painful.

Allergic causes

Increased capillary permeability occurs in acute allergic conditions: for example, an insect bite in an allergic individual. The affected area is usually red and pruritic (itchy) because of local release of histamine and other inflammatory mediators but, in contrast to inflammation, is not painful.

Angio-oedema is a severe form of allergic oedema affecting the face, lips and mouth, most commonly caused by insect bites, food allergy or drug reactions (Fig. 3.24). Swelling may develop rapidly and become life-threatening if the upper airway is involved.

Lumps and lymph nodes

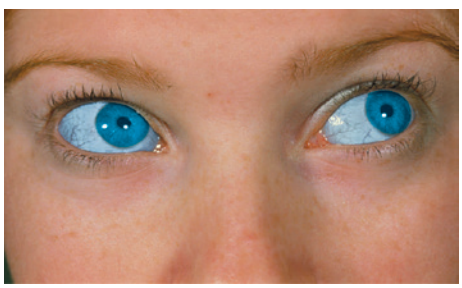
Patients often present with a lump or enlarged lymph nodes (lymphadenopathy), which, while usually benign, can herald a serious underlying infective or malignant process. Alternatively, when examining a patient you may find a lump of which they were unaware.

Lumps

Ask about the rapidity of onset of the lump and the presence of any associated pain, tenderness or colour change. Document the following features (Box 3.8):

3.9 Conditions with characteristic facial appearances

Diagnosis	Facial features
Hypothyroidism (see Fig. 10.5)	Sparse, coarse hair and eyebrows, periorbital puffiness, dry, waxy skin, apathetic expression, macroglossia
Graves' disease (autoimmune thyrotoxicosis) (see Fig. 10.2A)	Staring appearance due to lid retraction, proptosis, evidence of weight loss
Hypopituitarism (see Fig. 10.10A)	Pale, often unwrinkled skin with loss of hair
Acromegaly (see Fig. 10.9A)	Thickened, coarse skin with enlarged nose and frontal bones, prognathism (lower jaw protrusion), widely spaced teeth, macroglossia
Cushing's syndrome (see Fig. 10.11A)	Moon-shaped plethoric facies
Osteogenesis imperfecta (see Fig. 3.30A)	Blue sclerae
Hereditary haemorrhagic telangiectasia (see Fig. 3.30B)	Telangiectasia on and around lips
Systemic sclerosis (see Fig. 3.30C)	Tight skin constricting mouth, 'beaking' of nose, loss of nasolabial folds
Myotonic dystrophy (see Fig. 3.30D)	Frontal balding, paucity of expression, bilateral ptosis
Down's syndrome (see Fig. 3.31)	Flat facial profile, up-slanting palpebral fissures, small, low-set ears, macroglossia, Brushfield spots in iris
Systemic lupus erythematosus	'Butterfly' erythematous rash on cheeks



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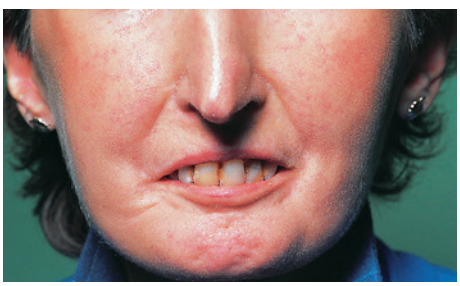

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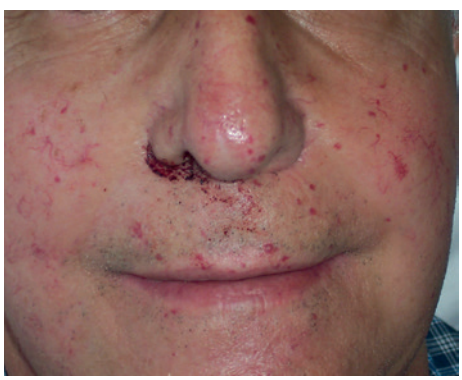

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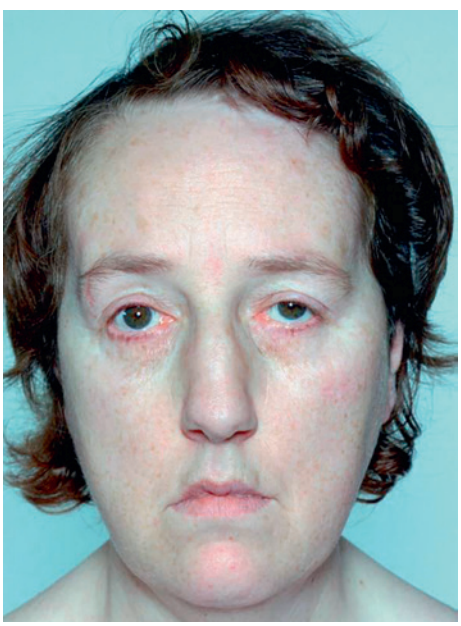

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Fig. 3.30 Characteristic facial features of some disorders. A Blue sclerae of osteogenesis imperfecta. B Telangiectasia around the mouth, typical of hereditary haemorrhagic telangiectasia. C Systemic sclerosis with 'beaking' of the nose and taut skin around the mouth. D Myotonic dystrophy with frontal balding and bilateral ptosis.

اسألكم الدعاء اخوكم علي محمد عبود

Macleod clinical picture

Respiratory system

Fig. 5.4 Common occupational and environmental causes of respiratory disease.

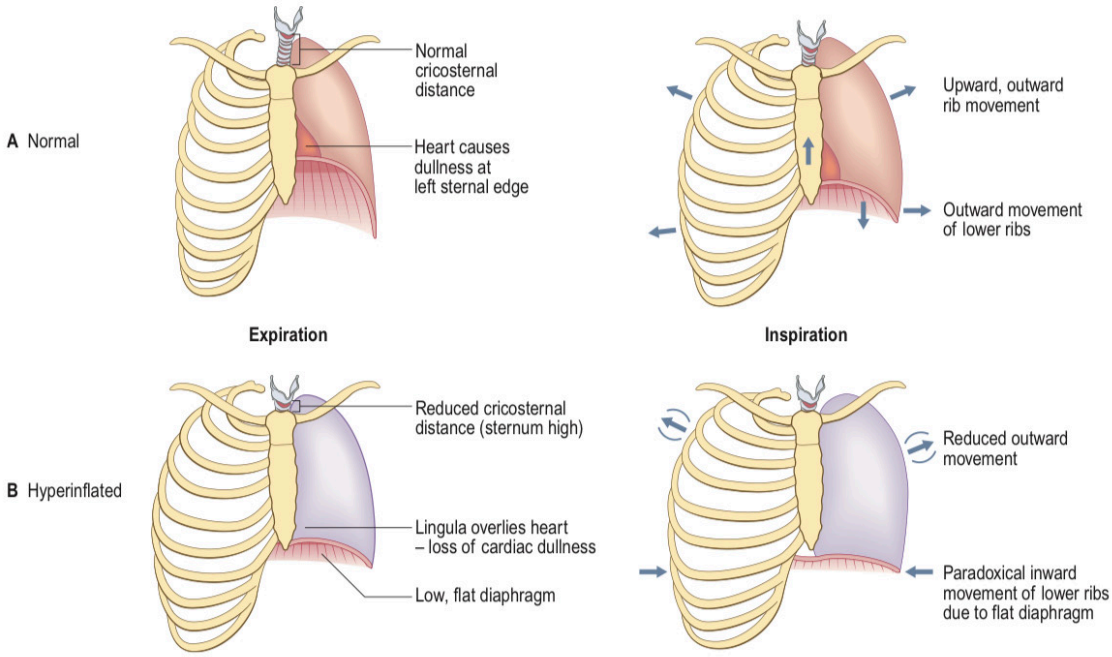


Fig. 5.5 **Respiratory movement of the ribs, sternum and diaphragm.** A In normal adults. B In chronic hyperinflation due to obstructive lung disease. Hyperinflation causes upward displacement of the sternum and clavicles, increased anteroposterior thoracic diameter, loss of cardiac dullness at the lower left sternal edge, and a low flat diaphragm that pulls the lower ribs in during inspiration.

Hyperinfated chest cause (loss of cardiac dullness,reduce cricosternal distance, Paradoxicall inword movement of lower ribs)

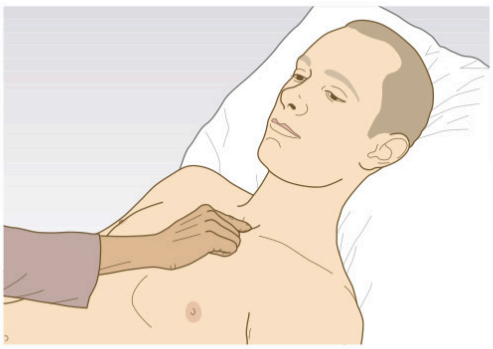


Fig. 5.13 Examining for tracheal shift.

BOX 5 – 3. Causes of tracheal deviation		
Towards the side of the lung lesion	Away from the side of the lung lesion	Upper mediastinal mass
Upper lobe or lung collapse Upper lobe or lung fibrosis Pneumonectomy	Tension pneumothorax Massive pleural effusion	Retrosternal goiter Lymphoma or lung cancer

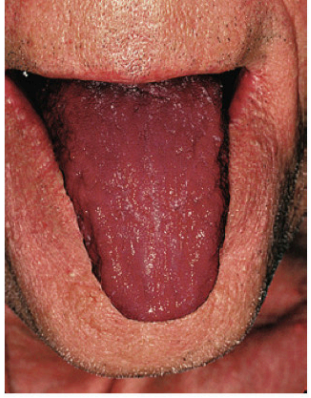


Fig. 5.12 Central cyanosis of the tongue.

BOX 2 – 7. Types and causes of cyanosis	
Central cyanosis	Peripheral cyanosis
Found in the mucous membrane and skin	Found in the skin only
Hot extremities	Cold extremities
Clubbing of finger may be present	No clubbing of finger
This type of cyanosis results from 1. Cyanotic congenital heart disease 2. Respiratory failure 3. Heart failure 4. Abnormal hemoglobin derivatives e.g. methemoglobin and sulfhemoglobin	This type of cyanosis results from 1. Exposure to cold 2. Heart failure 3. Arterial obstruction 4. Venous obstruction

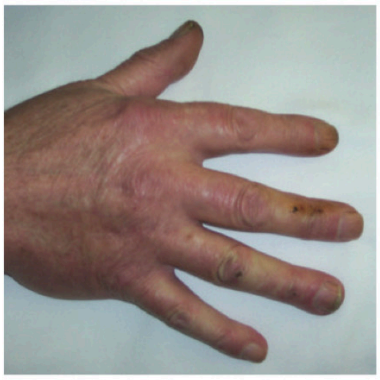


Fig. 5.8 Tobacco 'tar'-stained finger.

lung cancer , chronic bronchitis

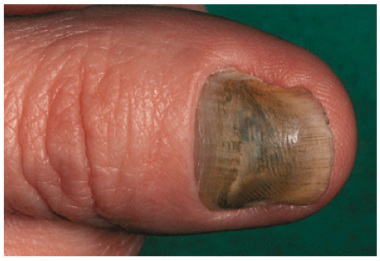


Fig. 5.9 Yellow nail syndrome.

rarely, yellow–brown discoloration of nails in yellow nail syndrome

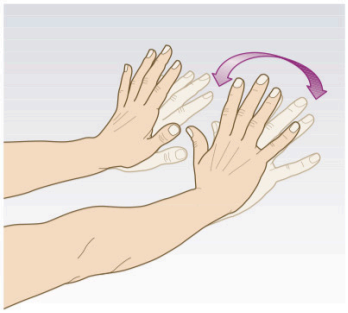
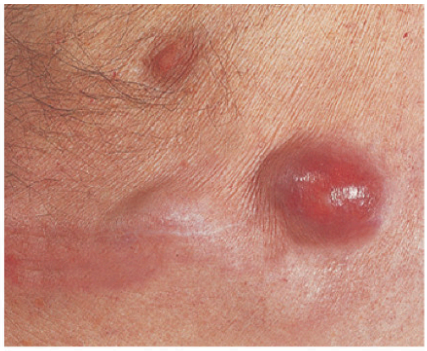
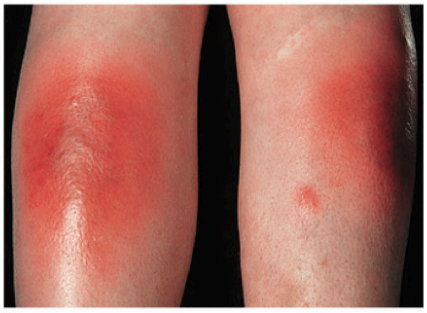


Fig. 5.10 Hand position for testing for the coarse tremor of CO_2 retention.

co2 retention



A



B

Fig. 5.7 Skin lesions associated with respiratory conditions. A Metastatic nodules of lung cancer. B Erythema nodosum on the shins in sarcoidosis.

metastatic nodules of lung cancer

Erythema nodosum in sarcoidosis

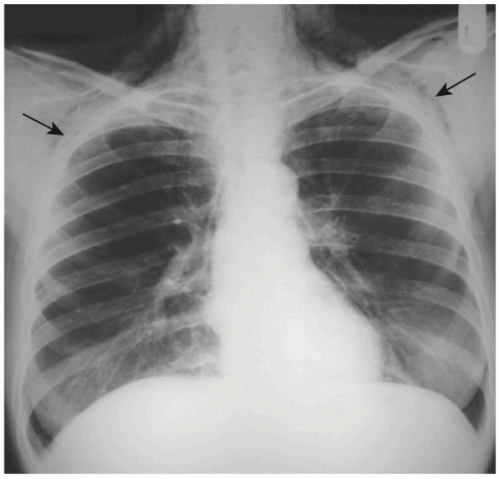
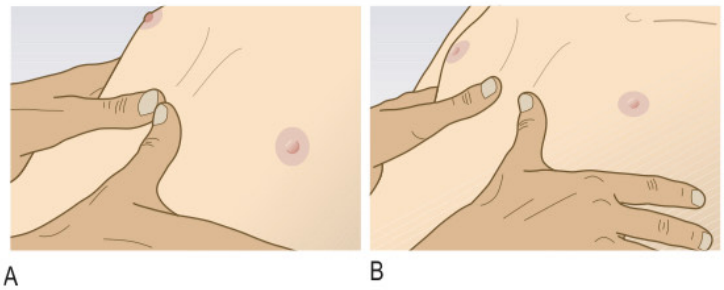


Fig. 5.15 Subcutaneous air (surgical emphysema) seen in the neck and chest wall on chest X-ray *(arrows)*.

surgical emphysema (This most commonly complicates pneumothorax with chest drainage or rib fracture, and feels like a palpable crackling under the skin of the upper thorax and neck.)

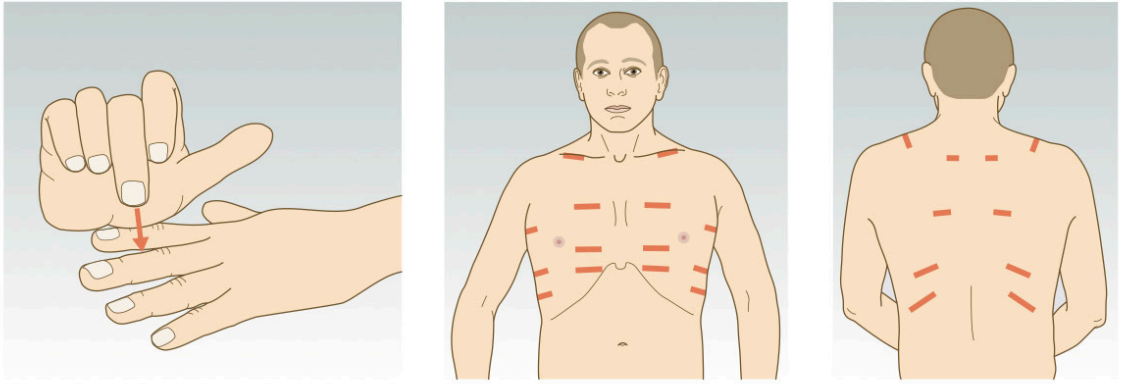


A B

Fig. 5.14 Assessing chest expansion from the front. A Expiration. B Inspiration.

chest expansion (normally from 5–8cm)

Reduce chest expansion in (fibrosis,collapse,consolidation,pleural effusion ,pneumothorax ,emphysema ,diffuse pulmonary fibrosis)



A B C

Fig. 5.16 **Percussion of the chest.** A Technique. B Anterior and lateral sites. C Posterior sites.

BOX 5 – 6. Features of percussion notes			
Resonant	Dull	Stony dull	Hyper-resonant
Normal lung	Consolidation Collapse Fibrosis ممكن ستوني اذا متركز	Pleural effusion Hemothorax Large hydatid cyst	Pneumothorax COPD

Auscultation

7.22 Causes of diminished vesicular breathing

Reduced conduction

- Obesity/thick chest wall
- Pleural effusion or thickening
- Pneumothorax

Reduced airflow

- Generalised, e.g. COPD
- Localised, e.g. collapsed lung due to occluding lung cancer

7.22 Causes of diminished vesicular breathing

Reduced conduction

- Obesity/thick chest wall
- Pleural effusion or thickening
- Pneumothorax

Reduced airflow

- Generalised, e.g. COPD
- Localised, e.g. collapsed lung due to occluding lung cancer

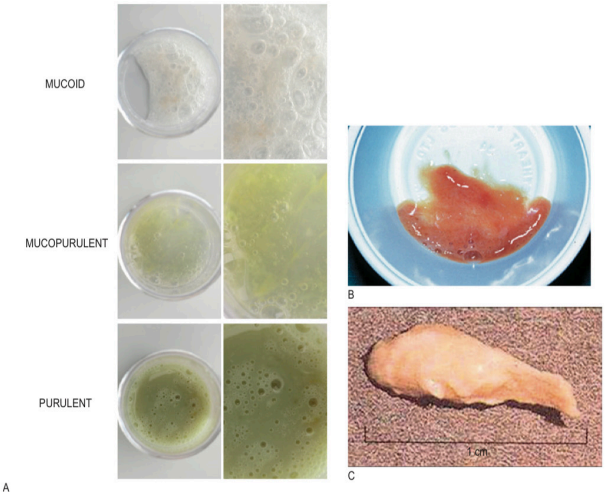
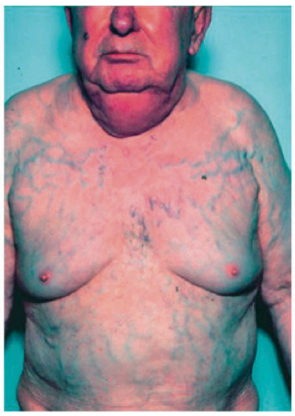


Fig. 5.3 **Sputum appearance in disease.** A Colour chart of sputum purulence used in bronchiectasis. B Rusty red sputum of pneumococcal pneumonia. C Mucus plug from a patient with asthma. *(A) Courtesy Medical Photography, NHS Lothian.*

Colour

- Clear (mucoid): COPD/bronchiectasis without current infection/rhinitis.
- Yellow (mucopurulent): acute lower respiratory tract infection/asthma.
- Green (purulent): current infection – acute disease or exacerbation of chronic disease, such as COPD.
- Red/brown (rusty): pneumococcal pneumonia (Fig. 5.3B). Try to distinguish between rusty and frank red blood (see below).
- Pink (serous/frothy): acute pulmonary oedema.



mediastinal spread of lung cancer cause SVC obstruction

Fig. 5.11 Superior vena cava obstruction. Dusky, swollen face and neck, and distended superficial collateral veins on the chest wall. *From Midthun DE, Jett JR. Clinical presentation of lung cancer. In Pass HI, Mitchel JB, Johnson DH, et al. (eds). Lung Cancer: Principles and Practice. Philadelphia: Lippincott–Raven; 1996, p. 421.*

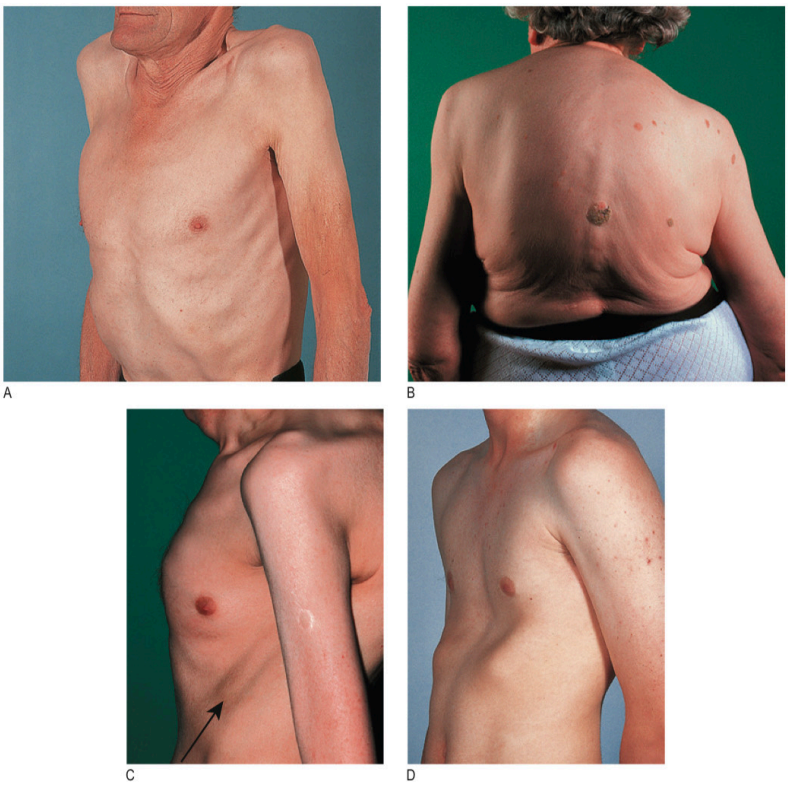


Fig. 5.6 **Abnormalities in the shape of the chest.** A Hyperinflated chest with raised sternum and shoulder girdle. B Kyphoscoliosis. C Pectus carinatum with Harrison's sulcus *(arrow)*. D Pectus excavatum.

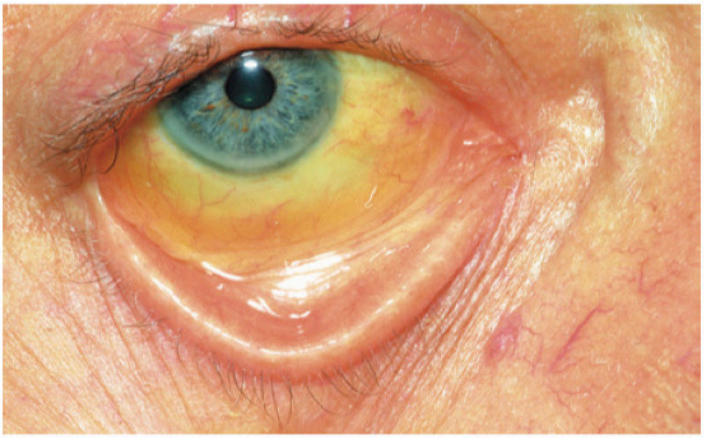


Fig. 6.8 Yellow sclera of jaundice.

6.6 Common causes of jaundice

Increased bilirubin production

- Haemolysis (unconjugated hyperbilirubinaemia)

Impaired bilirubin excretion

- Congenital:
  - Gilbert's syndrome (unconjugated)
- Hepatocellular:
  - Viral hepatitis
  - Cirrhosis
  - Drugs
  - Autoimmune hepatitis
- Intrahepatic cholestasis:
  - Drugs
  - Primary biliary cirrhosis
- Extrahepatic cholestasis:
  - Gallstones
  - Cancer: pancreas, cholangiocarcinoma

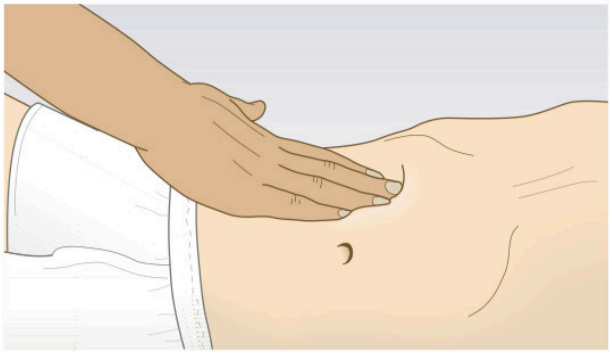


Fig. 6.14 Palpation of the liver.

6.10 Causes of hepatomegaly

Chronic parenchymal liver disease

- Alcoholic liver disease
- Hepatic steatosis
- Autoimmune hepatitis
- Viral hepatitis
- Primary biliary cirrhosis

Malignancy

- Primary hepatocellular cancer
- Secondary metastatic cancer

Right heart failure

Haematological disorders

- Lymphoma
- Leukaemia
- Myelofibrosis
- Polycythaemia

Rarities

- Amyloidosis
- Budd–Chiari syndrome
- Sarcoidosis
- Glycogen storage disorders

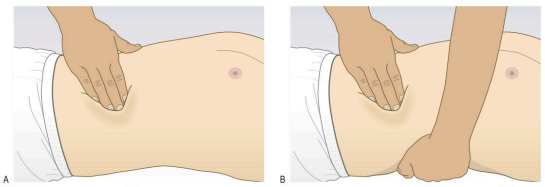


Fig. 6.16 Palpation of the spleen. A Initial palpation for the splenic edge moving diagonally from the umbilicus to the left hypochondrium. B If the spleen is impalpable by the method shown in A, use your left hand to pull the ribcage forward and elevate the spleen, making it more likely to be palpable by your right hand.

6.13 Causes of splenomegaly

Haematological disorders

- Lymphoma and lymphatic leukaemias
- Myeloproliferative diseases, polycythaemia rubra vera and myelofibrosis
- Haemolytic anaemia, congenital spherocytosis

Portal hypertension

Infections

- Glandular fever
- Malaria, kala-azar (leishmaniasis)
- Bacterial endocarditis
- Brucellosis, tuberculosis, salmonellosis

Rheumatological conditions

- Rheumatoid arthritis (Felty's syndrome)
- Systemic lupus erythematosus

Rarities

- Sarcoidosis
- Amyloidosis
- Glycogen storage disorders

8.37 Causes of hepatosplenomegaly

- Lymphoma
- Myeloproliferative diseases
- Cirrhosis with portal hypertension
- Amyloidosis, sarcoidosis, glycogen storage disease

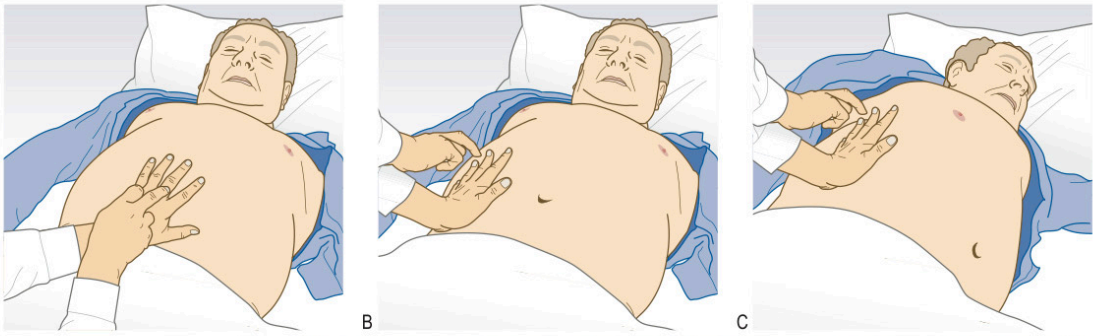


Fig. 6.17 **Percussing for ascites.** A and B Percuss towards the flank from resonant to dull. C Then ask the patient to roll on to their other side. In ascites the note then becomes resonant.

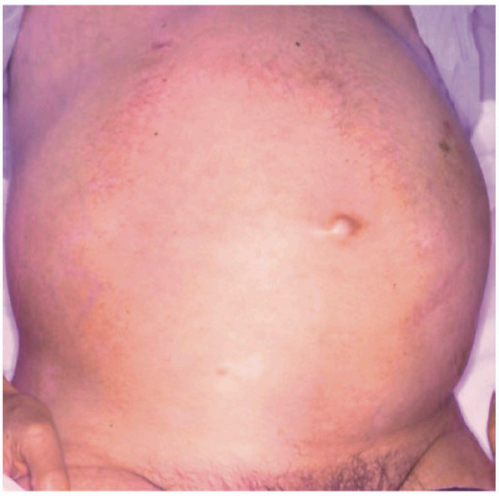


Fig. 6.6 Abdominal distension due to ascites.

6.14 Causes of ascites

Diagnosis	Comment
Common	
Hepatic cirrhosis with portal hypertension	Transudate
Intra-abdominal malignancy with peritoneal spread	Exudate, cytology may be positive
Uncommon	
Hepatic vein occlusion (Budd–Chiari syndrome)	Transudate in acute phase
Constrictive pericarditis and right heart failure	Check jugular venous pressure and listen for pericardial rub
Hypoproteinaemia (nephrotic syndrome, protein-losing enteropathy)	Transudate
Tuberculous peritonitis	Low glucose content
Pancreatitis, pancreatic duct disruption	Very high amylase content

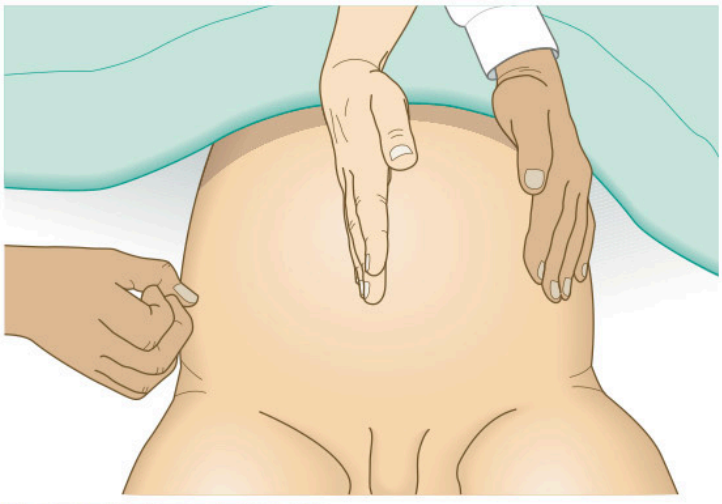
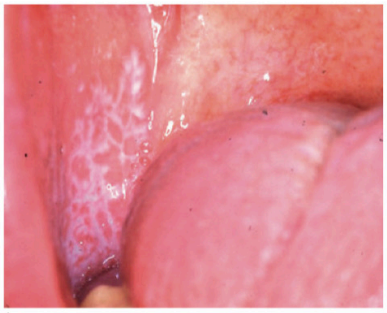
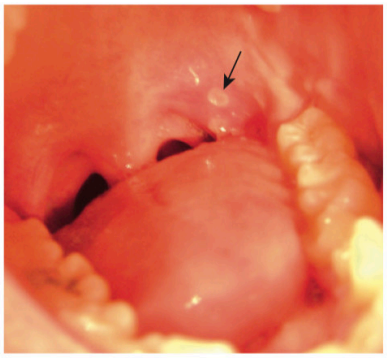


Fig. 6.18 Eliciting a fluid thrill.

detected only in gross ascites



A



B

Fig. 6.3 Some causes of a painful mouth. A Lichen planus. B Small, 'punched-out' aphthous ulcer *(arrow)*.

Lichen planus (dermatological disorders)

Aphthous ulcer (coeliac and inflammatory bowel disease)

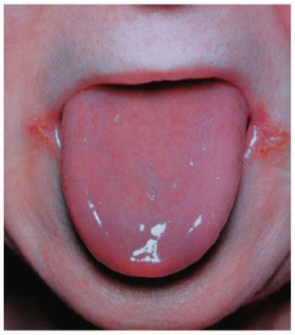
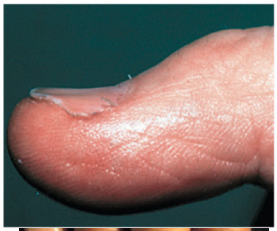


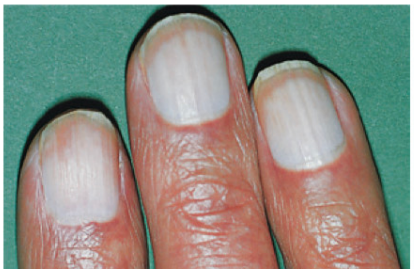
Fig. 3.16 Smooth red tongue (glossitis) and angular stomatitis of iron deficiency.

angular stomatitis and atrophic glossitis (iron deficiency anemia)



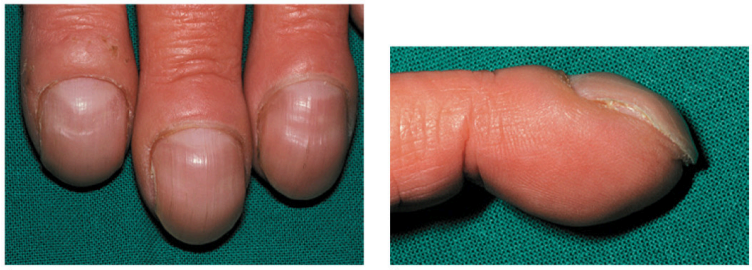
D

koilonychia (iron deficiency anemia)



3

leuconychia hypoalbuminaemia(chronic liver disease,nephrotic syndrome,kawskiorkor,celiac disease)



A B

Fig. 3.8 Clubbing. A Anterior view. B Lateral view.

3.5 Causes of clubbing

Congenital or familial (5–10%)

Acquired

- Thoracic (~70%):
  - Lung cancer
  - Chronic suppurative conditions: pulmonary tuberculosis, bronchiectasis, lung abscess, empyema, cystic fibrosis
  - Mesothelioma
  - Fibroma
  - Pulmonary fibrosis
- Cardiovascular:
  - Cyanotic congenital heart disease
  - Infective endocarditis
  - Arteriovenous shunts and aneurysms
- Gastrointestinal:
  - Cirrhosis
  - Inflammatory bowel disease
  - Coeliac disease
- Others:
  - Thyrotoxicosis (thyroid acropachy)
  - Primary hypertrophic osteoarthropathy

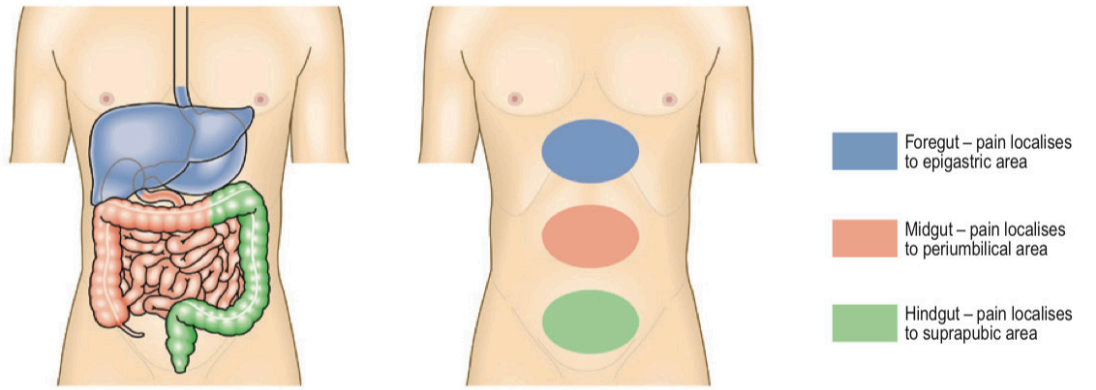


Fig. 6.4 Abdominal pain. Perception of visceral pain is localised to the epigastric, umbilical or suprapubic region, according to the embryological origin of the affected organ.

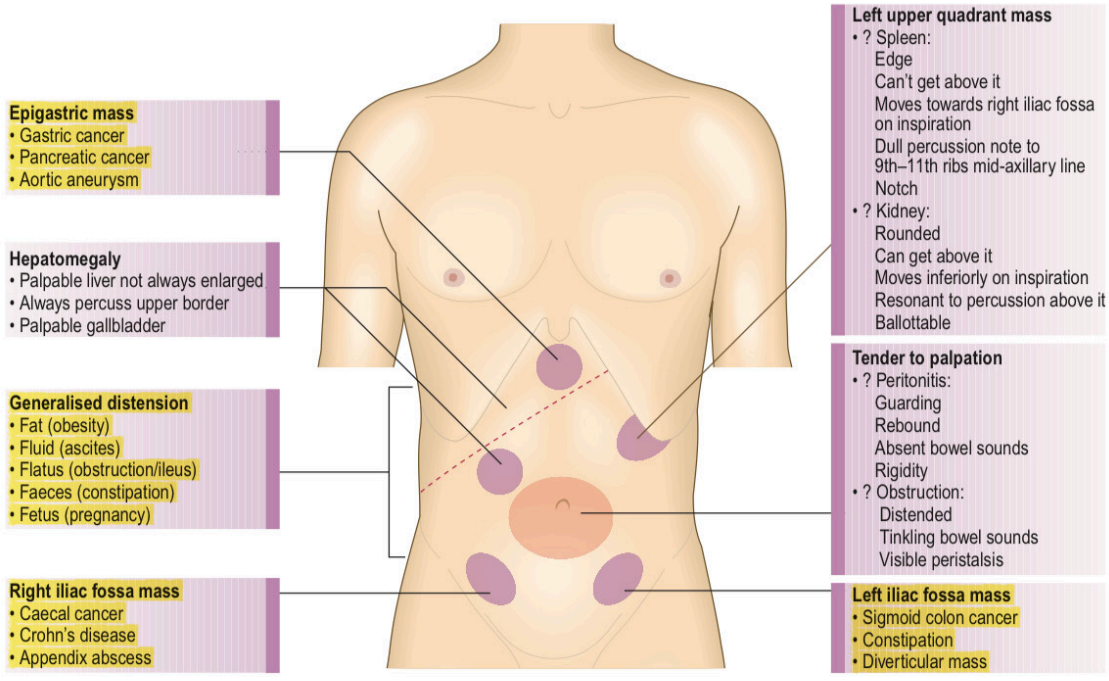


Fig. 6.12 Palpable abnormalities in the abdomen.

Macleod clinical picture

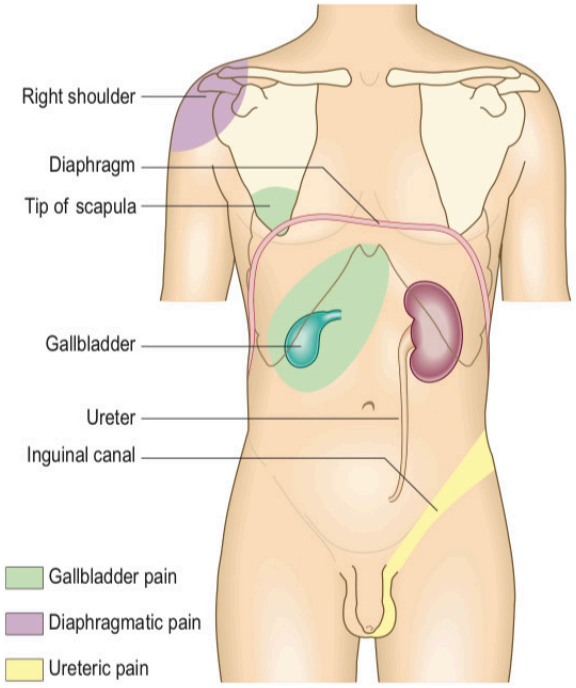


Fig. 6.5 Characteristic radiation of pain from the gallbladder, diaphragm and ureters.

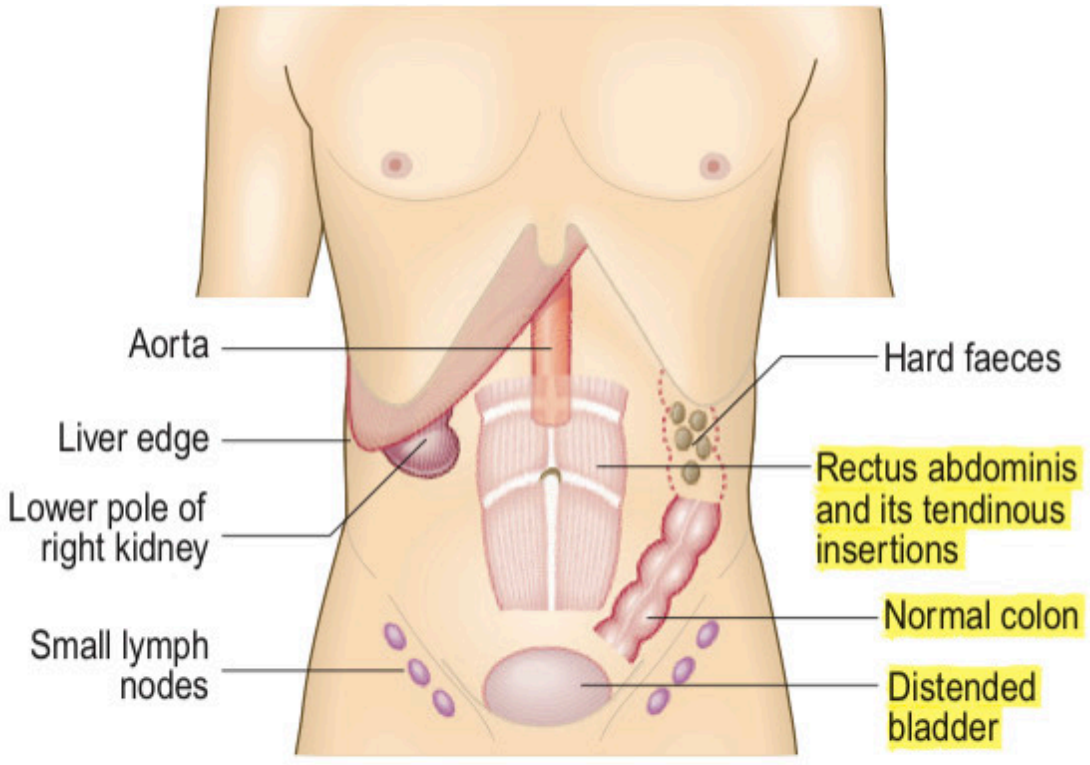


Fig. 6.13 Palpable masses that may be physiological rather than pathological.

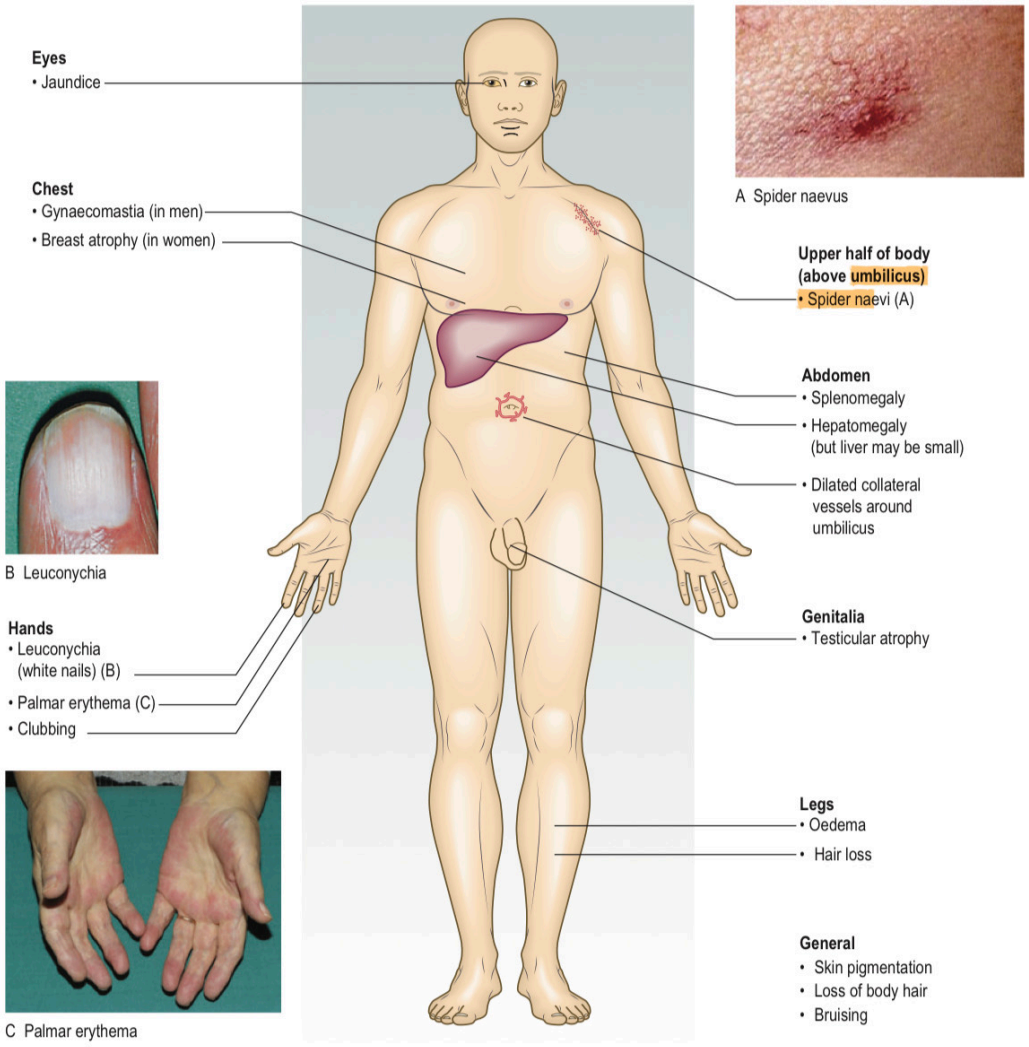


Fig. 6.9 Features of chronic liver disease.

السبب هوه زياده oxytocin يسبب

Palmar erythema,breast enlargement,spider naevi,testicular atrophy,hair loss

8.30 Signs of liver failure

- Fetor hepaticus: stale 'mousy' smell of the volatile amine, dimethyl sulphide, on the breath
- Flapping tremor of outstretched arms with hands dorsiflexed ('asterixis')

Mental state varies from drowsiness with day/night pattern reversed, through confusion and disorientation, to unresponsive coma

- Late neurological features
  - Spasticity and extension of the arms and legs
  - Extensor plantar responses

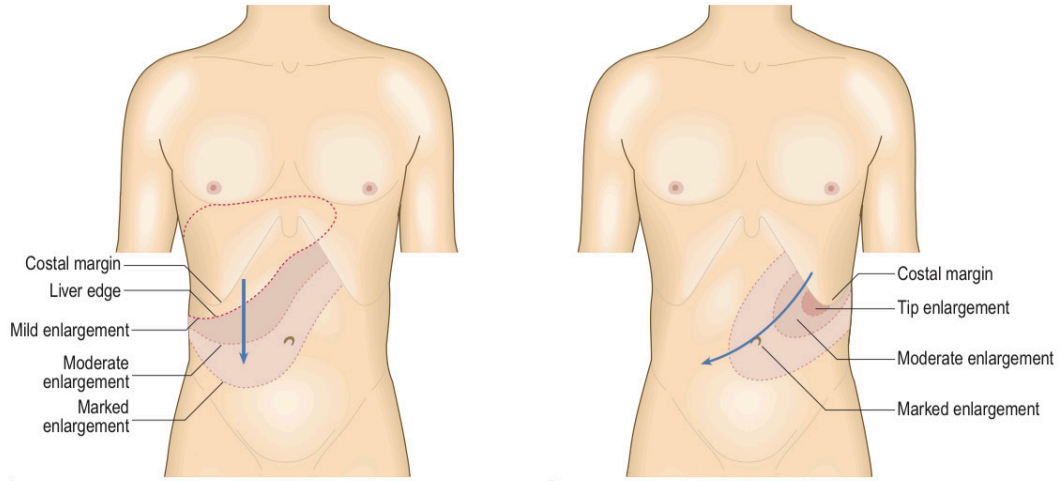


Fig. 6.15 Patterns of progressive enlargement of liver and of spleen. A Direction of enlargement of the liver. B Direction of enlargement of the spleen. The spleen moves downwards and medially during inspiration.

6.12 Differentiating a palpable spleen from the left kidney

Distinguishing feature	Spleen	Kidney
Mass is smooth and regular in shape	More likely	Polycystic kidneys are bilateral irregular masses
Mass descends in inspiration	Yes, travels superficially and diagonally	Yes, moves deeply and vertically
Ability to feel deep to the mass	Yes	No
Palpable notch on the medial surface	Yes	No
Bilateral masses palpable	No	Sometimes, e.g. polycystic kidneys
Percussion resonant over the mass	No	Sometimes
Mass extends beyond the midline	Sometimes	No (except with horseshoe kidney)

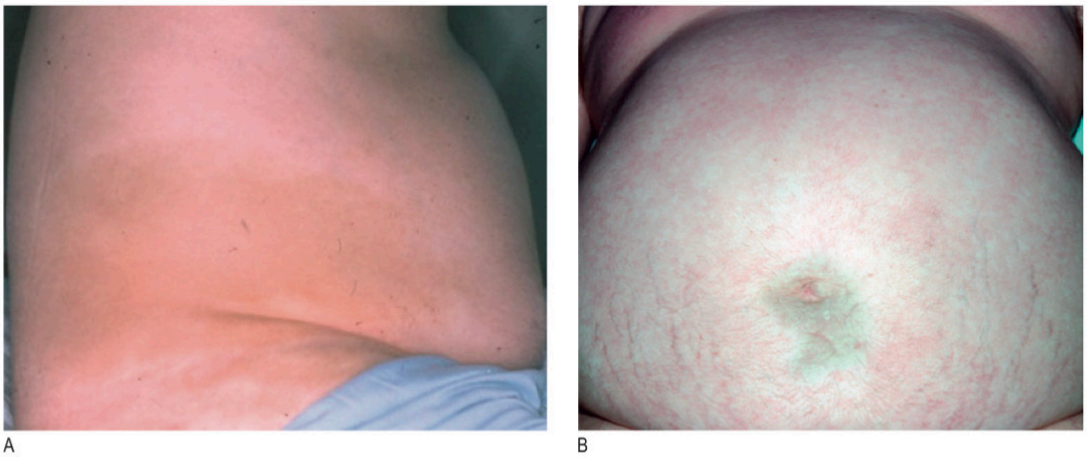
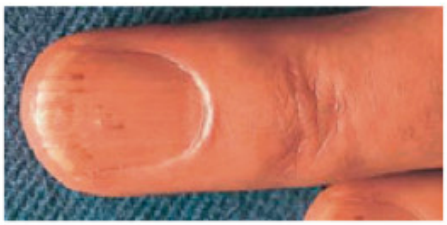
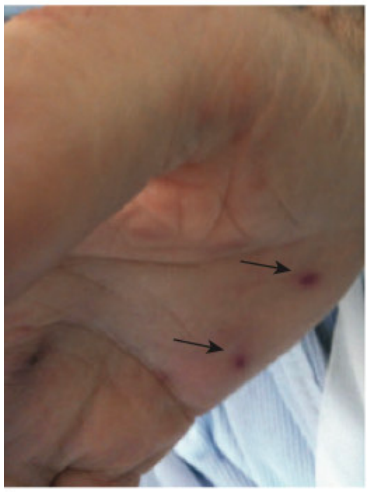


Fig. 6.25 Acute pancreatitis. A Bruising over the flanks (Grey Turner's sign). B Bruising round the umbilicus (Cullen's sign).

Cardiology

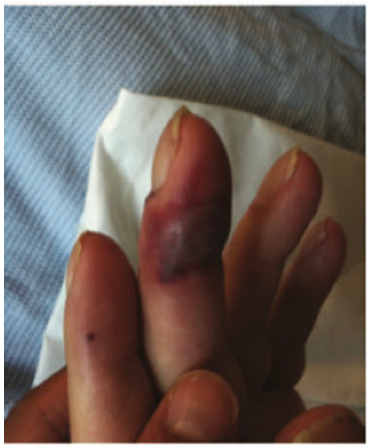


splinter haemorrhage (trauma, infective endocarditis)



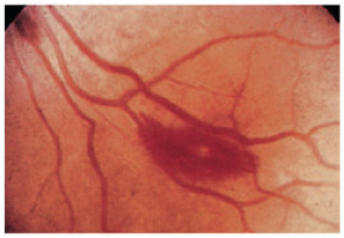
A

Janeway lesion (infective endocarditis)



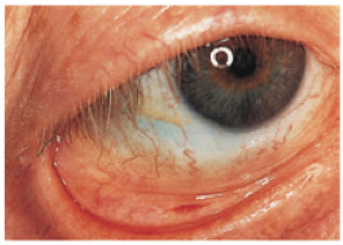
C

Osler nodues(infective endocarditis)



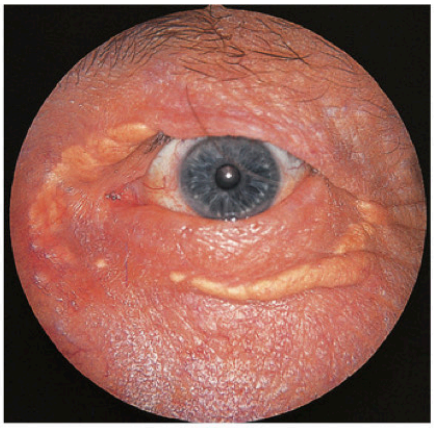
D

retinal haemorrhage (hypertension)



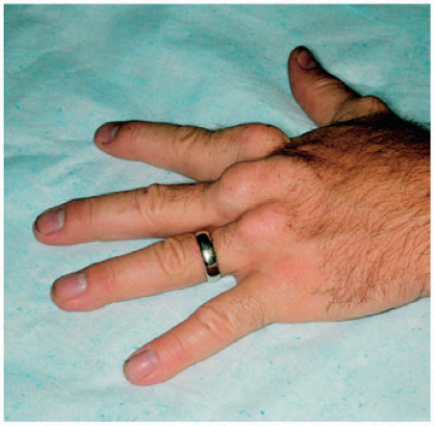
E

Petechial haemorrhage (infective endocarditis,trauma,anticoagulant, hypertension)



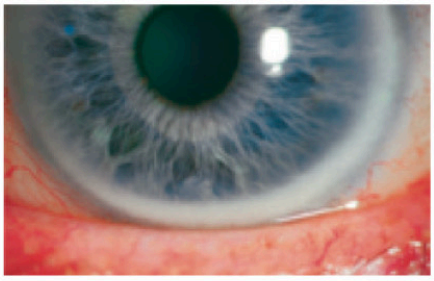
A

Xanthelasma (Hypercholesterolemia)



B

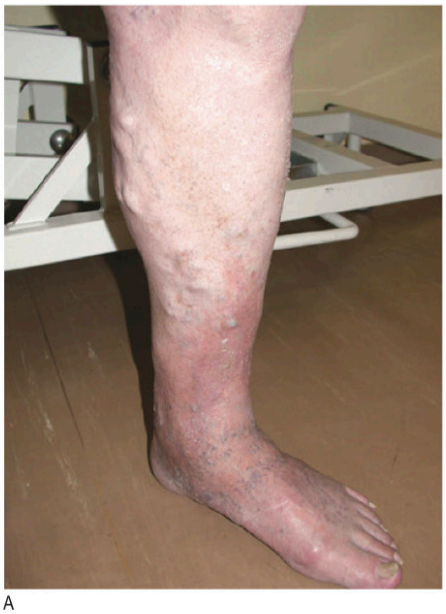
Tendon xanthoma (familial Hypercholesterolemia)



C

corneal arcus (hypercholesterolemia)

Fig. 4.6 Features of hyperlipidaemia. A Xanthelasmata. B Tendon xanthomata. C Corneal arcus. *(B) From Swartz M. Textbook of Physical Diagnosis. 6th edn. Philadelphia: Saunders; 2009. (C) From Kanski J. Clinical Diagnosis in Ophthalmology. London: Mosby; 2006.*



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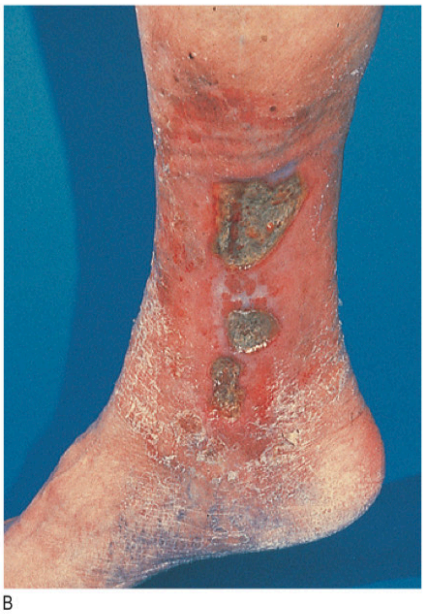


B

Fig. 4.37 Lower limb venous disease. A Varicose veins and associated haemosiderin deposition. B Venous ulcer. *(A) From Metcalfe M, Baker D. Varicose veins. Surgery (Oxford) 2008; 26(1):4–7.*

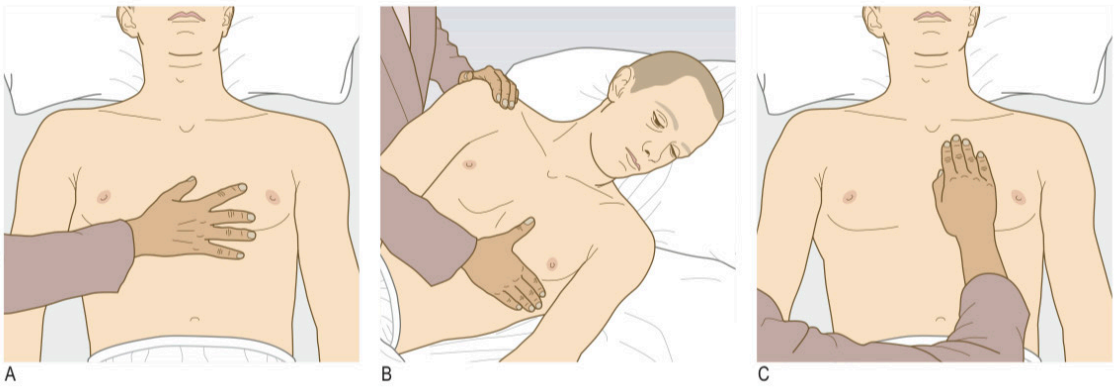
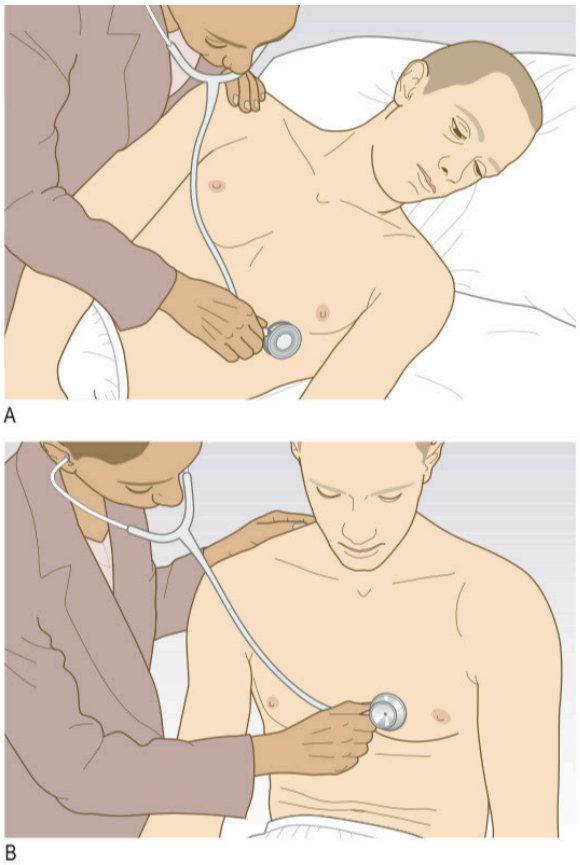


Fig. 4.18 **Palpating the heart.** A Use your hand to palpate the cardiac impulse. B Localise the apex beat with your finger (if necessary, roll the patient into the left lateral position). C Palpate from apex to sternum for parasternal pulsations.

Impalpable apex(obese,musculature,dextrocardia,pleural effusion,pericardial effusion,emphysema and asthma)



Auscultation of mid diastolic murmur of mitral stenosis

Causes of murmur (VSD,PDA,stenosis,regurgitation, functional)

Auscultation of early diastolic murmur of aortic regurgitation

Fig. 4.19 **Auscultating the heart.** A Listen for the murmur of mitral stenosis using the bell lightly applied with the patient in the left lateral position. B Listen for the murmur of aortic regurgitation using the diaphragm with the patient leaning forwards.

Fig. 4.7 The radial, brachial and carotid pulses. A Locating and palpating the radial pulse. B Feeling for a collapsing radial pulse. C Assessing the brachial pulse. D Locating the right carotid pulse with the fingers.

4.9 Causes of abnormal pulse rate or rhythm

Abnormality	Sinus rhythm	Arrhythmia
Fast rate (tachycardia, >100 bpm)	Exercise Pain Excitement/anxiety Fever Hyperthyroidism Medication: Sympathomimetics, e.g. salbutamol Vasodilators	Atrial fibrillation Atrial flutter Supraventricular tachycardia Ventricular tachycardia
Slow rate (bradycardia, <60 bpm)	Sleep Athletic training Hypothyroidism Medication: Beta-blockers Digoxin Verapamil, diltiazem	Carotid sinus hypersensitivity Sick sinus syndrome Second-degree heart block Complete heart block
Irregular pulse	Sinus arrhythmia Atrial extrasystoles Ventricular extrasystoles	Atrial fibrillation Atrial flutter with variable response Second-degree heart block with variable response

4.12 Causes of increased pulse volume

Physiological

- Exercise
- Pregnancy
- Advanced age
- Increased environmental temperature

Pathological

- Hypertension
- Fever
- Thyrotoxicosis
- Anaemia
- Aortic regurgitation
- Paget's disease of bone
- Peripheral atrioventricular shunt

4.11 Common causes of atrial fibrillation

- Hypertension
- Heart failure
- Myocardial infarction
- Thyrotoxicosis
- Alcohol-related heart disease
- Mitral valve disease
- Infection, e.g. respiratory, urinary
- Following surgery, especially cardiothoracic surgery

6.27 Abnormalities of the second heart sound

Quiet

Low cardiac output
Calcific aortic stenosis
Aortic regurgitation

Loud

Systemic hypertension (aortic component)
Pulmonary hypertension (pulmonary component)

Split

Widens in inspiration (enhanced physiological splitting):

Right bundle branch block
Pulmonary stenosis
Pulmonary hypertension
Ventricular septal defect

Fixed splitting (unaffected by respiration):

Atrial septal defect

Widens in expiration (reversed splitting):

Aortic stenosis
Hypertrophic cardiomyopathy
Left bundle branch block
Ventricular pacing

4.18 Abnormalities of intensity of the first heart sound

Quiet

- Low cardiac output
- Poor left ventricular function
- Rheumatic mitral regurgitation
- Long P–R interval (first-degree heart block)

Loud

- Increased cardiac output
- Large stroke volume
- Mitral stenosis
- Short P–R interval
- Atrial myxoma (rare)

Variable

- Atrial fibrillation
- Extrasystoles
- Complete heart block

6.28 Causes of a third heart sound

Physiological

- Healthy young adults
- Athletes
- Pregnancy
- Fever

Pathological

- Large, poorly contracting left ventricle
- Mitral regurgitation

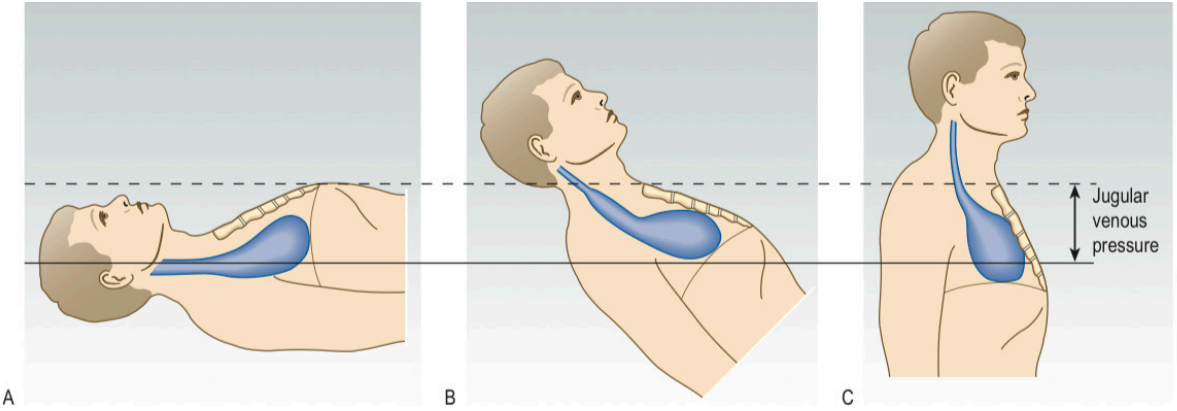


Fig. 4.14 Jugular venous pressure in a healthy subject. A Supine: jugular vein distended, pulsation not visible. B Reclining at 45 degrees: the point of transition between the distended and the collapsed vein can usually be seen to pulsate just above the clavicle. C Upright: the upper part of vein is collapsed and the transition point obscured.

4.15 Differences between carotid artery and jugular venous pulsation

Carotid	Jugular
Rapid outward movement	Rapid inward movement
One peak per heart beat	Two peaks per heart beat (in sinus rhythm)
Palpable	Impalpable
Pulsation unaffected by pressure at the root of the neck	Pulsation diminished by pressure at the root of the neck
Independent of respiration	Height of pulsation varies with respiration
Independent of the position of the patient	Varies with the position of the patient
Independent of abdominal pressure	Rises with abdominal pressure

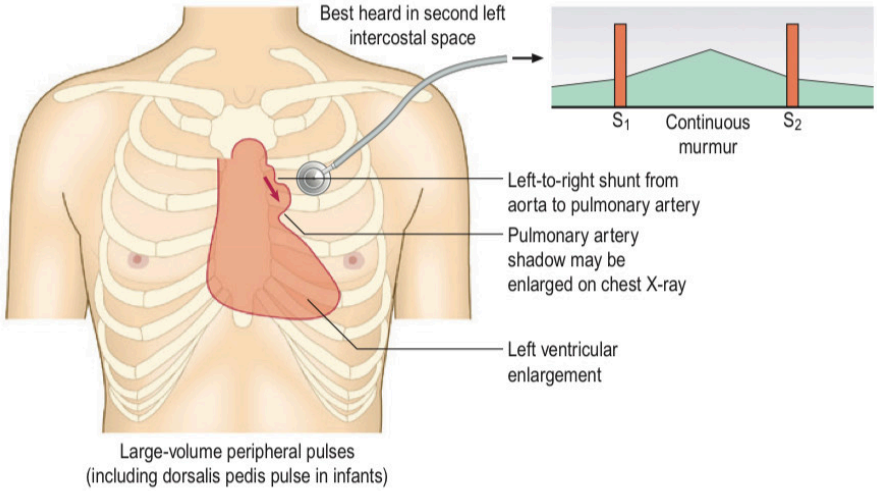


Fig. 4.27 Persistent patent ductus arteriosus. A continuous 'machinery' murmur is heard because aortic pressure always exceeds pulmonary arterial pressure, resulting in continuous ductal flow. The pressure difference is greatest in systole, producing a louder systolic component to the murmur.

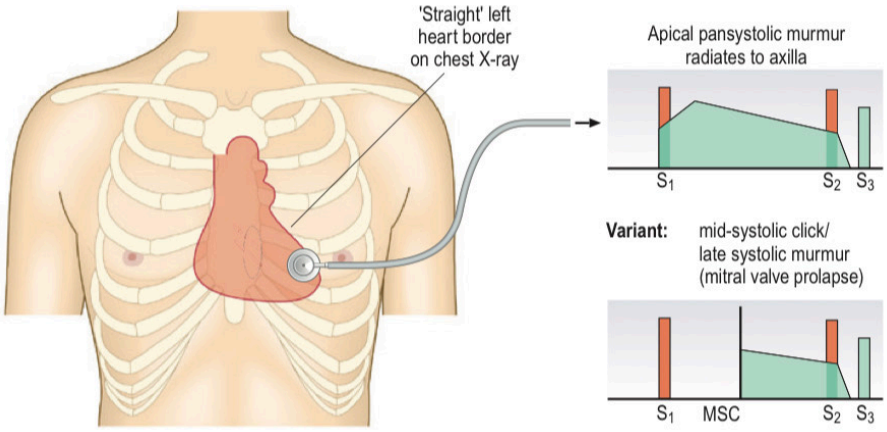


Fig. 4.23 Mitral regurgitation. Mitral regurgitation is caused by dilatation of the left ventricle and failure of leaflets to co-apt. The murmur begins at the moment of valve closure and may obscure the first heart sound. It varies little in intensity throughout systole. In mitral valve prolapse the murmur begins in mid- or late systole and there is often a mid-systolic click (MSC).

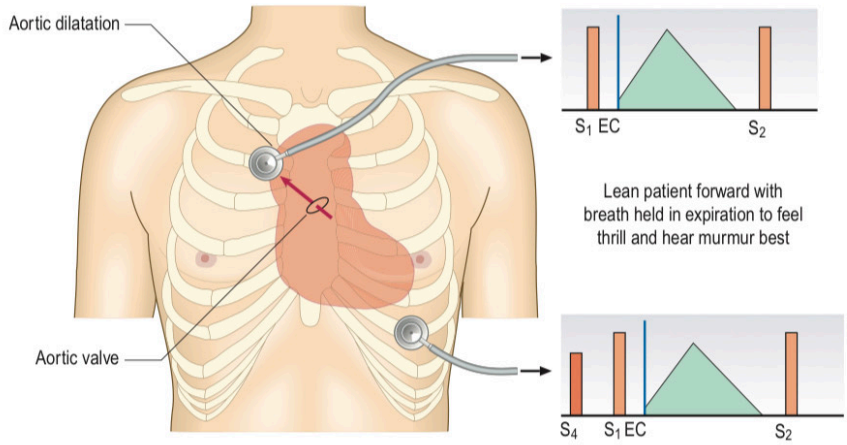


Fig. 4.24 Aortic stenosis. There is a systolic pressure gradient across the stenosed aortic valve. The resultant high-velocity jet impinges on the wall of the aorta *(arrow)*, and is best heard with the diaphragm in the aortic area. Alternatively, the bell may be placed in the suprasternal notch. The ejection systolic murmur follows an ejection click (EC).

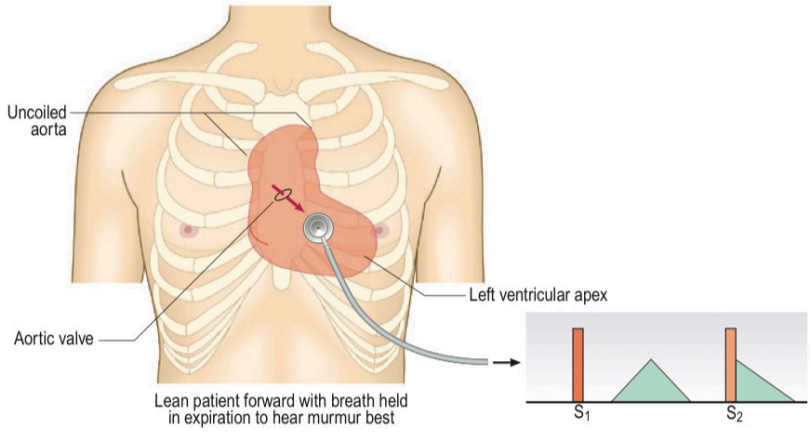


Fig. 4.25 Aortic regurgitation. The pulse pressure is usually increased. The jet from the aortic valve is directed inferiorly towards the left ventricular outflow tract *(arrow)* during diastole, producing an early diastolic murmur, best heard with the diaphragm during held expiration with the patient leaning forward. An associated systolic murmur is common because of the increased flow through the aortic valve in systole.

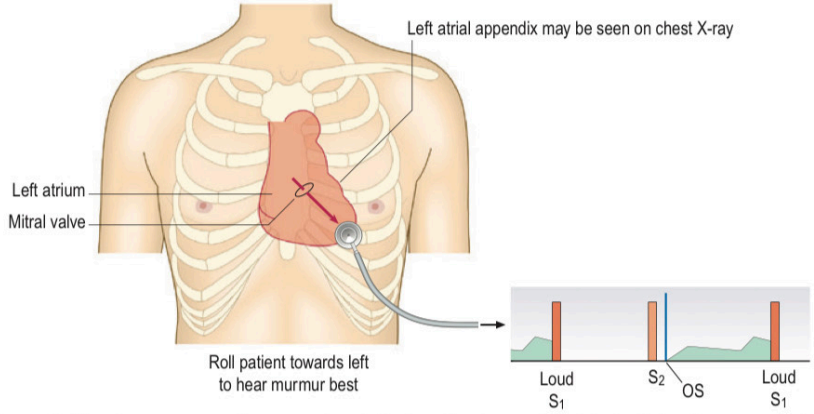


Fig. 4.26 Mitral stenosis. There is a pressure gradient across the mitral valve, giving rise to a low-pitched mid-diastolic murmur that is heard best with the bell at the apex and the patient rolled on to their left-hand side. The jet through the stenosed valve *(arrow)* strikes the endocardium at the cardiac apex. Occasionally, an opening snap (OS) can arise due to the sharp movement of the tethered anterior cusp of the mitral valve at the time when the flow commences.

Macleod clinical picture

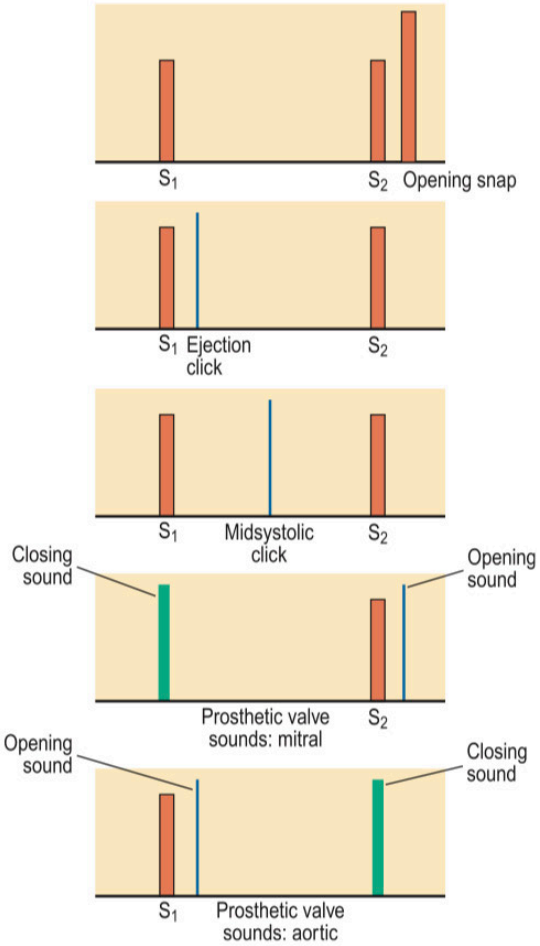
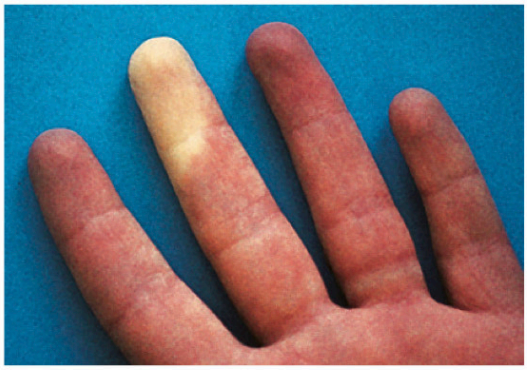


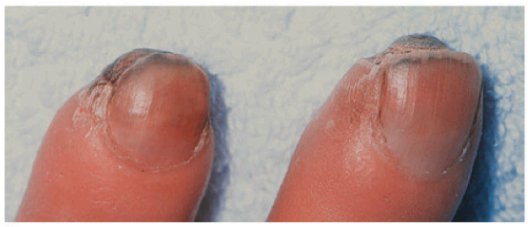
Fig. 4.22 'Added sounds' on auscultation.

4.20 Grades of intensity of murmur	
Grade	**Description**
1	Heard by an expert in optimum conditions
2	Heard by a non-expert in optimum conditions
3	Easily heard; no thrill
4	A loud murmur, with a thrill
5	Very loud, often heard over a wide area, with thrill
6	Extremely loud, heard without a stethoscope

General examination



A

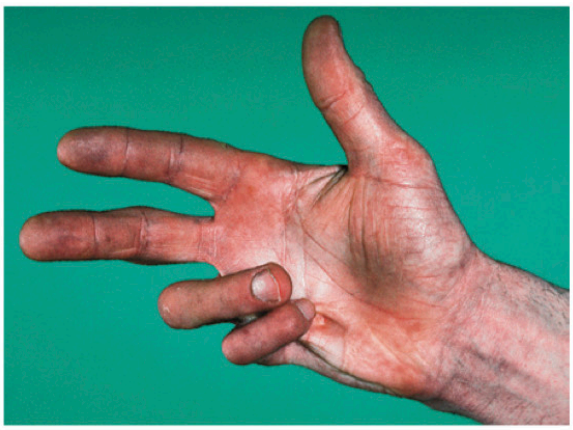


B

Fig. 4.33 Raynaud's syndrome. A The acute phase, showing severe blanching of the tip of one finger. B Raynaud's syndrome occasionally progresses to fingertip ulceration or even gangrene. *(A and B) From Forbes CD, Jackson WF. Color Atlas of Clinical Medicine. 3rd edn. Edinburgh: Mosby; 2003.*

Raynaud's phenomenon is digital ischaemia induced by cold and emotion. It has three phases

- pallor: due to digital artery spasm and/or obstruction
- cyanosis: due to deoxygenation of static venous blood (this phase may be absent)
- redness: due to reactive hyperaemia.



Dupuytren's contracture
Is a thickening of the palmer fascia causing fixed flexion deformity and usually affecting the little and ring fingers .

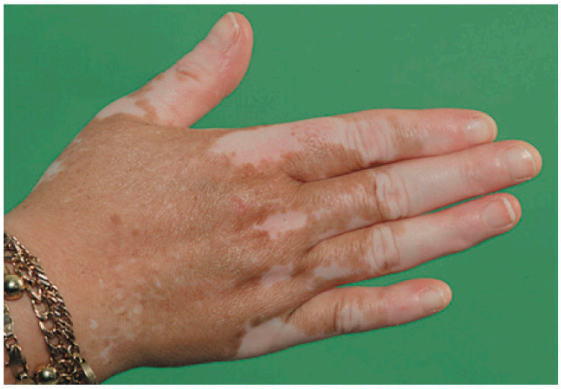


Fig. 3.10 Vitiligo.

Depigmentation occurs in the autoimmune condition vitiligo, in which there is often bilateral symmetrical depigmentation, commonly of the face, neck and extensor aspects of the limbs, resulting in irregular pale patches of skin

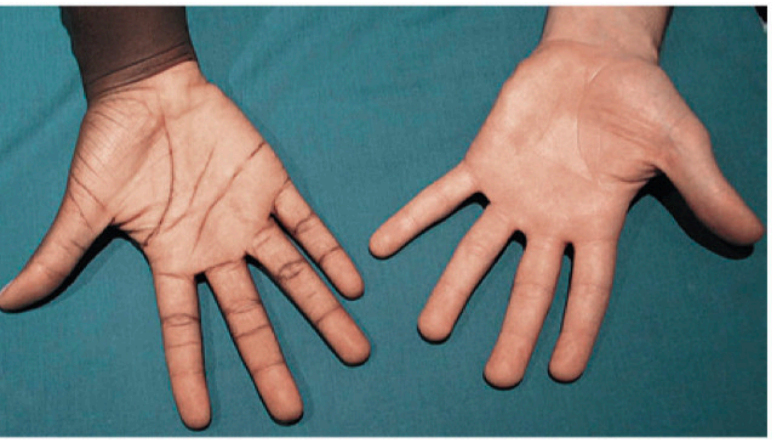


Fig. 3.6 Normal palms. African (left) and European (right).

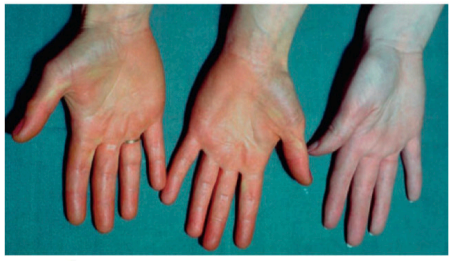


Fig. 3.13 **Hypercarotenaemia.** A control normal hand is shown on the right for comparison.

excessive ingestion of carotene-containing vegetables or in situations of impaired metabolism such as hypothyroidism or anorexia nervosa

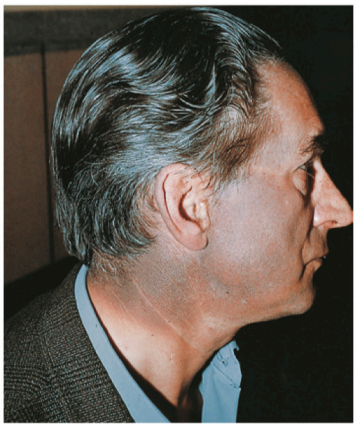


Fig. 3.14 Phenothiazine-induced pigmentation.

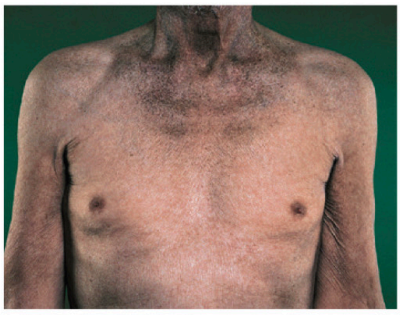


Fig. 3.11 Haemochromatosis with increased skin pigmentation.

excessive iron absorption results in skin hyperpigmentation due to iron deposition and increased melanin production

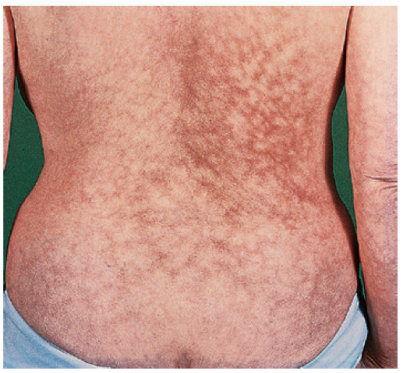
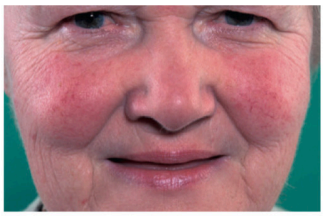
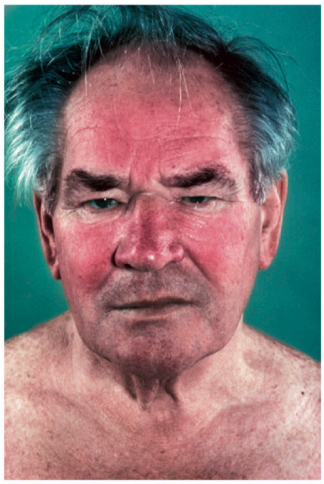


Fig. 3.12 Erythema ab igne.

Local deposition haemosiderin occurs with heat damage to the skin from sitting too close to a fire or from applying local heat, such as a hot water bottle, to the site of pain



A



B

Fig. 3.17 **Flushing due to carcinoid syndrome.** A Acute carcinoid flush. B Chronic telangiectasia.

3.6 Conditions associated with facial flushing

Physiological
- Fever
- Exercise
- Heat exposure
- Emotional

Drugs (e.g. glyceryl trinitrate, calcium channel blockers, nicotinic acid)

Anaphylaxis

Endocrine
- Menopause
- Androgen deficiency (in men)
- Carcinoid syndrome
- Medullary thyroid cancer

Others
- Serotonin syndrome
- Food/alcohol ingestion
- Neurological (e.g. Frey's syndrome)
- Rosacea
- Mastocytoses

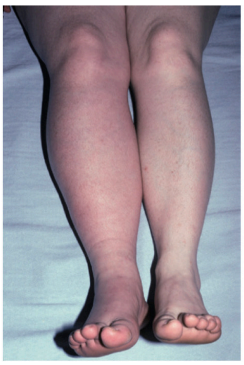


Fig. 3.22 **Swollen right leg, suggesting deep vein thrombosis or inflammation.** Causes include soft tissue infection or a ruptured Baker's cyst.

Swollen right leg, suggesting deep vein thrombosis or inflammation

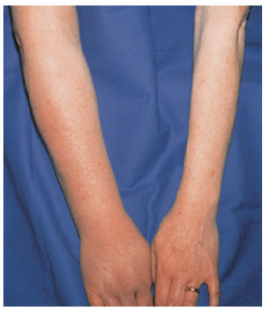


Fig. 3.23 Lymphoedema of the right arm following right-sided mastectomy and radiotherapy.

lymphoedema usually follows radical mastectomy and/ or irradiation for breast cancer.

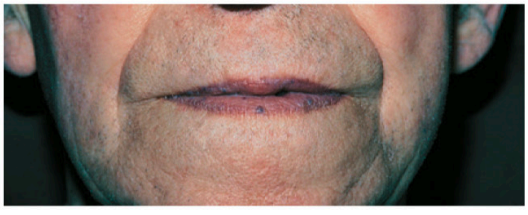
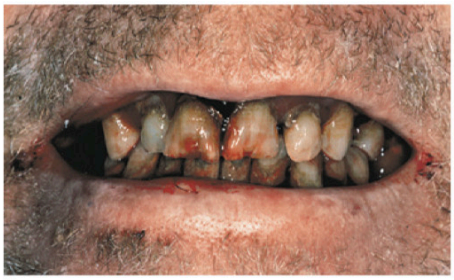
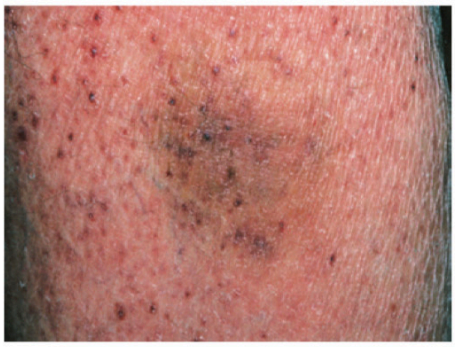


Fig. 3.18 Central cyanosis of the lips.



A



B

Fig. 3.19 Scurvy. A Bleeding gums. B Bruising and perifollicular haemorrhages.

scurvy is vitamin c deficiency

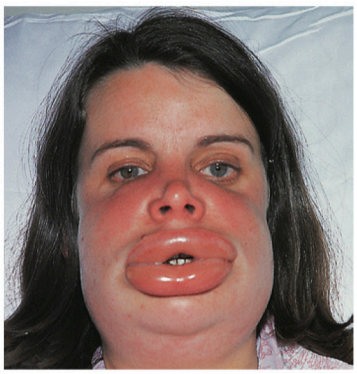
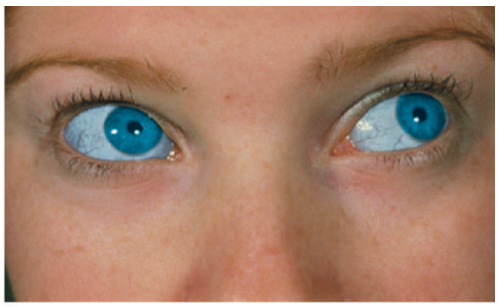


Fig. 3.24 Angio-oedema following a wasp sting.

3

Angio-oedema is a severe form of allergic oedema affecting the face, lips and mouth, most commonly caused by insect bites, food allergy or drug reactions. Swelling may develop rapidly and become life-threatening if the upper airway is involved.



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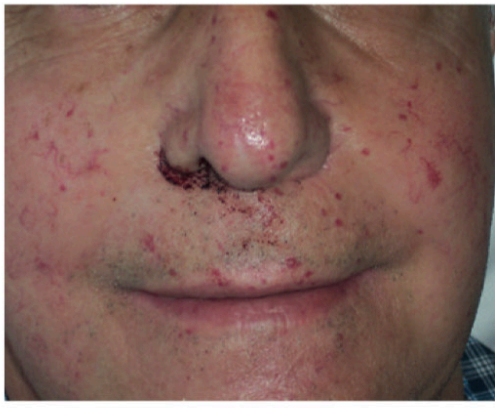
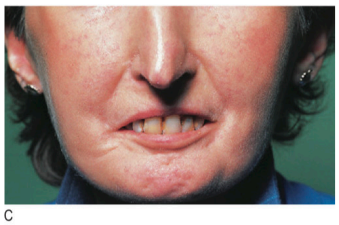


B

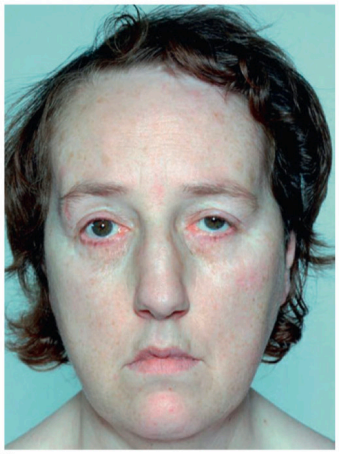
Osteogenesis imperfecta (Blue sclerae)

Hereditary haemorrhagic telangiectasia(Telangiectasia on and around lips)



C

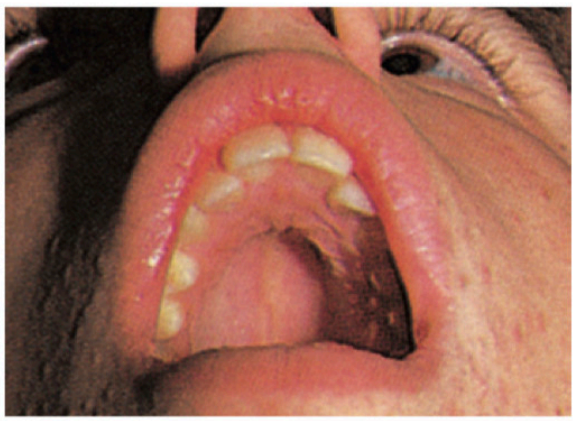
Systemic sclerosis with ‘beaking’ of the nose and taut skin around the mouth



D

myotonic dystrophy (Frontal balding, paucity of expression, bilateral ptosis)

A



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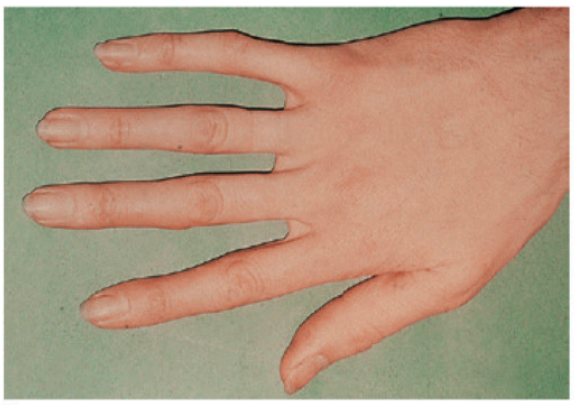

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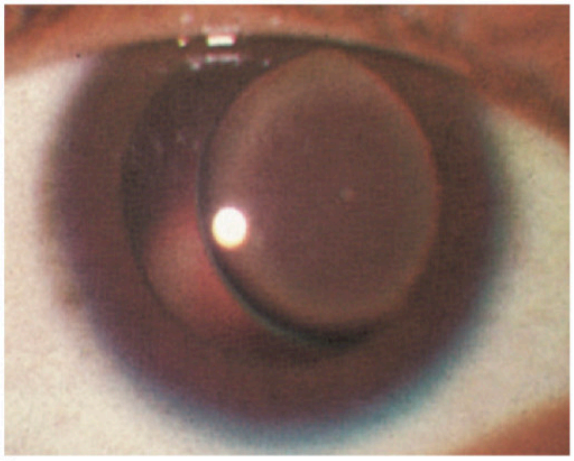
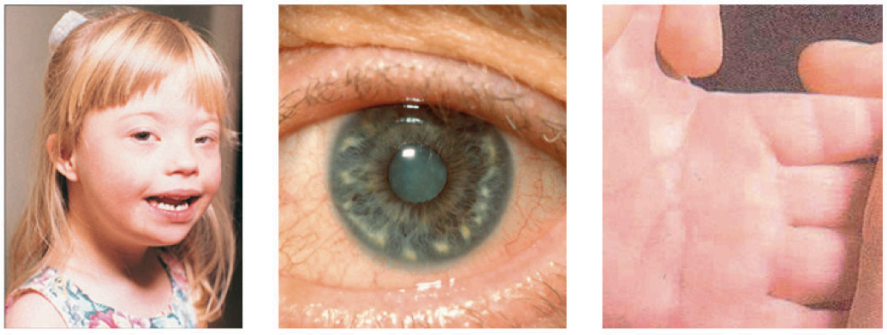


D

Fig. 3.21 Marfan’s syndrome, an autosomal dominant condition. A Tall stature, with the torso shorter than the legs (note surgery for aortic dissection). B Long fingers. C High-arched palate. D Dislocation of the lens in the eye. *(A–D) From Forbes CD, Jackson WF. Color Atlas of Clinical Medicine. 3rd edn. Edinburgh: Mosby; 2003.*



A B C

Fig. 3.31 Down's syndrome. A Typical facial appearance. B Brushfield spots: grey–white areas of depigmentation in the iris. C Single palmar crease. A *From Kerryn Phelps, Craig Hassed; Genetic conditions. In General Practice: The Integrative Approach, 1e, Churchill Livingstone; 2011.*

Flat facial profile,up-slanting palpebral fissures ,small, low-set ears,macroglossia, Brushfield spots in iris

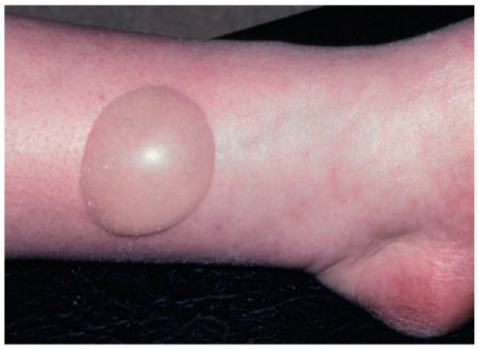


Fig. 3.25 Blister on a leg.

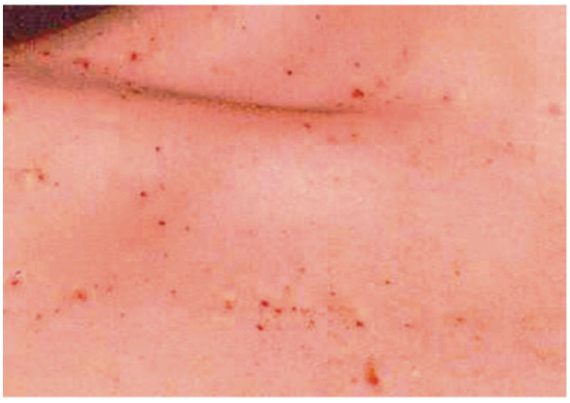
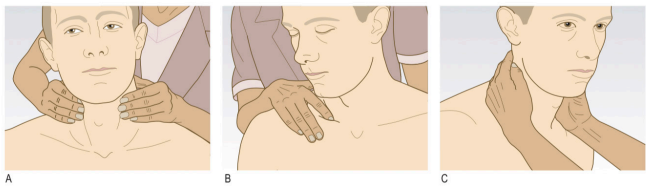
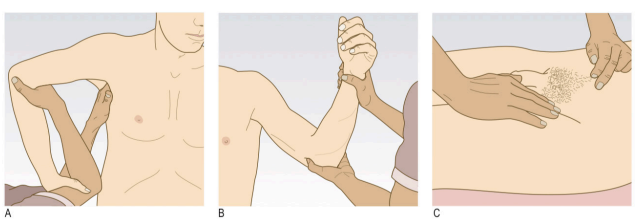


Fig. 3.29 Petechiae.



A B C

Fig. 3.27 Palpation of the cervical glands. A Examine the glands of the anterior triangle from behind, using both hands. B Examine for the scalene nodes from behind with your index finger in the angle between the sternocleidomastoid muscle and the clavicle. C Examine the glands in the posterior triangle from the front.



A B C

Fig. 3.28 Palpation of the axillary, epitrochlear and inguinal glands. A Examination for right axillary lymphadenopathy. B Examination of the left epitrochlear glands. C Examination of the left inguinal glands.

Examination of cervical lymph nodes

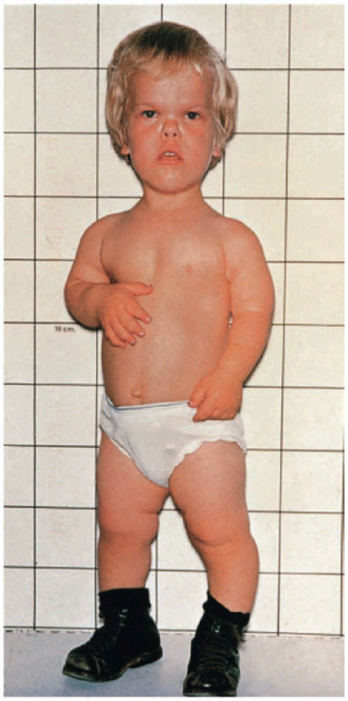


Fig. 3.33 **Child with achondroplasia.** *From Keith L. Moore, T. V. N. Persaud. Congenital Anatomic Anomalies or Human Birth Defects. in the Developing Human: Clinically Oriented Embryology, 8e; 2008.*

caused by mutation of the fibroblast growth factor gene. Although the trunk is of normal length, the limbs are very short and broad (Fig. 3.33). The vault of the skull is enlarged, the face is small and the bridge of the nose is flat.

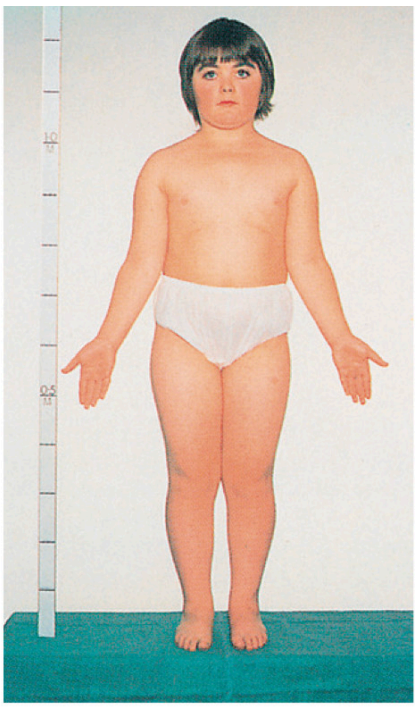


Fig. 3.32 **Turner's syndrome.** *From Henry M. Seidel, Jane Ball, Joyce Dain, G. William Benedict. Growth and measurement. In: Mosby's Guide to Physical Examination, 6e; 2006.*

Typical features include short stature, webbing of the neck, small chin, low-set ears, low hairline, short fourth finger, increased carrying angle at the elbows and widely spaced nipples ('shield-like chest').

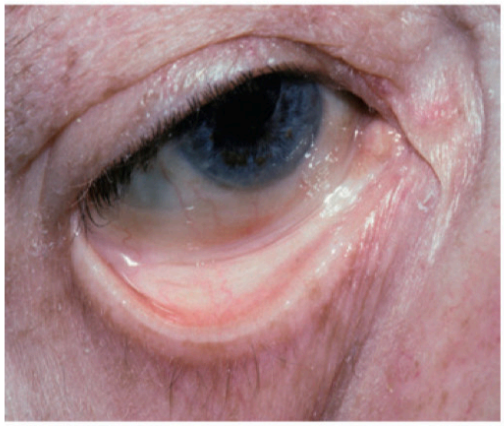


Fig. 3.15 **Conjunctival pallor.**

anemia

3.9 Conditions with characteristic facial appearances

Diagnosis	Facial features
Hypothyroidism (see Fig. 10.5)	Sparse, coarse hair and eyebrows, periorbital puffiness, dry, waxy skin, apathetic expression, macroglossia
Graves' disease (autoimmune thyrotoxicosis) (see Fig. 10.2A)	Staring appearance due to lid retraction, proptosis, evidence of weight loss
Hypopituitarism (see Fig. 10.10A)	Pale, often unwrinkled skin with loss of hair
Acromegaly (see Fig. 10.9A)	Thickened, coarse skin with enlarged nose and frontal bones, prognathism (lower jaw protrusion), widely spaced teeth, macroglossia
Cushing's syndrome (see Fig. 10.11A)	Moon-shaped plethoric facies
Osteogenesis imperfecta (see Fig. 3.30A)	Blue sclerae
Hereditary haemorrhagic telangiectasia (see Fig. 3.30B)	Telangiectasia on and around lips
Systemic sclerosis (see Fig. 3.30C)	Tight skin constricting mouth, 'beaking' of nose, loss of nasolabial folds
Myotonic dystrophy (see Fig. 3.30D)	Frontal balding, paucity of expression, bilateral ptosis
Down's syndrome (see Fig. 3.31)	Flat facial profile, up-slanting palpebral fissures, small, low-set ears, macroglossia, Brushfield spots in iris
Systemic lupus erythematosus	'Butterfly' erythematous rash on cheeks

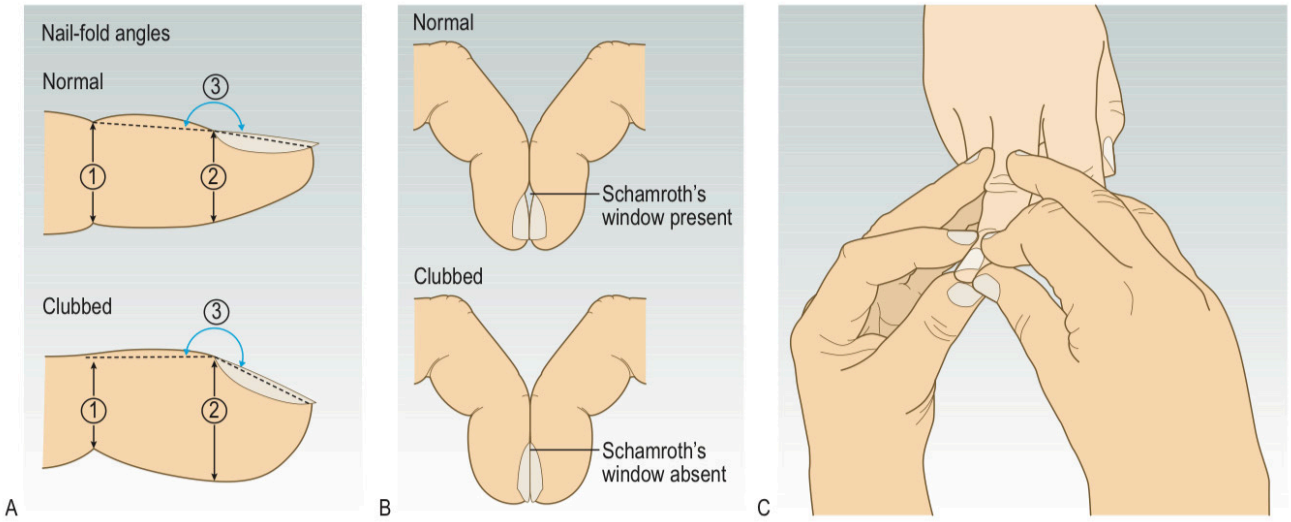
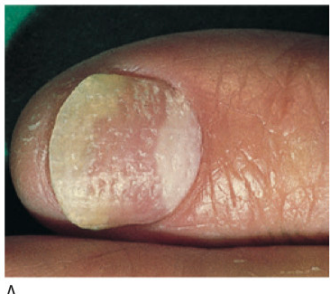
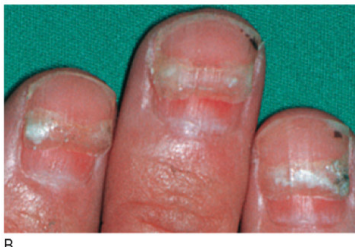


Fig. 3.9 Examining for finger clubbing. A Assessing interphalangeal depth at (**1**) interphalangeal joint and (**2**) nail bed, and nail-bed angle (**3**). B Schamroth's window sign. C Assessing nail-bed fluctuation.



A

Pitting nail (psoriasis)



B

Beau's lines ,are transverse depress white ridges, due to arrest of nail growth cause by (MI, infection, CT , Postoperative & poor nutrition).