joints. There are five distinct groups of psoriatic arthritis, but overlap is common.

1. Monoarticular and asymmetrical oligoarticular arthritis of the hands and feet and other joints (note sausage-shaped digits in Figure 9.46). Most psoriatic arthritis is of this type.

2. Symmetrical polyarthritis, similar to rheumatoid arthritis (but seronegative).

3. Distal interphalangeal joint involvement with psoriatic nail changes (Figure 9.12).

4. Arthritis mutilans (destructive polyarthritis).

5. Sacroiliitis with or without peripheral joint involvement.

X-ray findings (Figure 9.56): in mild cases X-rays are normal or show only joint-space narrowing and erosive changes. Unlike the X-rays of rheumatoid joints the bone density is maintained and there may be sclerotic changes in the small bones. Ankylosis of peripheral joints and *arthritis mutilans* can occur in either condition. The involvement of the spine and sacroiliac joints is asymmetrical, as in Reiter's syndrome.

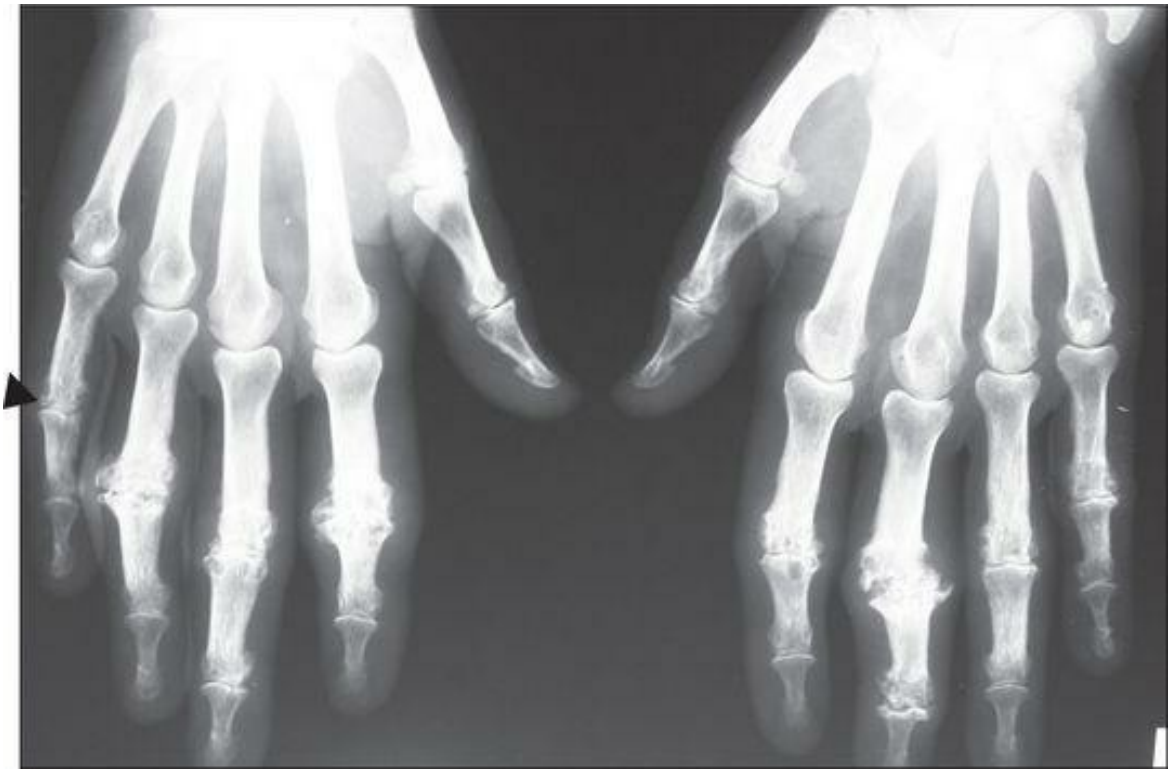


Figure 9.56 Psoriatic arthritis
X-ray of the wrist of a patient with psoriatic arthritis; note the 'pencil in cup' deformity (tapered proximal osseous surface and expanded base) of the distal bone of the fingers, early

(tapered proximal osseous surface and expanded base) of the distal bone of the fingers, early ankylosis [arrowhead] of the proximal interphalangeal joint of the right little finger and erosions with proliferative change of the little finger). Note also the lack of osteoporosis.

Courtesy Canberra Hospital X-ray library.

Enteropathic arthritis

There are two patterns of involvement of the joints with ulcerative colitis and Crohn's disease.

1. **Peripheral joint disease.** This is an asymmetrical oligoarthropathy, usually affecting the lower limbs, especially the knees and ankles. It rarely causes deformity.

2. **Sacroiliitis.** This is indistinguishable clinically from ankylosing spondylitis.

Gouty arthritis

Begin with the feet, as acute gouty arthritis affects the metatarsophalangeal joint of the great toe in 75% of cases. Next examine the ankles and knees, which tend to be involved after recurrent attacks. The fingers, wrists and elbows are affected late (Figure 9.57). Inspect and palpate for gouty tophi (these are urate deposits with inflammatory cells surrounding them) (Latin *tophus,* 'chalk stone'). The presence of tophi indicates chronic recurrent gout. They tend to occur over the joint synovia, the olecranon bursa, the extensor surface of the forearm, the helix of the ear (Figure 9.58), and in the infrapatellar and Achilles tendons.

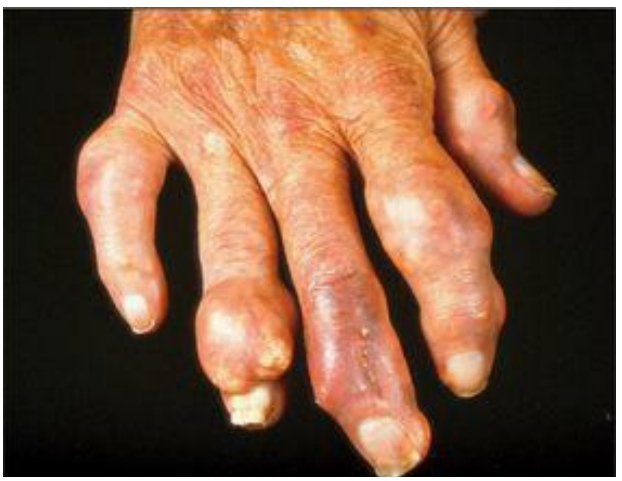


Figure 9.57 Gouty tophi of the fingers

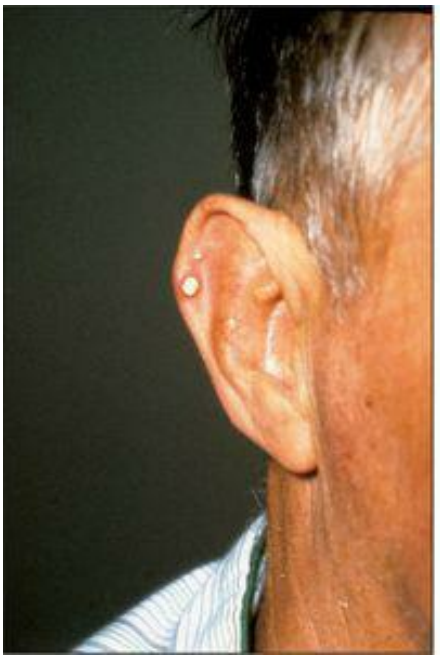
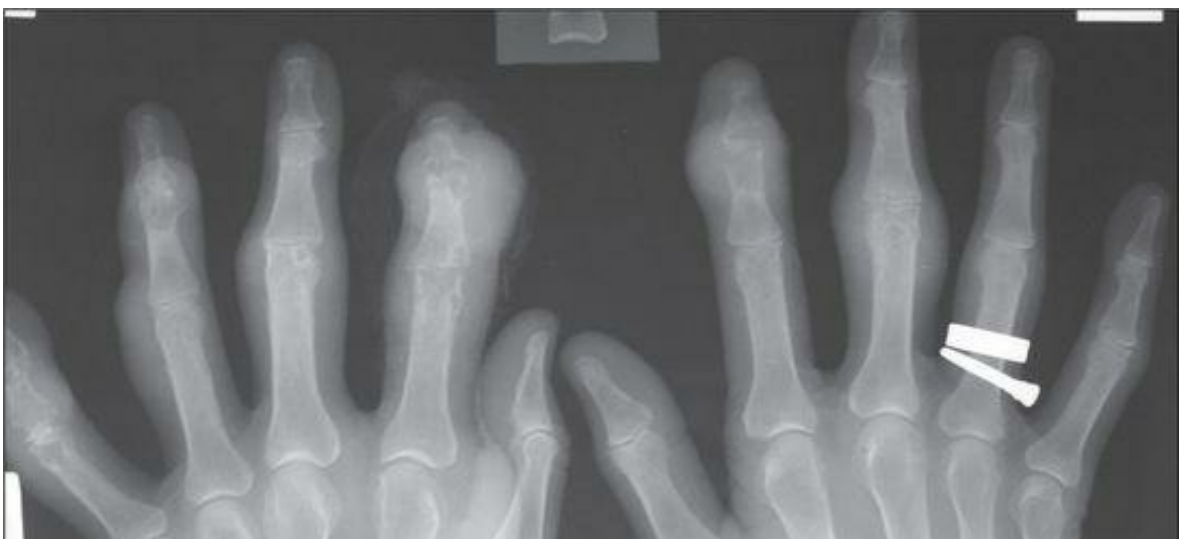


Figure 9.58 Gouty tophus of the ear

Finally, examine for signs of the causes of secondary gout: increased purine turnover due to myeloproliferative disease (page 236), lymphoma (page 237) or leukaemia; and decreased renal urate excretion due to renal disease or hypothyroidism. Hypertension, diabetes mellitus and ischaemic heart disease are more common among sufferers of gout.

X-rays (Figure 9.59) show multiple juxta-articular erosions which may obliterate the joint space.



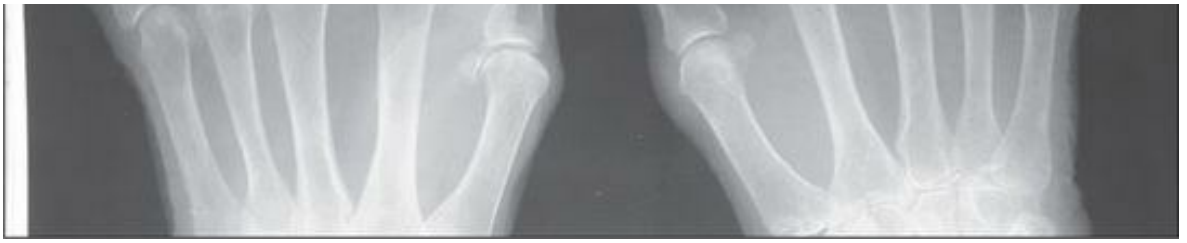


Figure 9.59 Gout
X-ray of the hands of a patient with severe gouty arthritis. Note multiple juxta-articular erosions with relative preservation of the joint space, and erosions with overhanging edges. There are large soft-tissue swellings over the distal interphalangeal joints of the index fingers.

Courtesy Canberra Hospital X-ray library.

Calcium pyrophosphate arthropathy (pseudogout)

This may present a similar picture to that described above for true gout, but usually large joints (especially the knees) and wrists are involved. In a minority of patients there will be signs of hyperparathyroidism, haemochromatosis or true gout.

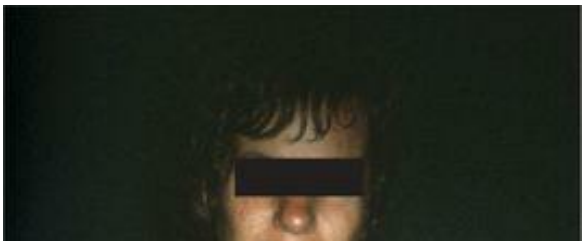
X-rays show joint-space narrowing, cyst formation under the cartilage and calcification of the joint cartilage (chondrocalcinosis). Chondrocalcinosis on X-ray is typical of pseudogout but is not always present.

Calcium hydroxyapatite arthropathy

This causes large-joint arthritis (especially knee and shoulder) and is more common in elderly patients.

Systemic lupus erythematosus (SLE)

This is a multisystemic chronic inflammatory disease of unknown origin, named because the erosive nature of the condition was likened to the damage caused by a hungry wolf (Latin *lupus*[x] 'wolf') (Figure 9.60).



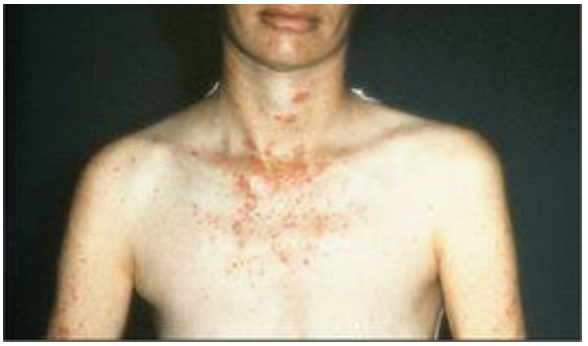


Figure 9.60 Systemic lupus erythematosus

1. **General inspection**
   Cushingoid appearance
   Weight
   Mental state

2. **Hands**
   Vasculitis
   Rash
   Arthropathy

3. **Arms**
   Livedo reticularis
   Purpura
   Proximal myopathy (active disease or steroids)

4. **Head**
   Alopecia, with or without scarring, lupus hairs
   Eyes—scleritis, cytoid lesions, etc.
   Mouth—ulcers, infection
   Rash—butterfly
   Cranial nerve lesions
   Cervical adenopathy

5. **Chest**
   Cardiovascular system—pericarditis
   Respiratory system—pleural effusion, pleurisy, pulmonary fibrosis, collapse or infection

6. **Abdomen**
   Hepatosplenomegaly
   Tenderness

7. **Hips**
   Aseptic necrosis

8. **Legs**
   Feet—red soles, small-joint synovitis
   Rash
   Ulcers over the malleoli, e.g. antiphospholipid syndrome
   Proximal myopathy
   Neuropathy
   Mononeuritis multiplex
   Cerebellar ataxia
   Hemiplegia

9. Other
   Urine analysis (proteinuria)
   Blood pressure (hypertension)
   Temperature chart

General inspection

Look for weight loss (due to chronic inflammation) or a Cushingoid appearance (steroid treatment). While taking the history note any abnormal mental state—psychosis may occur due to the lupus itself or to steroid therapy.

Hands

Note any vasculitic-appearing lesions around the nail bed, or telangiectasia and erythema of the skin of the nail base. A rash may occur—photosensitivity is common. The hand rash of lupus tends to occur over the phalanges, as opposed to that of dermatomyositis which affects the knuckles.

Raynaud's phenomenon may occur if the weather is cold (Table 9.7).

Examine for arthropathy: synovitis of the proximal and metacarpophalangeal joints. The arthritis of SLE is not erosive, but if severe may lead to reducible deformities due to damage to supporting structures.

Forearms

Livedo reticularis may occur here; in Latin this describes skin discoloration in the form of a small net. This is formed by connected bluish-purple streaks without discrete borders. They occur usually on the limbs and are associated

with various connective tissue diseases.[11] Look for purpura (due to vasculitis[12] or autoimmune thrombocytopenia). Examine for a proximal myopathy (due to the disease itself or to steroid treatment). Subcutaneous nodules very rarely occur in SLE. The axillary nodes may be enlarged but will not be tender.

The head and neck

Alopecia (hair loss) is an important diagnostic clue that occurs in about two-thirds of patients and may be associated with scarring. Look especially for lupus hairs, which are short, broken hairs above the forehead. The hair as a whole may be coarse and dry, as in hypothyroidism.

Examine the eyes for scleritis and episcleritis (see Figure 9.50). The eyes may be red and dry (Sjögren's syndrome). Pallor of the conjunctivae occurs with anaemia, usually due to chronic disease. Occasionally jaundice due to autoimmune haemolytic anaemia may be found. Perform a fundoscopy for cytoid bodies, which are hard exudates (white spots) due to aggregates of swollen nerve fibres and are secondary to vasculitis.

A facial rash may be diagnostic (Figure 9.61). The classical rash is an erythematous 'butterfly rash' over the cheeks and bridge of the nose and must be distinguished from rosacea. Mouth ulcers on the soft or hard palate may occur and the mouth may be dry (Sjögren's syndrome).

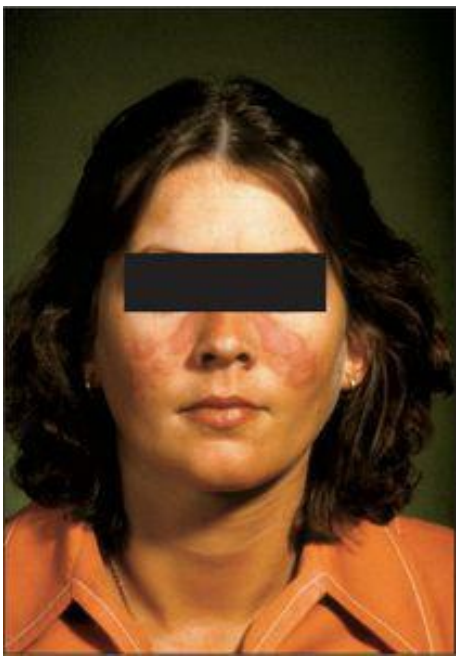


Figure 9.61 Butterfly rash of systemic lupus erythematosus

The *rash of discoid lupus* may be found in the same area or affect

different parts of the body. Lesions begin as spreading red plaques which have a central area of hyperkeratosis and follicular plugging. An active lesion has an oedematous edge. The appearance may suggest psoriasis. A healed lesion may have marginal hyperpigmentation with central atrophy and depigmentation. The scalp, external ear and face are most commonly affected, but in some patients lesions may occur all over the arms and chest. Extensive annular or psoriaform lesions may indicate the presence of subacute cutaneous lupus.

After examining the face, feel for cervical lymphadenopathy, which is usually non-tender.

The chest

Signs of a pericardial rub (from pericarditis) may be found. In the respiratory examination a pleural rub (pleuritis) or signs suggesting the presence of a pleural effusion, pulmonary fibrosis, pulmonary collapse or pulmonary hypertension may be detected. Chest disease is probably most often secondary to an interstitial pneumonitis rather than to vasculitis of the lungs.

The abdomen

Splenomegaly, usually mild, can be detected in 10% of cases. Hepatomegaly (mild) may occur in uncomplicated cases. Chronic liver disease due to chronic hepatitis ('lupoid hepatitis') is a separate autoimmune disease rather than a variant of SLE.

The hips

Examine the hip joint movements: in aseptic (avascular) necrosis there is pain on movement, with preservation of hip extension but loss of the other movements. This is due to ischaemia of the femoral head and may be related to steroid use or to the SLE itself.

The legs

Examine for proximal myopathy and peripheral neuropathy (mainly sensory).

Rarely there may be signs of hemiplegia, cerebellar ataxia or chorea.

Leg ulceration over the malleoli, due to vasculitis or the antiphospholipid syndrome,[11] is important. Very occasionally the toes may be gangrenous. There may be ankle oedema from the nephrotic syndrome or fluid retention

from steroids. Livedo reticularis may be present on the legs.

Urine and blood pressure

Perform a urine analysis (for proteinuria and haematuria) and take the blood pressure (for hypertension). Renal disease is a common complication of SLE.

Temperature

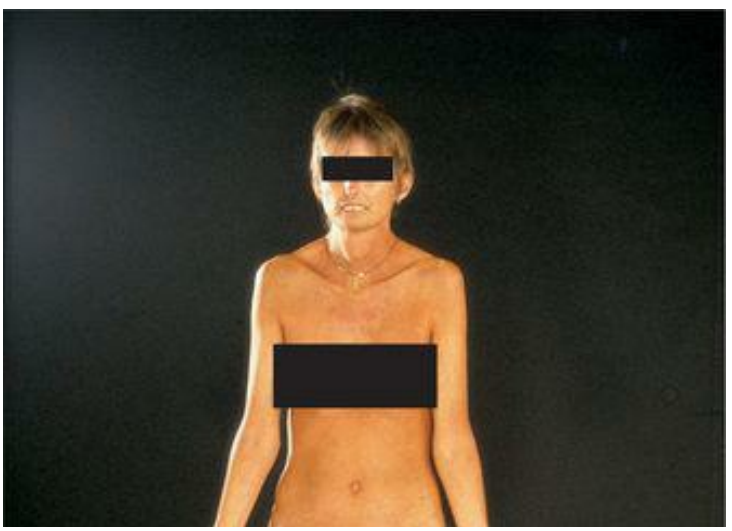
Take the temperature, as fever is common in SLE, either from secondary infection or from the disease per se.

Scleroderma (progressive systemic sclerosis)

This is a disorder of connective tissue with variable cutaneous fibrosis and with abnormalities of the microvasculature of the fingers, gut, lungs, heart and kidneys. In diffuse cutaneous scleroderma there is more prominent skin sclerosis and these patients may have pulmonary fibrosis. In limited cutaneous scleroderma (CREST syndrome—***C****alcinosis*, ***R****aynaud's* phenomenon, *o* ***E****sophageal* motility disturbance, ***S****clerodactyly* and ***T****elangiectasia*), diffuse skin sclerosis and interstitial lung disease do not occur but patients are at risk of developing pulmonary hypertension.

General inspection (Figure 9.62)

Look for cachexia due to dysphagia (from an oesophageal motility disturbance) or malabsorption (due to bacterial overgrowth).



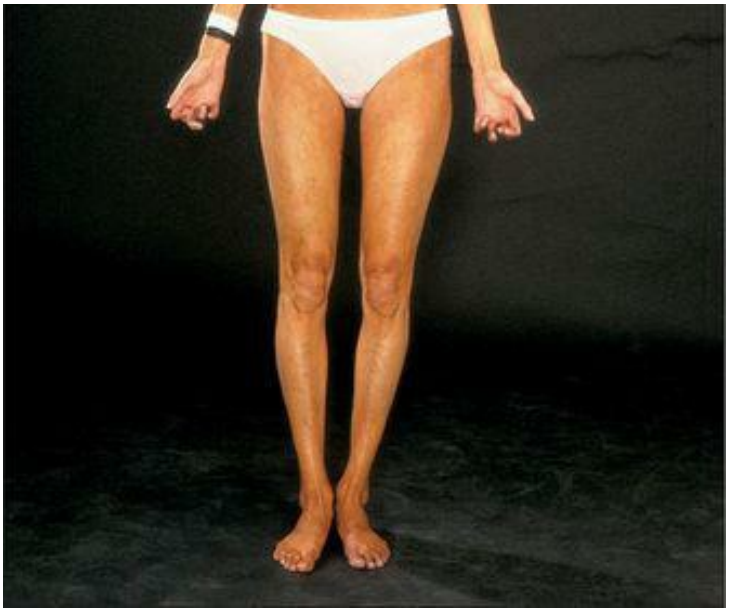


Figure 9.62 Scleroderma

1. General appearance
   'Bird-like' facies
   Weight-loss (malabsorption)

1. Hands
   CRST—calcinosis, atrophy distal tissue pulp (Raynaud's), sclerodactyly, telangiectasia
   Dilated capillary loops (nailfolds)
   Small-joint arthropathy and tendon crepitus
   Fixed flexion deformity
   Hand function

3. Arms
   Oedema (early), or skin thickening and tightening
   Pigmentation
   Vitiligo
   Hair loss
   Proximal myopathy

4. Head
   Alopecia
   Eyes—loss of eyebrows, anaemia, difficulty with closing
   Mouth—puckered, difficulty with opening
   Pigmentation
   Telangiectasia
   Neck muscles—wasting and weakness

5. Dysphagia

6. Chest
   Tight skin ('Roman breastplate')
   Heart—cor pulmonale, pericarditis, failure
   Lungs—fibrosis, reflux pneumonitis, chest infections

7. Legs
   Skin lesions
   Vasculitis

8. Other
   Blood pressure (hypertension with renal involvement)
   Urine analysis (proteinuria)
   Temperature chart (infection)
   Stool examination (steatorrhoea)

Skin changes in scleroderma vary. There may be an early oedematous phase with non-tender pitting oedema of the hands which appear tightly swollen. In patients with progressive disease the oedematous skin is replaced by indurated skin which appears thickened, hard and tight. This phase usually begins in the fingers (Table 9.17).

TABLE 9.17 Differential diagnosis of thickened tethered skin

Systemic sclerosis (scleroderma), diffuse type; milder changes in limited cutaneous scleroderma
Mixed connective tissue disease (a distinct disorder with features of scleroderma, systemic lupus erythematosus, rheumatoid arthritis and myositis)
Eosinophilic fasciitis—widespread skin thickening due to

inflammation of the fascia often following excessive muscle exercise; occurs in association with eosinophilia and hypergammaglobulinaemia
Localised morphea—heterogeneous group of disorders where there are small areas of sclerosis: most common type is morphea, which begins with large plaques of red or purple skin that evolve into sclerotic areas and may regress spontaneously over years
Chemically induced: vinyl chloride, pentazocine, bleomycin
Pseudoscleroderma: porphyria cutanea tarda, acromegaly, carcinoid syndrome
Scleroedema: thickened skin over the shoulders and upper back in diabetes mellitus
Graft versus host disease
Silicosis
Eosinophilic myalgia syndrome (L-tryptophan)
Toxic oil syndrome

The hands

Examine the hands. Note particularly *calcinosis* (palpable nodules due to calcific deposits in the subcutaneous tissue of the fingers), *Raynaud's phenomenon* sometimes causing atrophy of the finger pulps (due to ischaemia) (Figure 9.63), *sclerodactyly* (tightening of the skin of the fingers leading to tapering), and multiple large *telangiectasia* on the fingers (Figure 9.64).

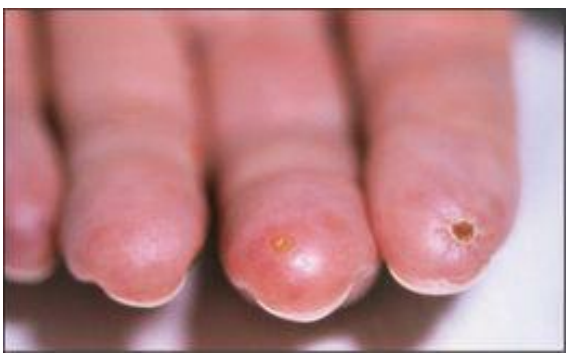


Figure 9.63 Digital infarcts

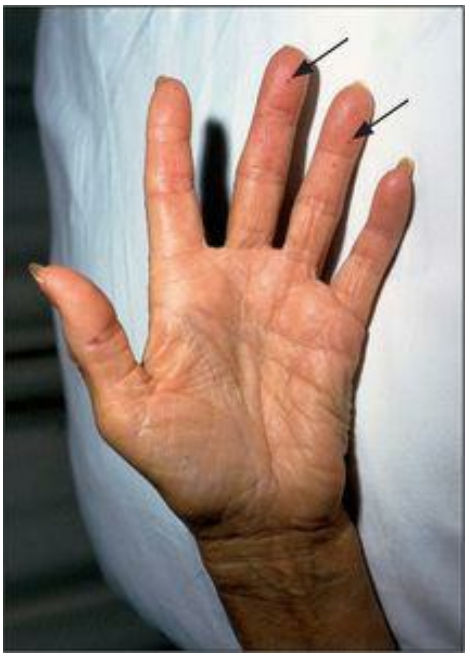
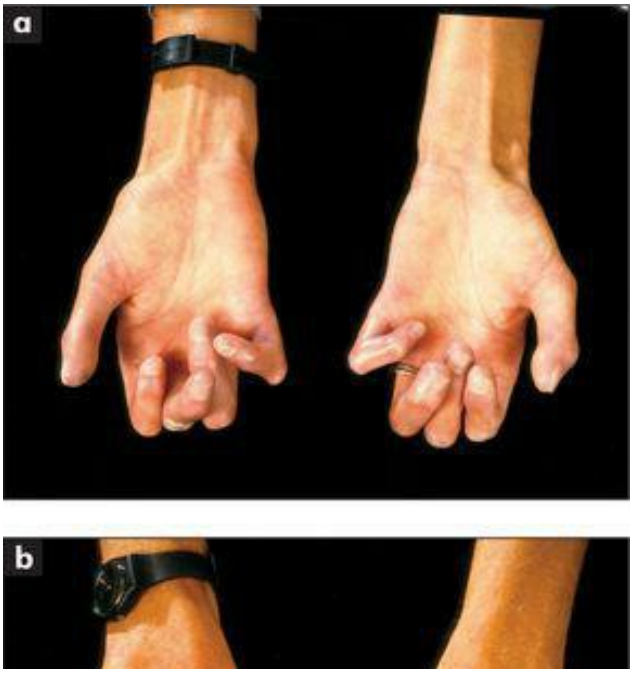


Figure 9.64 Telangiectasia of the hands in the CREST syndrome

Look for *contraction deformity of the fingers*, which is relatively common (Figure 9.65), and for synovitis, although this is uncommon. The nails can be affected by Raynaud's. It can be useful to inspect the nailfolds using a hand-held magnifying glass: in scleroderma you may see dilated capillary loops but this is not diagnostic. These are best viewed on the fourth digit. Assessing hand function is important in this disease.



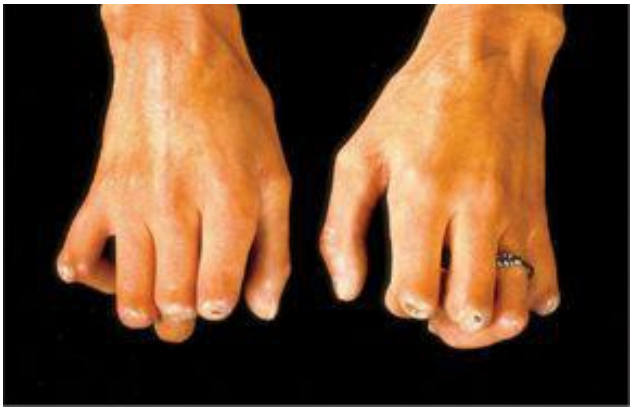


Figure 9.65 Systemic sclerosis: signs in the hands
Sclerodactyly, tethered smooth skin, calcinosis and ulceration, atrophy of finger pulps due to Raynaud's phenomenon, and fixed flexion deformities of the fingers.

The arms

Determine the extent of skin tethering in the arms. If the skin thickening extends above the wrists to the arms, legs or trunk, the diagnosis is *diffuse scleroderma* rather than *CREST*. If the skin thickening extends only to the elbows and face this is called *limited scleroderma*. Assess for proximal myopathy due to myositis.

The face

The skin of the face is involved in progressive disease. There is loss of normal wrinkles and skinfolds as well as of the eyebrows. The face appears pinched and expressionless ('bird-like' facies). Inspect for malar telangiectasia and look for salt-and-pepper pigmentation. Ask the patient to close the eyes—skin tethering may make this incomplete. The eyes may be dry (Sjögren's syndrome), though this is uncommon, and the conjunctivae pale (there are a number of reasons for anaemia, including the presence of chronic disease, bleeding from oesophagitis, watermelon stomach and microangiopathic haemolytic anaemia).

Ask the patient to open the mouth fully. It may appear puckered and narrow. Inability to open the mouth so that there is more than 3 cm of clearance between the incisors indicates abnormal restriction.

The chest

Inspect the skin of the chest wall, which may have acquired a tight, thickened

appearance, like ancient Roman breastplate armour.

Examine the lungs for pulmonary fibrosis, evidence of reflux pneumonitis, or (rarely) a pleural effusion or alveolar cell carcinoma.

Examine the heart for cor pulmonale secondary to pulmonary fibrosis or for pericarditis. Left ventricular failure may also occur due to myocardial involvement.

The legs

Look for signs of vasculitis, ulceration and skin involvement. Peripheral neuropathy is rare.

Urinalysis and blood pressure

These are very important because renal involvement is common in scleroderma and is often associated with severe hypertension. Renal disease is one of the most common causes of death.

The stool

Look for evidence of steatorrhoea (due to malabsorption from bacterial overgrowth). A summary of the physical signs that can be found in scleroderma is presented in Figure 9.62.

Rheumatic fever

This is an inflammatory disease which is a delayed sequel to infection with group A beta-haemolytic *Streptococcus*; it is uncommon in Western nations today. It is diagnosed by finding two major or one major and two minor criteria, plus evidence of recent streptococcal infection.

Major criteria: (i) carditis (causing tachycardia, murmurs, cardiac failure, pericarditis); (ii) polyarthritis; (iii) chorea (page 399); (iv) erythema marginatum (see below); (v) subcutaneous nodules (painless mobile swellings).

Minor criteria: (i) fever; (ii) arthralgia; (iii) previous rheumatic fever; (iv) acute phase proteins; (v) prolonged PR interval on the ECG.

Examining the patient with suspected rheumatic fever

First examine the large joints of the limbs for effusions and synovitis. Two or

First examine the large joints of the limbs for effusions and synovitis. Two or more joints must be involved (classically there is a transient migratory polyarthritis). Feel for subcutaneous nodules over bony prominences. Look for a rash. Erythema marginatum is a slightly raised pink or red rash that blanches with pressure. The red rings have a clear centre and round margins, and occur on the trunk and proximal limbs; the rash is not found on the face. Look for choreiform movements. Their onset is usually delayed until about 3 months after the throat infection.

Now examine the cardiovascular system for any signs of pancarditis: (i) a pericardial rub due to pericarditis; (ii) congestive cardiac failure due to myocarditis; (iii) mitral or aortic regurgitation due to acute endocarditis.

Finally, take the temperature.

The vasculitides

This is a heterogeneous group of disorders characterised by inflammation and damage to blood vessels.[12] The clinical features and major vessels involved are shown in Table 9.18.

TABLE 9.18 The vasculitides

Name	Vessels	Characteristics
Small vessel vasculitis		
Wegener's* granulomatosis	Small to medium-sized capillaries, venules, arterioles, small arteries	Granulomatous inflammation affecting the respiratory tract, often with necrotising glomerulonephritis
Saddle-nose deformity		
Churg-Strauss syndrome	Small	Asthma, eosinophilia, skin nodules, mononeuritis multiplex, pulmonary infiltrates
Henoch-Schonlein purpura	Small	Children affected; purpura over buttocks, abdominal pain, arthritis of knee and ankle, nephritis (40%)
Microscopic polyangiitis	Small	Glomerulonephritis, alveolar haemorrhage, neuropathy, pleural effusions
Mixed essential	Small	Arthritis, palpable purpura of extremities, Raynaud's disease,

cryoglobulinaemia	Small	neuropathy
		Hepatitis C common
Medium-sized vessel vasculitis		
Polyarteritis nodosa	Medium-sized to small	Myalgia, arthralgia, fever, palpable purpura, skin ulceration or infarction, weight loss, testicular tenderness, neuropathy (involvement of vasa nervorum), hypertension, renal infarction
		Hepatitis B associated
Kawasaki's disease	Medium-sized (coronary artery involvement)	Children affected; desquamating rash over extremities, strawberry tongue
Large vessel vasculitis		
Giant cell arteritis (temporal arteritis)	Medium to large (temporal and ophthalmic arteries and their branches)	Localised headache, systemic symptoms, tenderness temporal artery, jaw pain, visual loss—posterior ciliary artery (age ≥50 years)
Takayasu's disease[‡]	Large (aorta, brachial, carotid, ulnar and axillary arteries)	Systemic symptoms, claudication, loss of pulses (typically Asian race ≤40 years)

GIT = gastrointestinal tract. SLE = systemic lupus erythematosus. PAN = polyarteritis nodosa. CNS = central nervous system.

* Frederich Wegener, German pathologist, described this in 1936.

† Mikito Takayasu (1860–1938), Japanese professor of ophthalmology.

Soft-tissue rheumatism

This includes a number of common, painful conditions that arise in soft tissue, often around a joint. The problem may be general—for example, fibromyalgia; or restricted to a single anatomical region—for example, tendon, tenosynovium, enthesis or bursa. There are a large number of these conditions; the more common ones are described here.

Fibromyalgia syndrome

This syndrome is a common, frequently overlooked condition that mostly affects women in their 40s and 50s. It presents with a variable group of symptoms including widespread musculoskeletal aches and pains, and usually with symptoms of chronic fatigue. The musculoskeletal pain is mostly axial (neck and back) and diffuse. It is made worse by stress or cold. Pain may be felt 'all over' and is unresponsive to anti-inflammatory drug treatment. The combination of pain and fatigue may cause the patient severe disability. There is usually a poor sleep pattern. The patient wakes up not feeling refreshed, and more tired in the morning than later in the day. Note that no abnormal pathology has been found in the joints, muscles or tendons of these patients.

Examination

Test for the characteristic multiple hyperalgesic tender points (Figure 9.66). These areas may be tender to finger pressure in normal people but in affected patients there is marked tenderness and a definite withdrawal response. This response should be obtained in at least 11 of 18 sites in the upper and lower limbs and on both sides (i.e. it is widespread and symmetrical). Next examine for hyperalgesia at control sites such as the forehead or distal forearm, where it should be absent.

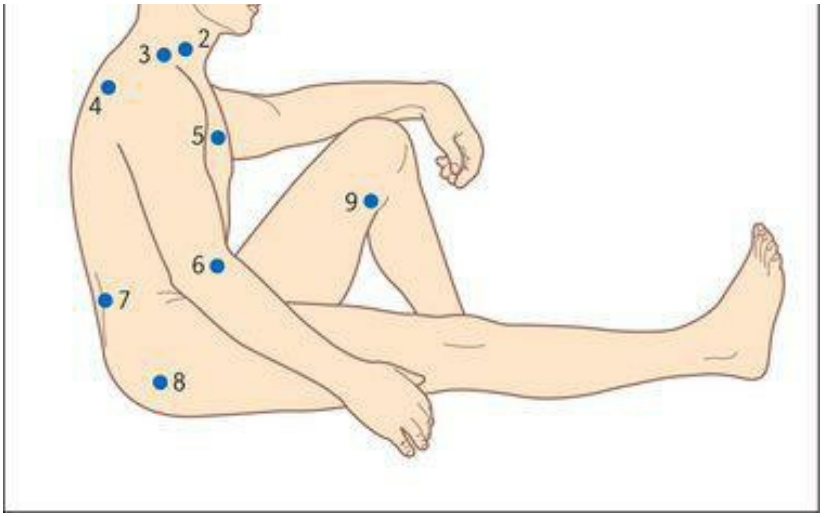


Figure 9.66 Frequent sites of localised tenderness in fibromyalgia
18 tender point sites (test bilaterally) are:

1. Insertion of suboccipital muscle
2. Under lower sternocleidomastoid muscle
3. Insertion of supraspinatus muscle
4. Trapezius muscle (mid upper)
5. Near second costochondral junction
6. 2 cm distal to lateral epicondyle
7. At prominence of greater trochanter
8. Upper outer quadrant of buttock
9. At medial fat pad of knee

The diagnosis is based on the presence of typical symptoms and multiple hyperalgesic tender sites (with negative control sites). Inflammatory and endocrine disease must be excluded.

Shoulder syndromes

Soft-tissue disorders of the shoulder are common and have certain particular clinical features.

Rotator cuff syndrome

Supraspinatus tendinitis is the commonest form of rotator cuff syndrome. It is associated with degeneration and subsequent inflammation in the supraspinatus tendon as it is compressed between the acromion and humeral head when the arm is raised. It mostly affects 40- to 50-year-olds. Symptoms may begin following unaccustomed physical activities such as gardening.

Examination

Examine the shoulder joint. Note pain on abduction of the arm (Figure 9.67), with a painful arc of movement between 60 and 120 degrees of abduction. Involvement of other rotator cuff tendons causes similar painful movement. Biceps tendinitis is present in the majority of patients with a rotator cuff syndrome. Yergason's[v] sign for biceps tendinitis is helpful (positive LR 2.8).[10] The patient flexes the elbow to 90 degrees and pronates the wrist. The examiner holds the wrists and attempts to prevent the patient's attempts to supinate the forearm. Inflammation of the head of the biceps causes pain in the shoulder since this muscle is the main supinator of the forearm.

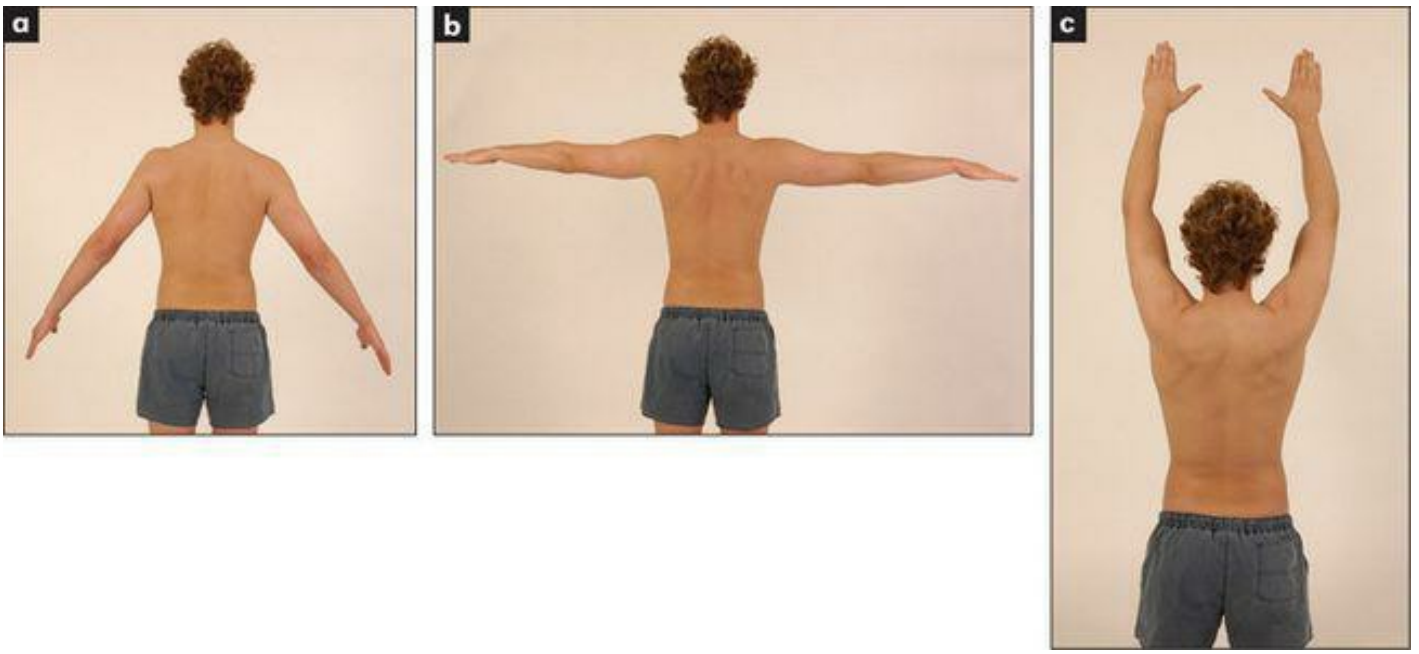


Figure 9.67 Inflammation of the rotator cuff tendons may cause a 'painful arc' during abduction of the arm
The initial movement (a) is painless but the next 90 degrees of movement (b) causes pain. When the arm reaches full abduction (c) the pain eases as the pressure is taken off the rotator cuff apparatus.

Frozen shoulder

Capsulitis of the shoulder, or frozen shoulder, is associated with limitation of active and passive arm movements in all directions. It may follow immobilisation of the arm after a stroke. There is typically a sudden onset of shoulder pain, which is worse at night and radiates to the base of the neck and down the arm. Pain is made worse by shoulder movement and may be bilateral. Pain and stiffness usually subside over a period of months. Complete movement may not be regained.

Examination

Examine the shoulders. There is global restriction of both active and passive movement of the shoulder—that is, it is frozen.

Elbow epicondylitis (tennis and golfer's elbow)

Many contact and non-contact sports can cause physical injury, although serious injuries are rather uncommon with certain sports (e.g. synchronised swimming). There may be pain over the epicondyles of the elbow. The *lateral epicondyle* is the most often affected and is called 'tennis elbow'. Pain arises from the site of insertion of the extensor muscle tendons into the lateral epicondyle (enthesis). Involvement of the medial epicondyle at the site of insertion of the flexor tendons of the forearm causes medial epicondylitis —'golfer's elbow'. These conditions are also common in manual workers such as painters.

Examination

Examine for local tenderness over the lateral (Figure 9.68) or medial epicondyle (Figure 9.69). Ask the patient to extend the fingers against resistance (Figure 9.70). This will make the pain of lateral epicondylitis worse. Ask the patient to flex the fingers against resistance. This will exacerbate the pain of medial epicondylitis.

Figure 9.68 Examination of the elbow
Looking for signs of lateral epicondylitis. Palpation over the forearm extensor muscle origin elicits pain. Straining the muscles by resisted extension of the wrist exacerbates the symptoms.

Figure 9.69 Examination to elicit signs of medial epicondylitis
Local pressure over the medial epicondyle elicits pain. Symptoms are exacerbated by resisted flexion of the wrist and fingers.

Figure 9.70 Testing for lateral epicondylitis

Figure 9.70 Testing for lateral epicondylitis

Tenosynovitis of the wrist

Inflammation of the synovial tubes in which tendons run can occur in patients with rheumatoid arthritis but also in otherwise healthy people. The cause is often unaccustomed repetitive movement. A common site for tenosynovitis is at the wrist, where it involves the long extensor and abductor tendons of the thumb (de Quervain's tenosynovitis; Figure 9.71).

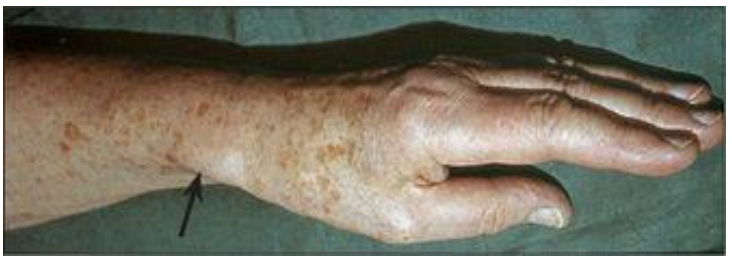


Figure 9.71 A patient with de Quervain's tenosynovitis
There is characteristic swelling of the tendon sheath of the abductor pollicis brevis over the styloid process of the radius.

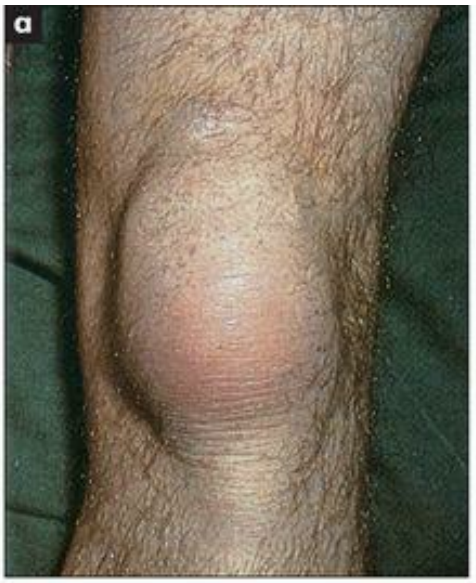
Examination

This reveals tenderness and swelling on the radial side of the wrist (radial styloid). There is pain on active or passive movement of the thumb. Confirm the diagnosis by performing Finkelstein's test.[z] Hold the patient's hand with the thumb tucked into the palm and then quickly turn the wrist into full ulnar deviation (Figure 9.72). An alternative approach that is reported to produce fewer false-positives involves gripping the patient's thumb rather than tucking the thumb into the palm.[13] Sharp pain will occur in the tendon sheath when the test is positive. Examine also the other common sites of tendon involvement—the flexor tendons of the fingers and the Achilles tendon.

Figure 9.72 Finkelstein's test

Bursitis

Bursae are found in areas exposed to mechanical strain or trauma, either at the site where muscle or tendon glides over bone or muscle, or superficially where bony prominences are exposed to mechanical stress. Bursitis usually occurs as a local soft-tissue inflammatory reaction to unusual mechanical pressure. It may be associated with rheumatoid arthritis, gout or sepsis. Common sites include the prepatellar area (housemaid's knee) (Figure 9.73), over the olecranon (olecranon bursitis) and over the greater trochanter (trochanteric bursitis).



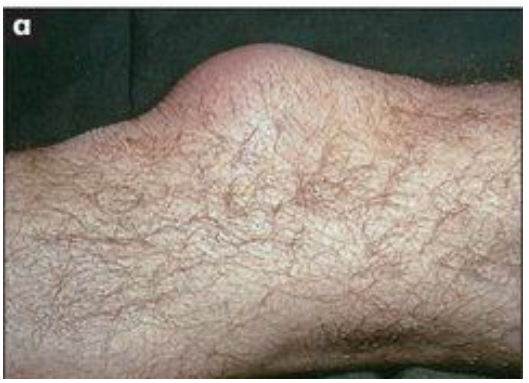


Figure 9.73 A red, swollen and painful prepatellar bursa (a) Anterior view. (b) Lateral view.

Nerve entrapment syndromes

These are caused by compression of peripheral nerves at vulnerable sites and are associated with pain, paraesthesiae and numbness in a particular nerve distribution.

Carpal tunnel syndrome

Compression of the median nerve at the wrist is the most frequent form. This seems hardly surprising when one remembers that the carpal tunnel, sandwiched between the carpal bones and the carpal ligament, contains 9 flexor tendons as well as the median nerve. These patients complain of numbness and paraesthesiae in the median nerve distribution (the 3 radial fingers and the radial side of the ring finger). Symptoms often wake patients from sleep and may radiate up the forearm (one-third of cases). The commonest cause is an overuse tenosynovitis of the flexor tendon sheaths at the wrist. Fluid retention during pregnancy or from use of the oral contraceptive pill can also produce carpal tunnel symptoms. In addition, median nerve compression can occur in rheumatoid arthritis, hypothyroidism, acromegaly and amyloidosis.

Examination

Symptoms can be reproduced by gentle percussion over the carpal tunnel while the wrist is held in extension (Tinel's sign). This sign is negative in up to 30% of patients with electrophysiologically proven median nerve compression. Prolonged (60 seconds) passive wrist flexion (Phalen's test) has a lower false-negative rate (positive LR 1.3, negative LR 0.7).[10] Look for wasting in the median nerve distribution, and loss of motor (thenar muscle strength: weak thumb abduction) and sensory function. These signs occur only in advanced cases.

Meralgia[aa] paraesthetica

Compression of the lateral cutaneous nerve of the thigh causes paraesthesiae

Compression of the lateral cutaneous nerve of the thigh causes paraesthesiae and sensory loss over the lateral side of the thigh (page 375). This entirely sensory nerve passes through the lateral part of the inguinal ligament only just medial to the anterior superior iliac spine. Here it is subject to compression in patients who are obese, wear tight or heavy belts or spend long periods sitting. Diabetes, pregnancy and trauma can also be causes of problems with the nerve.

Tarsal tunnel syndrome

This may be caused by compression of the posterior tibial nerve in its fibro-osseous canal formed by the flexor retinaculum and the tarsal bones. Symptoms include burning pain and paraesthesiae in toe, sole and heel. Patients are often woken with pain at night and, as with the carpal tunnel syndrome, this may radiate upwards. Walking may improve the symptoms. Causes include diabetes, synovitis from rheumatoid arthritis, bony deformity and flexor tenosynovitis. Hypertrophy of the abductor hallucis muscle, which occurs in intemperate runners, is an occasional cause.

Examination

There is usually tenderness over the nerve posterior to the medial malleolus. There may be a positive Tinel's sign over the tarsal tunnel. Motor findings include weakness of toe flexion and of the intrinsic muscles of the foot.

Morton's 'neuroma'

This is caused by compression of one or more of the interdigital plantar nerves by the transverse metatarsal ligament. Patients complain of a burning pain or ache that extends distally from the affected web space to the toes (most often the third and fourth).

Metatarsalgia is a non-localised ache that spreads across the forefoot involving the area of some or all of the metatarsal heads. It can occur in normal feet after prolonged standing but also occurs in a number of other foot conditions (Table 9.19), and is often associated with poor-fitting shoes. *Morton's metatarsalgia* is interdigital nerve entrapment (usually between the third and fourth metatarsal bones. Patients describe burning pain between the metatarsal bones and may have numbness on the adjacent toes. They get relief by removing their shoes and massaging the foot.

TABLE 9.19 Causes of metatarsalgia

Tight or pointed shoes
Atrophy of metatarsal fat pad in elderly people
Plantar calluses
Metatarsophalangeal joint arthritis
Flat or cavus foot deformity
Overlapping toes
Interdigital entrapment
Hemiplegia
Peripheral vascular disease

Examination

There is often tenderness between the involved metatarsal heads, and a painful nodule may be palpable.

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12. Stevens G.L., Adelman H.M., Wallach P.M. Palpable purpura: an algorithmic approach. *Am Fam Phys*. 1995;52:1355-1362.

13. Elliott B.G. Finkelstein's test: a descriptive error that can produce a false-positive. *J Hand Surg Br*. 1992;17:481-482. Careful explanation of the performance of this test (which is often misunderstood) appears in this article. Movement with the thumb folded into the hand can produce a false-positive result

Suggested reading

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Solomon L., Nayagam D., Warwiak D. *Apley's system of orthopaedics and fractures*, 9th edn. London: Butterworth-Heineman; 2009.

[a] Maurice Raynaud (1834–1881) described this in his first work, published in Paris in 1862.

[b] Halushi Behçet (1889–1948), Turkish dermatologist.

[c] The traditional treatment, striking the lesion very hard with the family Bible, is not effective.

[d] Jean Martin Charcot (1825–1893), Parisian physician and neurologist. He became professor of nervous diseases, holding the first Chair of Neurology in the world. His pupils included Babinski, Marie and Freud.

[e] William Heberden (1710–1801), London physician, and doctor to George III and Samuel Johnson, described these in 1802. He was the first person to describe angina.

[f] Charles Jacques Bouchard (1837–1915), Parisian physician.

[g] Fritz de Quervain (1868–1940), professor of surgery in Berne, Switzerland.

[h] George Phalen, orthopaedic surgeon, the Cleveland Clinic.

[i] Jules Tinel (1879–1952), physician and neurologist in Paris. In 1915 he described tingling in the distribution of a nerve that had been severed and was regrowing when it was percussed.

[j] Alan Apley, orthopaedic surgeon, St Thomas's Hospital, London.

[k] Hubert von Luschka (1820--75), professor of anatomy in Tübingen.

[l] Charles E Lasègue (1816–83), Professor of Medicine in Paris and pupil of Trousseau.

[m] Roman goddess of love—her ancient Greek equivalent was Aphrodite.

[n] Hugh Thomas (1834–91), 'the father of orthopaedic surgery', worked in Liverpool as a bone-setter but did not have a hospital appointment.

[o] Friedrich Trendelenburg (1844–1924), professor of surgery at Rostock, Bonn and Leipzig.

[p] Robert Osgood (1873–1956). He worked in France during the First World War and then at the Massachusetts General Hospital where he founded the X-ray department and subsequently developed several radiation-induced skin tumours.arl Schlatter (1864–1934), professor of surgery in Zurich. He pioneered a total gastrectomy operation in 1897.

[q] William Baker (1839–96), surgeon at St Bartholomew's Hospital, London, described this in 1877.

[r] Thomas McMurray (1888–1949), the first professor of orthopaedic surgery in Liverpool.

[s] Hans Reiter (1881–1969), professor of hygiene in Berlin, described the syndrome in 1916. This was well before he became an enthusiastic Nazi.

[t] Thomas Morton (1835–1903), general and eye surgeon, Philadelphia Hospital, performed one of the first appendicectomies.

[u] Franklin Simmonds, orthopaedic surgeon, Rowley Bristow Hospital, Surrey, UK; he is now retired.

[v] Anthony Caplan, Welsh physician, described this in 1953.

[w] A syndesmosis is a joint where the bones are joined by fibrous ligaments or sheets.

[x] Lupus has been used as a name for any erosive disease of the skin; for example, lupus vulgaris is tuberculosis of the skin.

[y] Robert Mosley Yergason, American surgeon born in 1885, described this sign in 1931.

[z] Harry Finkelstein (1865–1939), surgeon, Hospital for Joint Diseases, New York.

[aa] The Greek word *meros* means thigh and *algia* means painful.

Chapter 10

The endocrine system

> A physician is obligated to consider more than a diseased organ, more even than the whole man—he must view the man in his world.
> *Harvey Cushing (1869–1939)*

The endocrine history

Presenting symptoms (Table 10.1)

Hormones control so many aspects of body function that the manifestations of endocrine disease are protean. Symptoms can include changes in body weight, appetite, bowel habit, hair distribution, pigmentation, sweating, height and menstruation, galactorrhoea (unexpected breast-milk production—in men and women), as well as polydipsia, polyuria, lethargy, headaches and loss of libido and erectile dysfunction. Many of these symptoms have other causes as well and must be carefully evaluated. On the other hand, the patient may know which endocrine organ or group of endocrine organs has been causing a problem. In particular, there may be a history of a thyroid condition or diabetes mellitus. A list of common symptoms associated with various endocrine diseases is presented in Table 10.1. In this section some of the important symptoms associated with endocrine disease will be discussed.

TABLE 10.1 Endocrine history

Major symptoms
Appetite and weight changes
Disturbed defecation
Sweating
Hair distribution
Lethargy

Skin changes

Pigmentation

Stature

Loss of libido, erectile dysfunction

Menstruation

Polyuria

Lump in the neck (goitre)

Endocrine abnormalities and typical symptoms and signs

Thyrotoxicosis: preference for cooler weather, weight loss, increased appetite (polyphagia), palpitations, increased sweating, nervousness, irritability, diarrhoea, amenorrhoea, muscle weakness, exertional dyspnoea

Hypothyroidism (myxoedema): preference for warmer weather, lethargy, swelling of eyelids (oedema), hoarse voice, constipation, coarse skin, hypercarotenaemia

Diabetes mellitus: polyuria, polydipsia, thirst, blurred vision, weakness, infections, groin itch, rash (pruritus vulvae, balanitis), weight loss, tiredness, lethargy, disturbance of conscious state

Hypoglycaemia: morning headaches, weight gain, seizures, sweating

Primary adrenal insufficiency: pigmentation, tiredness, loss of weight, anorexia, nausea, diarrhoea, nocturia, mental changes, seizures (hypotension, hypoglycaemia)

Acromegaly: fatigue, weakness, increased sweating, heat intolerance, weight gain, enlarging hands and feet, enlarged and

coarsened facial features, headaches, decreased vision, voice change, decreased libido, erectile dysfunction (impotence)

Cushing's syndrome: truncal obesity, purple striae, moon-like facies, buffalo hump, myopathy, bruises

Changes in appetite and weight

An increased appetite associated with weight loss classically occurs in thyrotoxicosis or uncontrolled diabetes mellitus. An increased appetite with weight gain may occur in Cushing's syndrome, hypoglycaemia or in hypothalamic disease. A loss of appetite with weight loss can occur with adrenal insufficiency but is also seen in anorexia nervosa and with gastrointestinal disease (particularly malignancy). A loss of appetite with weight gain can occur in hypothyroidism.

Changes in bowel habit

Diarrhoea and an increase in the frequency of bowel movements are associated with hyperthyroidism and adrenal insufficiency, while constipation may occur in hypothyroidism and hypercalcaemia.

Changes in sweating

Increased sweating is characteristic of hyperthyroidism, phaeochromocytoma, hypoglycaemia and acromegaly, but may also occur in anxiety states and at the menopause (page 411).

Changes in hair distribution

Hirsutism refers to an increased growth of body hair in women. The clinical evaluation and differential diagnosis are presented on page 315. The absence of facial hair in a man suggests hypogonadism, while temporal recession of the scalp hair in women occurs with androgen excess. The decrease in adrenal androgen production that occurs as a result of hypogonadism, hypopituitarism or adrenal insufficiency can cause loss of axillary and pubic hair in both sexes.

Lethargy

This common symptom can be due to a number of different diseases. Patients with hypothyroidism, Addison's disease and diabetes mellitus can present with this problem. Anaemia, connective tissue diseases, chronic infection (e.g. HIV, infective endocarditis), drugs (e.g. sedatives, diuretics causing electrolyte disturbances), chronic liver disease, renal failure and occult malignancy may also result in lethargy. Importantly, depression is a common cause of this symptom (page 411).

Changes in the skin and nails

The skin becomes coarse, pale and dry in hypothyroidism, and dry and scaly in hypoparathyroidism. Flushing of the skin of the face and neck occurs in the carcinoid syndrome (due to the release of vasoactive peptides from the tumour). Soft-tissue overgrowth occurs in acromegaly and skin tags may appear in the axillae. These are called molluscum fibrinosum. Acanthosis nigricans can also occur in acromegaly and in insulin-resistant states including Cushing's syndrome and polycystic ovarian syndrome. Xanthelasma can be present in patients with diabetes or hypothyroidism.

Onycholysis occurs in Graves' disease and Cushing's syndrome is associated with spontaneous ecchymoses, thin skin and purple striae.

Changes in pigmentation

Increased pigmentation may be reported in primary adrenal insufficiency, Cushing's syndrome or acromegaly. Decreased pigmentation occurs in hypopituitarism. Localised depigmentation is characteristic of vitiligo, which may be associated with certain endocrine diseases such as Hashimoto's[a] disease with hypothyroidism and Addison's disease with adrenal insufficiency as well as other auto-immune conditions.

Changes in stature

Tallness may occur in children for constitutional reasons (tall parents) or, rarely, may reflect growth hormone excess (leading to gigantism), gonadotrophin deficiency, Klinefelter's syndrome[b], Marfan's syndrome or generalised lipodystrophy. Short stature can also result from endocrine disease, as discussed on page 313.

Erectile dysfunction (impotence)

A persistent inability to attain or sustain penile erections may occasionally be due to primary hypogonadism or to secondary hypogonadism due to hyperprolactinaemia or hypopituitarism. More often, it is related to emotional disorders. Vascular disease, autonomic neuropathy (e.g. in diabetes mellitus or alcoholism), spinal cord disease or testicular atrophy can also cause this problem.

Galactorrhoea

Hyperprolactinaemia (usually the result of pituitary adenoma) can cause galactorrhoea in up to 80% of women and 30% of men. Galactorrhoea in men occurs from a normal-appearing male breast.

Menstruation

Failure to menstruate is termed *amenorrhoea*.

Primary amenorrhoea is defined as a failure to start menstruating by 17 years of age. True primary amenorrhoea may result from ovarian failure (e.g. X chromosomal abnormalities such as Turner's syndrome) or from pituitary or hypothalamic disease (e.g. tumour, trauma or idiopathic disease). Excess androgen production or systemic disease (e.g. malabsorption, chronic renal failure, obesity) can also result in primary amenorrhoea.

Apparent primary amenorrhoea can also occur if menstrual flow cannot escape: for example, if there is an imperforate hymen.

Secondary amenorrhoea is defined as the cessation of menstruation for 6 months or more. Pregnancy and menopause are common causes. The polycystic ovarian syndrome, hyperprolactinaemia, virilising syndromes or hypothalamic or pituitary disease can also result in this problem, as can use of the contraceptive pill or psychiatric disease.

Polyuria

Polyuria is defined as a urine volume of more than 3 litres/day. Patients who report urinary frequency may find it difficult to tell if large volumes of urine are being passed. Causes include diabetes mellitus (due to excessive filtration of glucose, a poorly resorbed solute); diabetes insipidus (due to inadequate renal water conservation from a central deficiency of antidiuretic hormone, or a lack of renal responsiveness to this hormone); primary polydipsia, where a

patient drinks excessive water (due to psychogenic or hypothalamic disease or drugs such as chlorpromazine or thioridazine); hypercalcaemia; and tubulointerstitial or cystic renal disease.

Past history

A previous history of any endocrine condition must be uncovered. This includes surgery on the neck for a goitre. A partial thyroidectomy or radio-iodine (^{131}I) treatment in the past can lead to eventual hypothyroidism. The same may apply to radiation of the thyroid for carcinoma. A woman may have been diagnosed with diabetes mellitus after the birth of a large baby. There may be a past history of hypertension, which is occasionally due to an endocrine condition (e.g. phaeochromocytoma, Cushing's syndrome or Conn's syndrome). Previous thyroid surgery can be associated with hypoparathyroidism because of surgical damage to the parathyroid glands.

Previous treatment of a patient's thyroid problems may have included the use of antithyroid drugs, thyroid hormone or radioactive iodine. Surgery on the adrenals or pituitary may have been performed and this may leave the patient with decreased adrenal or pituitary function.

Patients with diabetes mellitus have an important chronic condition (*Questions box 10.7*, page 316). Treatment may be with diet, insulin or oral hypoglycaemic agents. One must determine how well the patient understands the condition, and whether he or she understands the principles of the diabetic diet and adheres to it. Find out how the blood sugar levels are monitored and whether or not the patient adjusts the insulin dose. Most patients should now be able to monitor their own blood sugar levels at home using a glucometer. There is now good evidence that tight control of blood sugar levels reduces the incidence of diabetic complications. Patients should have records of home blood sugar measurements, and may know the results of tests such as the haemoglobin A1c (a measure of average blood sugar levels) and of tests of renal function and for protein in the urine.

The patient should be aware of the need for care of the feet and eyes to help prevent complications. Most diabetics have regular ophthalmological review, often using retinal photography. There may be a history of laser treatment for proliferative diabetic retinopathy.

Patients with hypopituitarism or hypoadrenalism may be on glucocorticoid (steroid) replacement; the latter also require mineralocorticoid replacement. Details of the patient's dosage schedule should be obtained.

Social history

Many of these conditions are chronic and their complications serious. How well the patient copes with various problems and the conditions at home and work will have an important effect on the success of treatment.

Family history

There may be a history in the family of thyroid conditions or diabetes mellitus. Occasionally a family history of a multiple endocrine neoplasia (MEN) syndrome may be obtained. These are rare autosomal-dominant conditions. They include pituitary tumours, medullary carcinoma of the thyroid, hyperparathyroidism, phaeochromocytoma and pancreatic islet cell tumours.

The endocrine examination

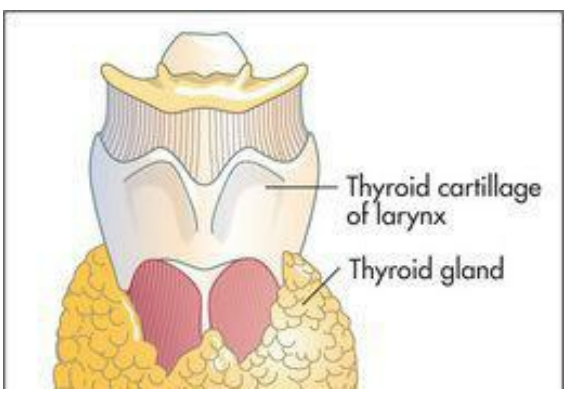
A formal examination of the whole endocrine system is set out on page 322. Usually there will be some clue from the history and general inspection to indicate what specific endocrine diseases should be pursued.

The thyroid

The thyroid gland[c]

Examination anatomy

Even when it is not enlarged, the thyroid (Figure 10.2) is the largest



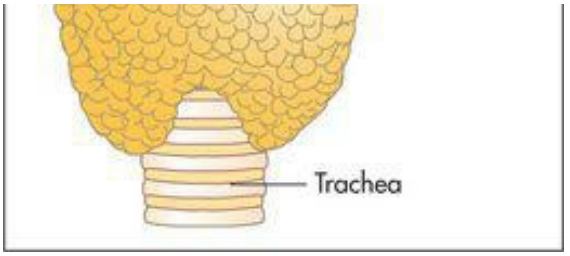


Figure 10.2 The anatomy of the thyroid

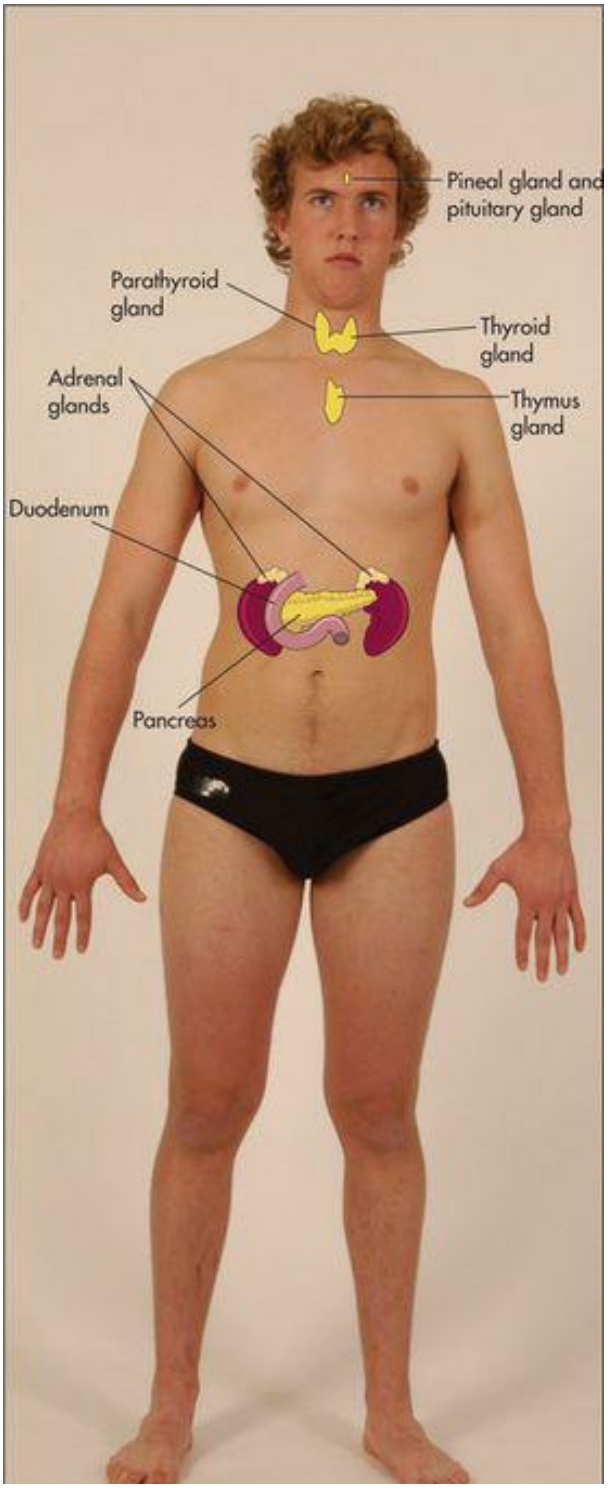


Figure 10.1 The endocrine glands

endocrine gland. Enlargement is common, occurring in 10% of women and 2% of men and more commonly in iodine-deficient parts of the world. The normal gland lies anterior to the larynx and trachea and below the laryngeal prominence of the thyroid cartilage. It consists of a narrow isthmus in the middle line (anterior to the second to fourth tracheal rings and 1.5 cm in size), and two larger lateral lobes each about 4 cm long. Although the position of the larynx varies, the thyroid gland is almost always about 4 cm below the larynx.

Inspection

The normal thyroid may be just visible below the cricoid cartilage in a thin young person (Table 10.2).[1,2]. Usually only the isthmus is visible as a diffuse central swelling. Enlargement of the gland, called a goitre (Latin *guttur*, 'throat'), should be apparent on inspection (see *Good signs guide 10.1*), especially if the patient extends the neck. Look at the front and sides of the neck and decide whether there is localised or general swelling of the gland. In normal people the line between the cricoid cartilage and the suprasternal notch should be straight. An outward bulge suggests the presence of a goitre (Figure 10.3). Remember that 80% of people with a goitre are biochemically euthyroid, 10% are hypothyroid and 10% are hyperthyroid.

TABLE 10.2 Causes of neck swellings

Midline
Goitre (moves up on swallowing)
Thyroglossal cyst (moves on poking out the tongue with the jaw stationary)
Submental lymph nodes

Lateral
Lymph nodes*
Salivary glands (e.g. stone, tumour) • Submandibular gland • Parotid gland (lower pole)
Skin: sebaceous cyst or lipoma
Lymphatics: cystic hygroma (translucent)
Carotid artery: aneurysm or rarely tumour (pulsatile)
Pharynx: pharyngeal pouch, or brachial arch remnant (brachial cyst)
Parathyroid gland (very rare)

* Aulus Celsus (page 297), the Roman medical writer who was active early in the 1st century AD, was the first to publish work distinguishing a goitre from cervical lymphadenopathy.

GOOD SIGNS GUIDE 10.1 Detection of a goitre (compared with ultrasound findings)

Sign	Positive LR	Negative LR*

No goitre on inspection or palpation	0.4	—
Goitre palpated and visible only on neck extension	NS	—
Goitre palpated and visible with neck in normal position	26.3	—

NAS = not significant.

* No values available.

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

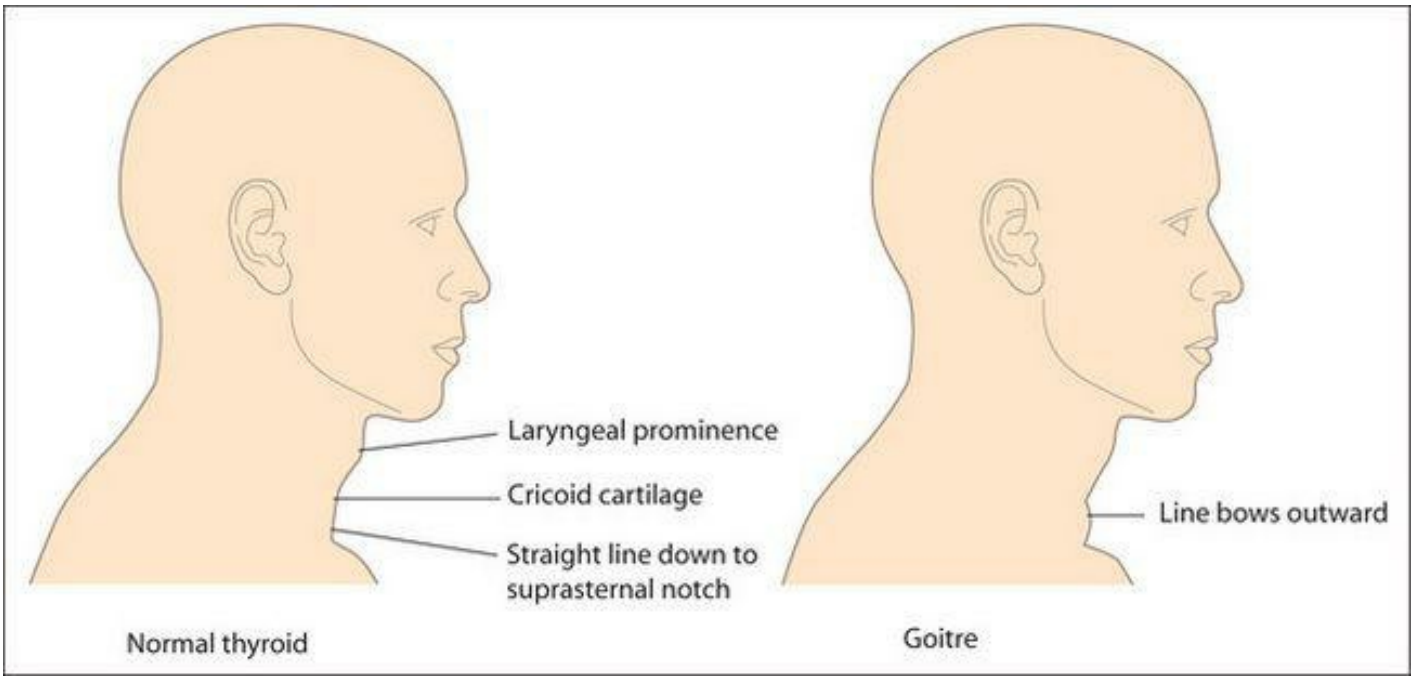


Figure 10.3 The thyroid and goitre

Adapted from McGee S, Evidence-based physical diagnosis, 2nd edition, St Louis, Saunders, 2007.

The temptation to begin touching a swelling as soon as it has been detected should be resisted until a glass of water has been procured. The patient takes sips from this repeatedly so that swallowing is possible without discomfort. Ask the patient to swallow, and watch the neck swelling carefully. Only a goitre or a thyroglossal cyst, because of attachment to the larynx, will rise during swallowing. The thyroid and trachea rise about 2 cm as the patient swallows; they pause for half a second and then descend. Some non-thyroid masses may rise slightly during swallowing but move up less than the trachea and fall again without pausing. A thyroid gland fixed by

neoplastic infiltration may not rise on swallowing, but this is rare. Swallowing also allows the shape of the gland to be seen better.

It should be noted whether an inferior border is visible as the gland rises. The thyroglossal cyst is a midline mass that can present at any age. It is an embryological remnant of the thyroglossal duct. Characteristically it rises when the patient protrudes the tongue.

Inspect the skin of the neck for scars. A thyroidectomy scar forms a ring around the base of the neck in the position of a high necklace. Also look for prominent veins. Dilated veins over the upper part of the chest wall, often accompanied by filling of the external jugular vein, suggest retrosternal extension of the goitre (thoracic inlet obstruction). Rarely, redness of the skin over the gland occurs in cases of suppurative thyroiditis.

Palpation

Palpation is best begun from *behind* (Figure 10.4) but warn the patient. Both hands are placed with the pulps of the fingers over the gland. The patient's neck should be slightly flexed so as to relax the sternomastoid muscles. Feel systematically both lobes of the gland and its isthmus.

Figure 10.4 Palpating the thyroid from behind while the patient swallows sips of water

Consider the following:

• **Size:** only an approximate estimation is possible (Figure 10.5). Feel particularly carefully for a lower border, because its absence suggests

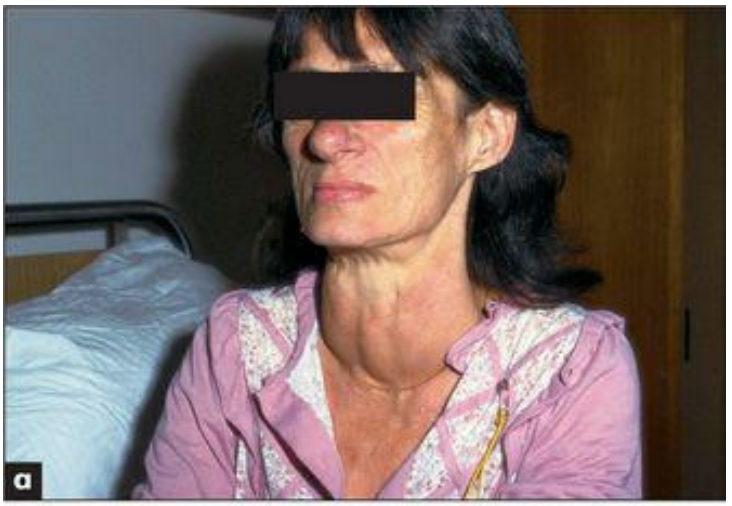
retrosternal extension.

• **Shape:** note whether the gland is uniformly enlarged or irregular and whether the isthmus is affected. If a *nodule* that feels distinct from the remaining thyroid tissue is palpable, determine its location, size, consistency, tenderness and mobility. Also decide whether the whole gland feels nodular (multinodular goitre).

• **Consistency:** may vary in different parts of the gland. Soft is normal; the gland is often firm in simple goitre and typically rubbery hard in Hashimoto's thyroiditis. A stony, hard node suggests carcinoma (Table 10.3), calcification in a cyst, fibrosis or Riedel's thyroiditis.

• **Tenderness:** a feature of thyroiditis (subacute or rarely suppurative), or less often of a bleed into a cyst or carcinoma.

• **Mobility:** carcinoma may tether the gland.



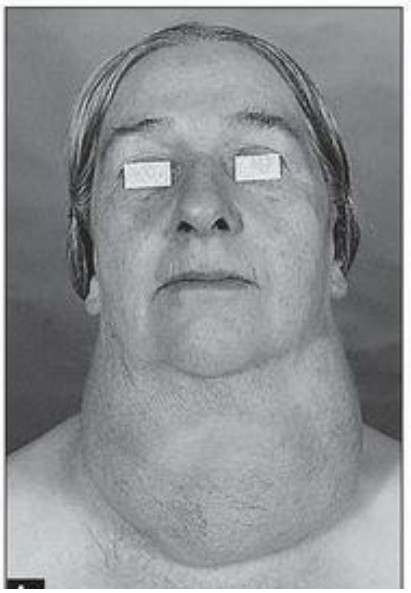


Figure 10.5 Goitre: (a) large; (b) massive

TABLE 10.3 Differential diagnosis of thyroid nodules

1 Carcinoma (5% of palpable nodules)—fixed to surrounding tissues, palpable lymph nodes, vocal cord paralysis, hard, larger than 4 cm (most are, however, smaller than this)
2 Adenoma—mobile, no local associated features
3 Big nodule in a multinodular goitre—palpable multinodular goitre

Repeat the assessment while the patient swallows

Decide if a thrill is palpable over the gland, as occurs when the gland is unusually metabolically active as in thyrotoxicosis.

Palpate the cervical lymph nodes (page 228). These may be involved in carcinoma of the thyroid.

Move to the *front*. Palpate again. Localised swellings may be more easily defined here. Note the position of the trachea, which may be displaced by a retrosternal gland.

Percussion

The upper part of the manubrium can be percussed from one side to the other. A change from resonant to dull indicates a possible retrosternal goitre, but this is not a very reliable sign.

Auscultation

Listen over each lobe for a bruit (a swishing sound coinciding with systole). This is a sign of increased blood supply which may occur in hyperthyroidism, or occasionally from the use of antithyroid drugs. The differential diagnosis

of occasionally from the use of antithyroid drugs. The differential diagnosis also includes a carotid bruit (louder over the carotid itself) or a venous hum (obliterated by gentle pressure over the base of the neck). If there is a goitre, apply mild compression to the lateral lobes and listen again for stridor.

Pemberton's sign

Ask the patient to lift both arms as high as possible. Wait a few moments, then search the face eagerly for signs of congestion (plethora) and cyanosis. Associated respiratory distress and inspiratory stridor may occur. Look at the neck veins for distension (venous congestion). Ask the patient to take a deep breath in through the mouth and listen for stridor. This is a test for thoracic inlet obstruction due to a retrosternal goitre or any retrosternal mass.[3] (Lifting the arms up pulls the thoracic inlet upward so that the goitre occupies more of this inflexible bony opening.)

Examination of the thyroid should be part of every routine physical examination. Causes of a goitre are listed in Table 10.4.

TABLE 10.4 Goitre

Causes of a diffuse goitre (patient often euthyroid)
Idiopathic (majority)
Puberty or pregnancy
Thyroiditis • Hashimoto's • Subacute (gland usually tender)
Simple goitre (iodine deficiency)
Goitrogens—iodine excess, drugs (e.g. lithium)
Inborn errors of thyroid hormone synthesis—e.g. Pendred's* syndrome (an autosomal-recessive condition associated with nerve deafness)

Causes of a solitary thyroid nodule

Benign:

- Dominant nodule in a multinodular goitre
- Degeneration or haemorrhage into a colloid cyst or nodule
- Follicular adenoma
- Simple cyst (rare)

Malignant:

- Carcinoma—primary or secondary (rare)
- Lymphoma (rare)

* Vaughan Pendred (b. 1869), London physician.

Hyperthyroidism (thyrotoxicosis)

This is a disease caused by excessive concentrations of thyroid hormones. The cause is usually overproduction by the gland but may sometimes be due to accidental or deliberate use of thyroid hormone (thyroxine) tablets; *thyrotoxicosis factitia*. Thyroxine is sometimes taken by patients as a way of losing weight. The cause may be apparent in these cases if a careful history is taken (*Questions box 10.1*). The anti-arrhythmic drug *amiodarone* which contains large quantities of iodine can cause thyrotoxicosis in up to 12% of patients in low-iodine-intake areas.

Questions box 10.1

Questions to ask the patient with suspected hyperthyroidism

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

1. Have you any history of thyroid problems?

2. Have you a family history of thyrotoxicosis?—There is a familial incidence of Graves disease and associated auto-immune conditions such as vitiligo, Addison's disease, pernicious anaemia, type 1 diabetes, myasthenia gravis and premature ovarian failure

3. Have you taken amiodarone or thyroxine?

4. Have you had recent exposure to iodine?—Iodinated X-ray contrast materials can precipitate thyrotoxicosis (usually in patients with an existing multinodular goitre)

5. Have you had palpitations?—Thyrotoxicosis can present with atrial fibrillation which may precipitate heart failure

6. Have you noticed insomnia, irritability or hyperactivity?

7. Have you had loss of weight, diarrhoea or increased stool frequency, increased sweating or heat intolerance?

8. Have you had muscle weakness?—Proximal muscle weakness is common and the patient may have noticed difficulty getting out of a chair

9. Have you had eye problems such as double vision, grittiness, redness or pain behind the eyes?

Many of the clinical features of thyrotoxicosis are characterised by signs of sympathetic nervous system overactivity such as tremor, tachycardia and sweating. The explanation is not entirely clear. Catecholamine secretion is usually normal in hyperthyroidism; however, thyroid hormone potentiates the effects of catecholamines, possibly by increasing the number of adrenergic receptors in the tissues.

The commonest cause of thyrotoxicosis in young people is Graves' disease,[d] an autoimmune disease where circulating immunoglobulins stimulate thyroid stimulating hormone (TSH) receptors on the surface of the thyroid follicular cells.

Examine a suspected case of thyrotoxicosis as follows (see *Good signs guide 10.2*).

GOOD SIGNS GUIDE 10.2 Thyrotoxicosis

Sign	Positive LR	Negative LR
Pulse		
≥90/min	4.4	0.2
Skin		
Moist and warm	6.7	0.7
Thyroid		
Enlarged	2.3	0.1
Eyes		
Eyelid retraction	31.5	0.7
Lid lag	17.6	0.8
Neurological		
Fine tremor	11.4	0.3

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

General inspection

Look for signs of weight loss, anxiety and the frightened facies of thyrotoxicosis.

The hands

Ask the patient to put out his or her arms and look for a fine tremor (due to sympathetic overactivity). Laying a sheet of paper over the patient's fingers may more clearly demonstrate this tremor, to the amazement of less-experienced colleagues.

Look at the nails for *onycholysis* (Plummer's[e] nails) (Figure 10.6). Onycholysis (where there is separation of the nail from its bed) is said to occur particularly on the ring finger, but can occur on all the fingernails, and is apparently due to sympathetic overactivity. Inspect now for thyroid acropathy (acropathy is another term for clubbing), seen rarely in Graves' disease but not with other causes of thyrotoxicosis.

Figure 10.6 Onycholysis (Plummer's nails)

Inspect for palmar erythema and feel the palms for warmth and sweatiness (sympathetic overactivity).

Take the pulse. Note the presence of sinus tachycardia (sympathetic overdrive) or atrial fibrillation (due to a shortened refractory period of atrial cells related to sympathetic drive and hormone-induced changes). The pulse may also have a collapsing character due to a high cardiac output.

Test for proximal myopathy and tap the arm reflexes for abnormal briskness, especially in the relaxation phase.

The eyes

Examine the eyes for exophthalmos, which is protrusion of the eyeball from the orbit (Figure 10.7, Table 10.5). This may be very obvious, but if not, look carefully at the sclerae, which in exophthalmos are not covered by the