General signs of acute leukaemia

Pallor (anaemia), fever (which usually indicates infection secondary to neutropenia) and petechiae (thrombocytopenia) are all due to bone marrow failure. Weight loss, muscle wasting (hypercatabolic state) and localised infections (e.g. of the tonsils or perirectal region, due to leucopenia) also occur.

Signs of infiltration of the haemopoietic system

These include: (i) bony tenderness, due to infiltra or infarction; (ii) lymphadenopathy (slight to moderate, especially in acute lymphoblastic leuka); (iii) splenomegaly (slight to moderate, occurs especially in acute lymphoblastic leukaemia; the spleen may be tender due to splenic infarction); and (iv) hepatomegaly (slight to moderate).

Signs of infiltration of other areas

There may be: (i) tonsillar enlargement (especially in acute lymphoblastic leukaemia); (ii) swelling or bleeding of the gums, especially in monocytic leukaemia; (iii) pleural effusions; (iv) nerve palsies, involving the spinal nerve roots or the cranial nerves; or (v) meningism due to infiltration of the meninges, especially in acute lymphoblastic leukaemia.

Chronic leukaemia

This is a haematological malignancy in which the leukaemic cell is at first well differentiated. These have a better prognosis untreated than acute leukaemia. There are two main types: chronic myeloid leukaemia and chronic lymphocytic leukaemia.

Signs of chronic myeloid leukaemia

This is one of the myeloproliferative disorders. There is an expanded granulocytic mass in the bone marrow, liver and spleen.

General signs may include pallor (anaemia due to bone marrow infiltration) and secondary gout (common).

Haemopoietic system signs include massive splenomegaly and moderate hepatomegaly. (*Note:* Lymphadenopathy is usually a sign of blast

moderate hepatomegaly. (*Note:* Lymphadenopathy is usually a sign of blast transformation.)

Signs of chronic lymphocytic leukaemia

There may be tiredness and pallor. Recurrent acute infections occur.

Haemopoietic system signs include marked or moderate lymphadenopathy and moderate hepatosplenomegaly.

Other abnormalities may include a Coombs'[h] test—positive haemolytic anaemia, herpes zoster skin infections and nodular infiltrates. Patients may note a hypersensitivity to insect bites before the diagnosis is made.

Myeloproliferative disease

This is a group of disorders of the haematopoietic stem cell. These include polycythaemia rubra vera, primary myelofibrosis, chronic myeloid leukaemia and essential thrombocythaemia. Overlapping clinical and pathological features occur in these disorders. Therefore, patients may have signs of one or more of the conditions. Any of them may progress to acute myeloid leukaemia.

Polycythaemia

This is an elevated haemoglobin concentration and can result from an increased red blood cell mass or a decreased plasma volume. Polycythaemia rubra vera results from an autonomous increase in the red blood cell production. Patients with polycythaemia often have a striking ruddy, plethoric appearance. To examine a patient with suspected polycythaemia, assess for both the manifestations of polycythaemia rubra vera and for other possible underlying causes of polycythaemia (Table 8.11).

TABLE 8.11 Polycythaemia

Signs of polycythaemia rubra vera
Plethoric appearance including engorged conjunctival and retinal vessels (not specific)

Scratch marks (generalised pruritus)

Splenomegaly (80%)

Bleeding tendency (platelet dysfunction)

Peripheral vascular and ischaemic heart disease (thrombosis, slow circulation)

Gout

Mild hypertension

Causes of polycythaemia

Absolute polycythaemia (increased red cell mass)

Idiopathic: polycythaemia rubra vera

Secondary polycythaemia

- Increased erythropoietin:
  - renal disease—polycystic disease, hydronephrosis, tumour; after renal transplantation
  - hepatocellular carcinoma
  - cerebellar haemangioblastoma
  - uterine fibroma
  - virilising syndromes
  - Cushing's syndrome
  - phaeochromocytoma
- Hypoxic states (erythropoietin secondarily increased):
  - chronic lung disease
  - sleep apnoea
  - living at high altitude
  - cyanotic congenital heart disease
  - abnormal haemoglobins
  - carbon monoxide poisoning

Relative polycythaemia (decreased plasma volume)

Dehydration

Stress polycythaemia: Gaisböck's* disease

* Felix Gaisböck (1868–1955), German physician, described this in 1905.

Look at the patient and estimate the state of hydration (dehydration alone can cause an elevated haemoglobin due to haemoconcentration). Note if there is a Cushingoid (page 309) or virilised (page 315) appearance. Cyanosis may be present because of an underlying condition such as cyanotic congenital heart disease or chronic lung disease. Look for nicotine staining (smoking). All these diseases can result in secondary polycythaemia.

The arms should be inspected for scratch marks; post-bathing pruritus occurs in polycythaemia rubra vera, possibly due to basophil histamine release. Take the blood pressure: very rarely a phaeochromocytoma will cause secondary polycythaemia and hypertension.

Examine the eyes. Look for injected conjunctivae. Fundal hyperviscosity changes, including engorged, dilated retinal veins and haemorrhages, may be present. Inspect the tongue for central cyanosis.

Examine the cardiovascular system for signs of cyanotic congenital heart disease and the respiratory system for signs of chronic lung disease. The abdomen must be carefully assessed for splenomegaly, which occurs in 80% of cases of polycythaemia rubra vera but does not usually occur with the other causes of polycythaemia. There may be evidence of chronic liver disease or hepatocellular carcinoma, which may cause secondary polycythaemia. Palpate for the kidneys and perform a urinalysis. In women palpate the uterus. Polycystic kidney disease, hydronephrosis, renal carcinoma and uterine fibromata can all rarely cause secondary polycythaemia.

The legs must be inspected for scratch marks, gouty tophi (Figure 9.57, page 282) and arthropathy, as well as for signs of peripheral vascular disease. In polycythaemia rubra vera, secondary gout occurs due to the increased cellular turnover resulting in hyperuricaemia. Peripheral vascular disease occurs in polycythaemia rubra vera because of thrombosis (as there is increased platelet adhesiveness and accelerated atherosclerosis) and slowed circulation due to hyperviscosity.

Look for cerebellar signs, which may be due to the presence of a cerebellar haemangioblastoma, a very rare cause of secondary polycythaemia. Examine the central nervous system for signs of a stroke due to thrombosis.

Primary myelofibrosis

This is a clonal haemopoietic stem cell disorder with fibrosis as a secondary phenomenon. Gradual replacement of the marrow by fibrosis and progressive splenomegaly characterise the disease.

• **General signs** include pallor (anaemia occurs in most patients eventually) and petechiae (in 20% of patients, due to thrombocytopenia).

• **Haemopoietic system signs** include splenomegaly (in almost all cases, and often to a massive degree—there may also be a splenic rub due to splenic infarction), hepatomegaly (occurs in 50% of patients and can be massive) and lymphadenopathy (very uncommon).

• **Other signs** are bony tenderness (uncommon) and gout (occurs in 5% of patients).

Chronic myeloid leukaemia

(See page 236.)

Essential thrombocythaemia

This is a sustained elevation of the platelet count above normal without any primary cause.

• **General signs** include spontaneous bleeding and thrombosis.

• **Haemopoietic system signs** include splenomegaly.

• **Causes of thrombocytosis** (platelet count more than 450×10^9/L) include: (i) following haemorrhage or surgery; (ii) postsplenectomy; (iii) iron deficiency; (iv) chronic inflammatory disease; (v) malignancy.

• **Causes of thrombocytosis** (platelet count more than 800×10^9/L) include: (i) myeloproliferative disease; (ii) secondary to recent splenectomy, malignancy, or marked inflammation occasionally.

Lymphoma (Figure 8.23)

This is a malignant disease of the lymphoid system. There are two main

clinicopathological types: Hodgkin's disease (with the characteristic Reed-Sternberg cell[i]) and non-Hodgkin's lymphoma. Signs of lymphoma depend on the stages of the disease (Table 8.12).

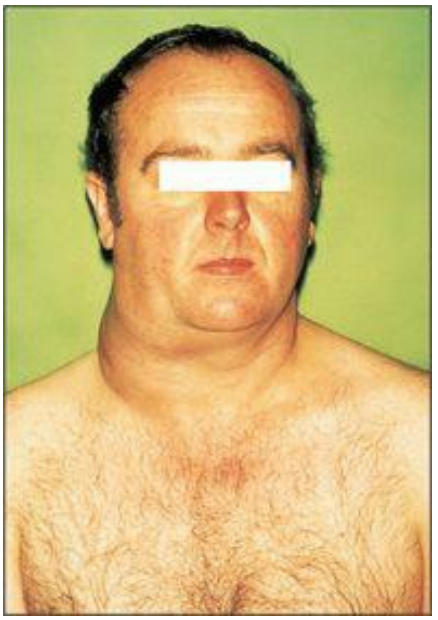

Figure 8.23 Cervical lymph node enlargement in a patient with lymphoma

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

TABLE 8.12 Staging of lymphoma: Ann Arbor classification

Stage I
Disease confined to a single lymph node region or a single extralymphatic site (IE)
Stage II
Disease confined to two or more lymph node regions on one side of the diaphragm

Stage III
Disease confined to lymph nodes on both sides of the diaphragm with or without localised involvement of the spleen (IIIS), other extralymphatic organ or site (IIIE), or both (IIIES)
Stage IV
Diffuse disease of one or more extralymphatic organs (with or without lymph node disease)
For any stage: a = no symptoms; b = fever, weight loss greater than 10% in 6 months, night sweats.
E involves direct invasion from lymph node into surrounding tissue.

Hodgkin's disease often presents in stage I or II, while non-Hodgkin's lymphoma usually presents in stage III or IV.

Signs of Hodgkin's disease

1. Lymph node enlargement: discrete, rubbery, painless, large and superficial nodes, often confined to one side and one lymph node group.

2. Weight loss and fever with or without infection (reduced cell-mediated immunity) suggest a poor prognosis.

3. Splenomegaly and hepatomegaly. Splenomegaly does not always indicate extensive disease.

4. Organ infiltration occurs with late disease. Look especially for signs of: (i) lung disease, such as a pleural effusion; (ii) bone pain or pathological fractures (rare); (iii) spinal cord or nerve compression (rare); and (iv) nodular skin infiltrates (rare).

Signs of non-Hodgkin's lymphoma

1. Lymph node enlargement: often more than one site is involved and Waldeyer's ring is more commonly affected.

2. Hepatosplenomegaly is common.

3. Systemic signs (for example weight loss or fever) are less common.

4. Signs of extranodal spread are more common.

5. The disease may sometimes arise at an extranodal site (e.g. the gastrointestinal tract).

Multiple myeloma

This is a disseminated malignant disease of plasma cells.

General signs

There may be signs of anaemia (due to bone marrow infiltration or as a result of renal failure), purpura (due to bone marrow infiltration and thrombocytopenia) or infection (particularly pneumonia).

Bony tenderness and pathological fractures may be present. Weight loss may be a feature.

Skin changes include hypertrichosis, erythema annulare, yellow skin and secondary amyloid deposits.

There may be signs of spinal cord compression, or mental changes (due to hypercalcaemia).

Look for signs of chronic renal failure (which may be due to tubular damage from filtered light chains, uric acid nephropathy, hypercalcaemia, urinary tract infection, secondary amyloidosis or plasma cell infiltration).

Summary

The haematological examination: a suggested method (Figure 8.24)

This will be a targeted examination during follow-up consultations but should be completed in full for the first visit.

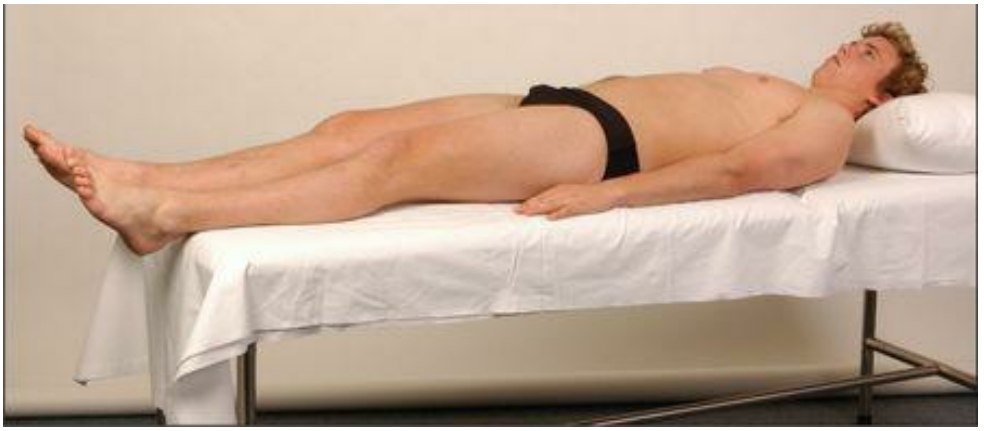

Figure 8.24 Haematological system examination
Lying flat (1 pillow)

1. **General inspection**
   Weight (normal, reduced, increased)
   Bruising (thrombocytopenia, scurvy etc)
   - Petechiae (pinhead bleeding)
   - Ecchymoses (large bruises)

   Pigmentation (lymphoma)
   Rashes and infiltrative lesions (lymphoma)
   Ulceration (neutropenia)
   Cyanosis (polycythaemia)
   Plethora (polycythaemia)
   Jaundice (haemolysis)
   Scratch marks (myeloproliferative diseases, lymphoma)
   Racial origin

2. **Hands**
   Nails—koilonychia, pallor
   Palmar crease pallor (anaemia)
   Arthropathy (haemophilia, secondary gout, drug treatment etc)
   Pulse

3. **Epitrochlear and axillary nodes**

4. **Face**
   Sclera—jaundice, pallor, conjunctival suffusion (polycythaemia)
   Mouth—gum hypertrophy (monocytic leukaemia etc.), ulceration, infection, haemorrhage (marrow aplasia etc.); atrophic glossitis, angular stomatitis (iron, vitamin deficiencies)
   Tongue—amyloidosis

5. **Cervical nodes (sitting up)**
   Palpate from behind

6. **Bony tenderness**
   Spine
   Sternum
   Clavicles
   Shoulders

7. **Abdomen (lying flat) and genitalia**
   Inguinal nodes
   Detailed examination

8. **Legs**
   Vasculitis (Henoch-Schönlein purpura—buttocks, thighs)
   Bruising
   Pigmentation
   Ulceration (e.g. haemoglobinopathies)
   Neurological signs (subacute combined degeneration, peripheral neuropathy)

9. **Other**
   Fundi (haemorrhages, infection etc)
   Temperature chart (infection)
   Urine analysis (haematuria, bile etc)
   Rectal and pelvic examination (blood loss)

Position the patient as for a gastrointestinal examination. Make sure he or she is fully undressed, in stages and with a gown for women. Look for bruising, pigmentation, cyanosis, jaundice and scratch marks (due to myeloproliferative disease or lymphoma). Also note the presence of frontal bossing and the racial origin of the patient.

Pick up the patient's **hands**. Look at the nails for koilonychia (spoon-shaped nails, which are rarely seen today and indicate iron deficiency) and the changes of vasculitis. Pale palmar creases may indicate anaemia (typically the haemoglobin level has to be lower than 70 g/L). Evidence of arthropathy may be important (e.g. rheumatoid arthritis and Felty's syndrome, recurrent haemarthroses in bleeding disorders, secondary gout in myeloproliferative disorders).

Examine the **epitrochlear nodes**. Note any bruising. Remember, petechiae are pinhead haemorrhages, while ecchymoses are larger bruises.

Go to the **axillae** and palpate the axillary nodes. There are five main areas: central, lateral (above and lateral), pectoral (most medial), infraclavicular and subscapular (most inferior).

Look at the **face**. Inspecting the eyes, note jaundice, pallor or haemorrhage of the sclerae, and the injected sclerae of polycythaemia. Examine the mouth. Look for peri-oral telangiectasiae. Note gum hypertrophy (e.g. from acute monocytic leukaemia or scurvy), ulceration, infection, haemorrhage, atrophic glossitis (e.g. from iron deficiency, or vitamin B_{12} or folate deficiency) and angular stomatitis. Look for tonsillar and adenoid enlargement (Waldeyer's ring).

Sit the patient up. Examine the **cervical nodes** from behind. There are eight groups: submental, submandibular, jugular chain, supraclavicular, posterior triangle, postauricular, preauricular and occipital. Then feel the supraclavicular area from the front. Tap the spine with your fist for **bony tenderness** (caused by an enlarging marrow—e.g. in myeloma or carcinoma). Also gently press the sternum, clavicles and shoulders for bony tenderness.

Lay the patient flat again. Examine the **abdomen**. Focus on the liver and spleen. Feel for para-aortic nodes. Don't forget to feel the testes, and to perform a rectal and pelvic examination (for tumour or bleeding). Spring the hips for pelvic tenderness. Palpate the inguinal nodes. There are two groups —along the inguinal ligament and along the femoral vessels.

Examine the **legs**. Note particularly leg ulcers. Examine the legs from a neurological aspect, for evidence of vitamin B_{12} deficiency or peripheral neuropathy from other causes. Remember, hypothyroidism can cause anaemia and neurological disease.

Finally, examine the **fundi**, look at the **tempera** chart, and test the **urine**.

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3. Sheth TN, Choudray NK, Bowes M, Detsky AS. The relation of conjunctival pallor to the presence of anemia. *J Gen Intern Med*. 1997;12:102-106. The presence of conjunctival pallor is a useful indicator of anaemia, but its absence is unhelpful. It is also a reliable sign

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Suggested reading

Hoffbrand A.V., Moss P., Pettit J.E. *Essential haematology*, 5th edn. London: Blackwell; 2006.

Provan D., editor. *ABC of clinical haematology*, 3rd edn., London: BMJ Publishing Group, 2007.

[a] EA von Willebrand (1870–1949), Swedish physician, described this in 1926.

[b] Augustus Roi Felty (1895–1963), physician, Hartford Hospital, Connecticut, described this in 1924.

[c] Alfred Hess (1875–1933), professor of paediatrics, New York, described this in 1914.

[d] This test is only of historical interest these days, as a platelet count can be obtained almost as quickly in most hospitals and clinics. A blood pressure cuff, placed over the upper arm, is inflated to a point 10 mmHg above the diastolic blood pressure. Wait for 5 minutes, then deflate the cuff and wait for another 5 minutes before inspecting the arm. Look for petechiae, which are usually most prominent in the cubital fossa and near the wrist, where the skin is most lax. Fewer than 5 petechiae per cm^2 is normal, while more than 20 is definitely abnormal, suggesting thrombocytopenia, abnormal platelet function or capillary fragility.

[e] Thomas Hodgkin (1798–1866), famous student at Guy's Hospital, London, described his disease in 1832. The first case he described was a patient of Richard Bright's. Hodgkin was one of the first to use the stethoscope in England. On failing to be appointed a physician, he gave up medicine and became a missionary.

[f] Heinrich Wilhelm Gottfried von Waldeyer-Hartz (1836–1921), Berlin anatomist.

[g] Henoch-Schönlein purpura is also characterised by glomerulonephritis (manifested by haematuria and proteinuria), arthralgias and abdominal pain.

[h] Robin Coombs (b. 1921), Quick professor of biology, Cambridge.

[i] Dorothy Reed (1874–1964), pathologist at Johns Hopkins Hospital, Baltimore, described these cells in 1906 and Karl Sternberg (1872–1935), pathologist, described giant cells in 1898.

Chapter 9

The rheumatological system

> The rheumatism is a common name for many aches and pains which have yet got no peculiar appellation, though owing to very different causes.
> *William Heberden (1710–1801)*

Rheumatology is 'the study of the Rheumatic Diseases including arthritis, rheumatic fever, fibrositis, neuralgia, myositis, bursitis, gout and other conditions producing somatic pain, stiffness and soreness' (Oxford English Dictionary, 2nd edn, 1989). The rheumatological system therefore includes diseases of the joints, tendons and muscles.

The rheumatological history

Presenting symptoms (Table 9.1)

Peripheral joints

Pain and swelling

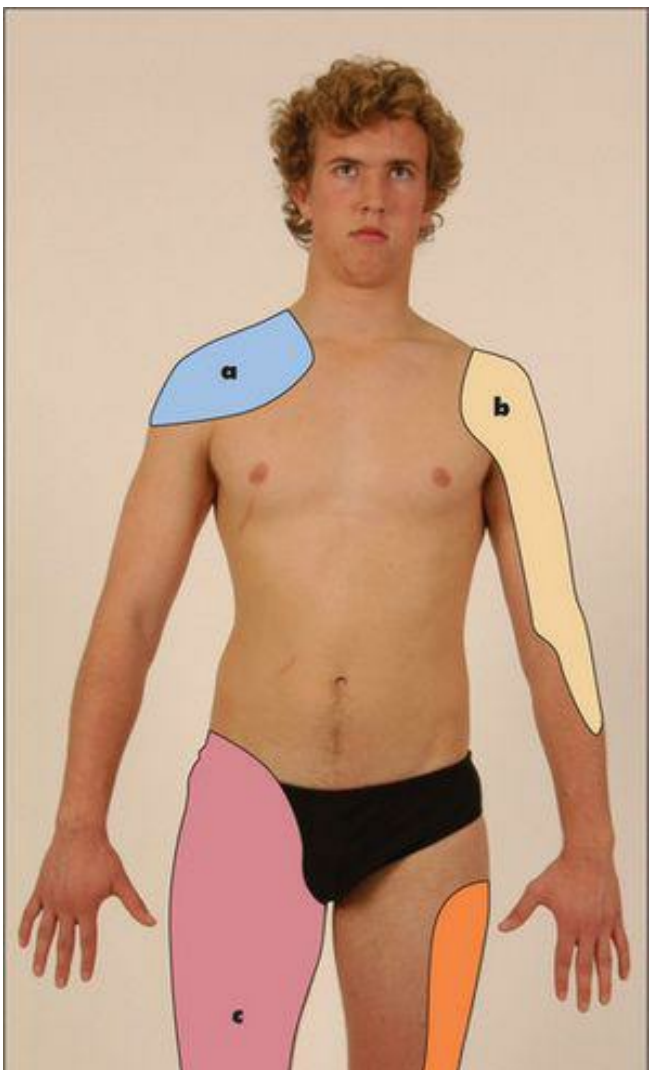
The underlying aetiology of joint pain can often be determined by establishing the distribution and duration of joint involvement. Remember, *arthralgia* is the presence of joint pain without swelling, while with *arthritis* there is usually pain and swelling. Determine whether one or many joints are involved.

TABLE 9.1 Rheumatological history—major symptoms

Joints
Pain
Swelling
Morning stiffness
Stiffness after inactivity
Loss of motion
Loss of function
Deformity
Weakness
Instability
Changes in sensation

Eyes Dry eyes and mouth Red eyes
Systemic Raynaud's phenomenon Rash, fever, fatigue, weight loss, diarrhoea, mucosal ulcers

It is often useful to ask the patient to point to the painful place or area. For example, pain said to affect the knee may be in the popliteal fossa, the knee joint itself, or in the supra- or infra-patellar bursa. Remember also that pain in the knee or lower thigh may be referred from the hip (Figure 9.1).



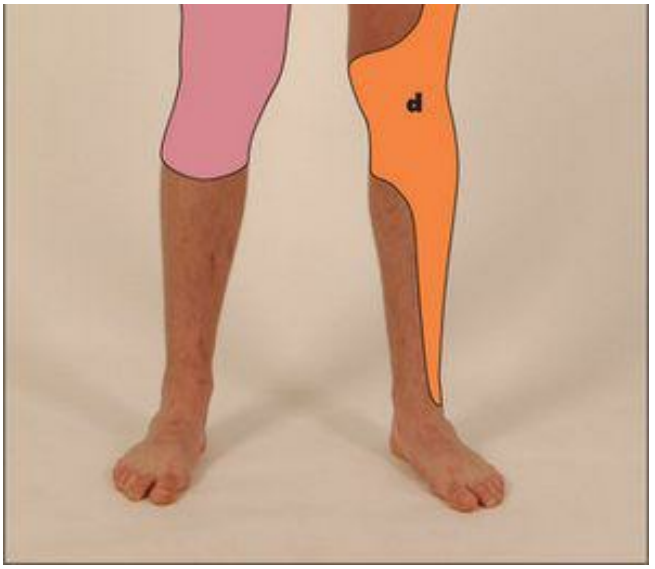


Figure 9.1 Map of referral patterns for different joints
Adapted from Epstein O et al, *Clinical Examination*, 4th edn. Edinburgh: Mosby, 2008.

(a) Acromioclavicular and sternoclavicular joints.

(b) Scapulohumeral joint. (c) Hip. (d) Knee.

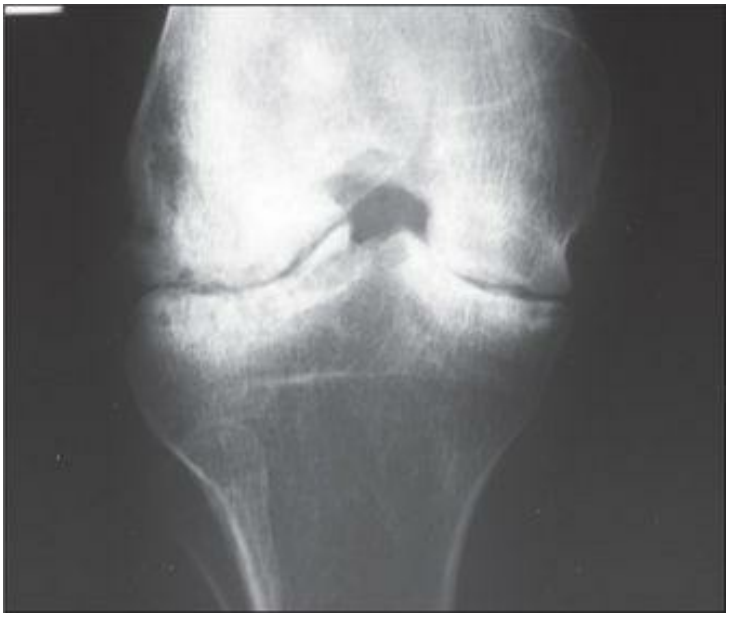


Figure 9.2 Haemophilia
X-ray of the knee showing loss of joint space and some deformity of the adjacent bone. Although the tibia and femur are sclerotic adjacent to the destructive change, the bones are generally osteopenic with mild overgrowth of the epiphysis.

Courtesy Canberra Hospital X-ray library.

Find out if the symptoms are of an acute or chronic nature and whether

Find out if the symptoms are of an acute or chronic nature and whether they are getting better or worse. The effect of rest and exercise on the joint pain should be determined. Patients with rheumatoid arthritis have joint symptoms which are worse after rest, while those with osteoarthritis have pain which is worse after exercise. Ask about the sequence of onset of joint involvement. Precipitating factors such as trauma should be noted. The causes of monoarthritis (single joint) and polyarthritis (more than one joint), and the patterns of polyarthritis in various diseases, are presented in Tables 9.2, 9.3 and 9.4.

TABLE 9.2 Causes of monoarthritis

A single hot red swollen joint (acute monoarthritis)
Septic arthritis • Haematogenous—e.g. staphylococcal or gonococcal (latter may be polyarticular) • Secondary to penetrating injury
Traumatic
Gout, pseudogout, or hydroxyapatite arthropathy
Haemarthrosis—e.g. haemophilia (Figure 9.2)
Seronegative spondyloarthritis (occasionally)
A single chronic inflamed joint (chronic monoarthritis)
Chronic infection—e.g. atypical mycobacterial infection
Seronegative spondyloarthritis
Pigmented villonodular synovitis
Synovial (osteo)chondromatosis

TABLE 9.3 Causes of polyarthritis

Acute polyarthritis
Infection—viral, bacterial
Onset of chronic polyarthritis
Chronic polyarthritis
Rheumatoid arthritis
Seronegative spondyloarthritis
Primary osteoarthritis
Gout, pseudogout, or hydroxyapatite arthropathy
Connective tissue disease, e.g. systemic lupus erythematosus
Infection, e.g. spirochaetal infection (rare)

TABLE 9.4 Patterns of polyarthropathy

Rheumatoid arthritis
This is usually a symmetrical polyarthritis.
Hands: proximal interphalangeal, metacarpophalangeal and wrist joints

Elbows
Small joints of the upper cervical spine
Knees
Ankles
Feet: tarsal and metatarsophalangeal joints
Cervical spine and temporomandibular joints may also be affected
Seronegative spondyloarthritis
Ankylosing spondylitis
Sacroiliac joints and spine
Hips, knees and shoulders
Psoriatic arthritis
Asymmetric oligoarthritis
Sausage digits
Terminal interphalangeal joints
Sacroiliac joints
Rheumatoid pattern
Reiter's syndrome
Sacroiliac joints and spine
Hips

Knees

Ankles and joints of the feet

Primary osteoarthritis

This is usually symmetrical and can affect many joints.

Fingers: distal (Heberden's nodes) and proximal (Bouchard's nodes) interphalangeal joints, and metacarpophalangeal joints of the thumbs

Acromioclavicular joints

Small joints of the spine (lower cervical and lumbar)

Knees

Metatarsophalangeal joints of the great toes

Secondary osteoarthritis

This is:

1. asymmetrical and affects previously injured, inflamed or infected weightbearing joints, particularly hip and knee
2. a result of metabolic conditions, e.g. haemochr omatosis; symptoms and findings are generalised

Morning stiffness

Ask about the presence of early-morning stiffness and the length of time that this stiffness lasts. Morning stiffness classically occurs in rheumatoid arthritis and other inflammatory arthropathies, and the duration of stiffness is a guide to its severity. Stiffness after inactivity, such as sitting, is characteristic of osteoarthritis of the hip or knee.

Deformity

The patient may have noticed deformity of a joint or bone. If there has been progressive change in the shape of the area this is more likely to be significant.

Instability

Joint instability may be described by the patient as a 'giving way', or occasionally 'coming out', of the joint in certain conditions. This may be due to true dislocation (for example, with the shoulder or the patella) or alternatively to muscle weakness or ligamentous problems.

Change in sensation

Change in sensation may occur as a result of nerve entrapment or injury, and sometimes as a result of ischaemia. Ask about numbness or paraesthesiae (pins and needles). The distribution of the change of sensation should help to distinguish nerve damage or entrapment (a specific distribution) from ischaemia.

Back pain

This is a very common symptom. It is most often a consequence of local musculoskeletal disease.

Ask where the pain is situated, whether it began suddenly or gradually, whether it is localised or diffuse, whether it radiates to the limbs or elsewhere, and whether the pain is aggravated by movement, coughing or straining. Musculoskeletal pain is characteristically well localised and is aggravated by movement. If there is a spinal nerve root irritation there may be pain that occurs in a dermatomal distribution. This helps to localise the level of the lesion. Diseases such as osteoporosis (with crush fractures), infiltration of carcinoma, leukaemia or myeloma may cause progressive and unremitting back pain, which is often worse at night (Table 9.6). The pain may be of sudden onset but is usually self-limiting if it results from the crush fracture of a vertebral body. In ankylosing spondylitis the pain is usually situated over the sacroiliac joints and lumbar spine, it is also worse at night and is associated with morning stiffness. The pain of ankylosing spondylitis is typically better with activity which helps distinguish it from mechanical back pain.[1,2] Pain from diseases of the abdomen and chest (e.g. dissecting

abdominal or thoracic aortic aneurysm) can also be referred to the back.

TABLE 9.6 Alarm features for back pain

Age > 50 years
Cancer history
Weight loss (unexplained)
Pain on waking from sleep
Pain for longer than one month and unresponsive to simple analgesics
Fever
History of drug use by injection
Bowel or bladder dysfunction

Limb pain

This can occur from disease of the musculoskeletal system, the skin, the vascular system or the nervous system.

Musculoskeletal pain may be due to trauma or inflammation. Muscle disease such as *polymyositis* can present with an aching pain in the proximal muscles around the shoulders and hips, associated with weakness. Pain and stiffness in the shoulders and hips in patients over the age of 50 years may be due to *polymyalgia rheumatica*. The acute or subacute onset of symptoms in multiple locations suggests an inflammatory process. Bone disease

TABLE 9.5 Functional assessment in rheumatoid arthritis

Class	Assessment
Class 1	Normal functional ability
Class 2	Ability to carry out normal activities, despite discomfort or limited mobility of one or more joints
Class 3	Ability to perform only a few of the tasks of the normal occupation or of self-care
Class 4	Complete or almost complete incapacity with the patient confined to wheelchair or to bed

such as osteomyelitis, osteomalacia, osteoporosis or tumours can cause limb pain. Inflammation of tendons (*tenosynovitis*) can produce local pain over the affected area.

Vascular disease may also produce pain in the limbs. Acute arterial occlusion causes severe pain of sudden onset, often with coolness or pallor. Chronic peripheral vascular disease can result in calf pain on exercise that is relieved by rest. This is called intermittent claudication. Venous thrombosis can also cause diffuse aching pain in the legs associated with swelling.

Spinal stenosis can cause pseudo-claudication—pain on walking but relieved by leaning forward.

Nerve entrapment and *neuropathy* can both cause limb pain which is often associated with paraesthesiae or weakness. The usual cause is synovial thickening or joint subluxation—especially for patients with rheumatoid arthritis. The vasculitis associated with the inflammatory arthropathies can also cause neuropathy leading to diffuse peripheral neuropathy or mononeuritis multiplex. Patients with chronic rheumatoid arthritis often develop subluxation of the cervical spine at the atlanto-axial joint. This is caused by erosion of the transverse ligament around the posterior aspect of the odontoid process (dens). The patient may describe shooting paraesthesiae down the arms and an occipital headache. Neck flexion leads to indentation of the cord by the dens and can cause tetraplegia or sudden death. The

abnormality may be obvious on lateral X-rays of the cervical spine (Figure 9.3). Injury to peripheral nerves can result in vasomotor changes and severe limb pain. This is called *causalgia*. Even following amputation of a limb, phantom limb pain may develop and persist as a chronic problem.

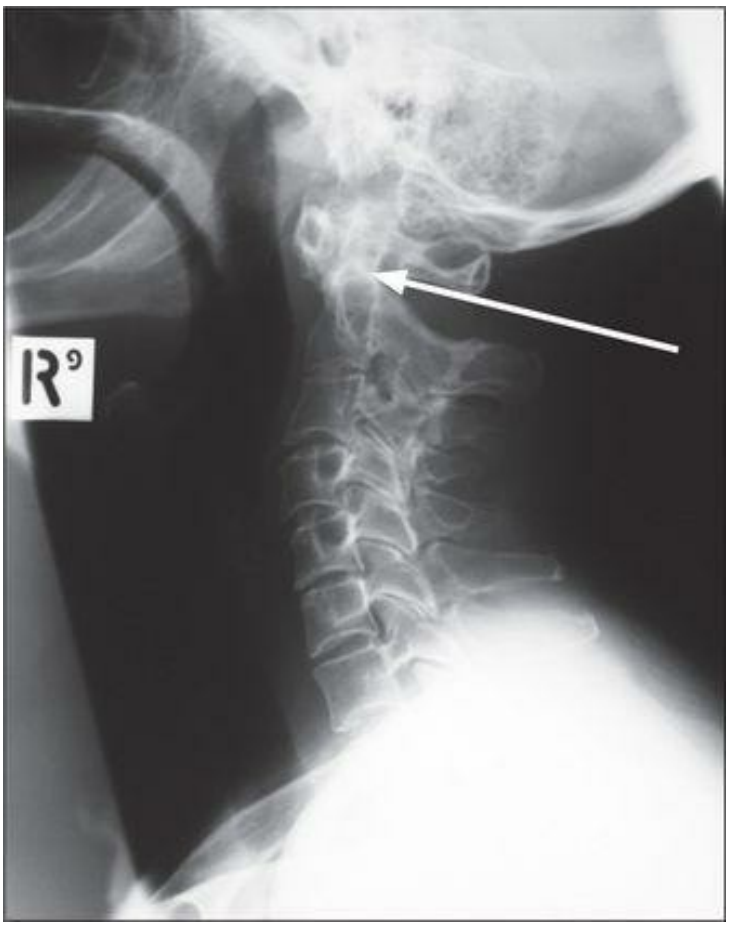


Figure 9.3 Rheumatoid arthritis
Lateral X-ray of the cervical spine showing anterior subluxation of the anterior arch of the dens of the axis (C2, arrow).

Courtesy Canberra Hospital X-ray library.

Raynaud's phenomenon

Raynaud's phenomenon[a] is an abnormal response of the fingers and toes to cold. Classically, the fingers first turn white, then blue and finally red after exposure to cold. It is during the red phase that the pain may be most severe, but pain during the white stage may also be severe, as a result of ischaemia. Patients with Raynaud's disease have Raynaud's phenomenon without an obvious underlying cause. The disease tends to be familial and females are more likely to be affected. In connective tissue diseases, especially

scleroderma, Raynaud's phenomenon can occur and may lead to the formation of digital ulcers (Table 9.7).

TABLE 9.7 Causes of Raynaud's phenomenon (white-blue-red fingers and toes in response to cold)

Reflex
Raynaud's disease (idiopathic)
Vibrating machinery injury
Cervical spondylosis
Connective tissue disease
Scleroderma, diffuse or limited type
Mixed connective tissue disease
Systemic lupus erythematosus
Polyarteritis nodosa
Rheumatoid arthritis
Polymyositis
Vasculitis
Arterial disease
Embolism or thrombosis

Buerger’s disease (thromboangiitis obliterans)—smokers Trauma
Haematological Polycythaemia Leukaemia Dysproteinaemia Cold agglutinin disease
Poisons Drugs: beta-blockers, ergotamine Vinyl chloride

Dry eyes and mouth

Dry eyes and dry mouth are characteristic of Sjögren’s syndrome (Table 9.8). This syndrome may occur in isolation (primary Sjögren’s) and is very common in association with rheumatoid arthritis and other connective tissue disease. Mucus-secreting glands become infiltrated with lymphocytes and plasma cells, which cause atrophy and fibrosis. The dry eyes can result in conjunctivitis, keratitis and corneal ulcers. Sjögren’s syndrome can also have an effect on other organs such as the lungs or kidneys.

TABLE 9.8 Clinical features of Sjögren’s syndrome

In this syndrome mucus-secreting glands are infiltrated by lymphocytes and plasma cells, which cause atrophy and fibrosis of glandular tissue.
1 Dry eyes: conjunctivitis, keratitis, corneal ulcers (rarely vascularisation of the cornea)
2 Dry mouth
3 Chest: infection secondary to reduced mucus secretion or interstitial pneumonitis
4 Kidneys: renal tubular acidosis or nephrogenic diabetes insipidus
5 Genital tract: atrophic vaginitis
6 Pseudolymphoma: lymphadenopathy and splenomegaly, which may rarely progress to a true (usually non-Hodgkin's) lymphoma

Note: This syndrome occurs in rheumatoid arthritis and with the connective tissue diseases.

Red eyes

The seronegative spondyloarthropathies and Behçet's[b] syndrome but not rheumatoid arthritis may be complicated by iritis (eye pain with central scleral injection—a 'red eye'—radiating out from the pupil) (see Figure 9.51, page 279). In other diseases, such as Sjögren's, red eyes may be due to dryness, episcleritis or scleritis.

Systemic symptoms

A number of other symptoms may occur with specific rheumatological diseases. *Fatigue* is common with connective tissue disease. *Weight loss* and *diarrhoea* may occur with scleroderma, because of small-bowel bacterial overgrowth. Mucosal ulcers are common in some connective tissue diseases such as systemic lupus erythematosus (SLE). Specific rashes can also occur. *Generalised stiffness* can be due to rheumatoid arthritis or scleroderma, but other causes include systemic infection (e.g. influenza), excessive exercise, polymyalgia rheumatica, neuromuscular disease (e.g. extrapyramidal disease,

tetanus, myotonia, dermatomyositis) and hypothyroidism. Finally, on occasion *fever* may be associated with the connective tissue diseases, especially SLE, but infection should always be excluded.

Treatment history

Document current and previous anti-arthritic medications (e.g. aspirin, other non-steroidal anti-inflammatory drugs, gold, methotrexate, penicillamine, chloroquine, steroids, anti-tumour necrosis factor α therapy, or other biological agents). Any side-effects of these drugs (e.g. gastric ulceration or haemorrhage, from aspirin) also need to be identified. Ask about physiotherapy and joint or tendon surgery in the past.

Past history

It is important to inquire about any history of trauma or surgery in the past. Similarly, a history of recent infection, including hepatitis, streptococcal pharyngitis, rubella, dysentery, gonorrhoea and tuberculosis, may be relevant to the onset of arthralgia or arthritis. A history of tick bite may indicate that the patient has Lyme disease. Inflammatory bowel disease can be associated with arthritis, as described on page 191. A history of psoriasis may indicate that the arthritis is due to psoriatic arthropathy. It is also important to inquire about any history of arthritis in childhood. The smoking history is important: rheumatoid arthritis is more common in smokers, and smoking adds to their already increased risk of cardiovascular disease.

Social history

Determine the patient's domestic set-up and occupation. This is particularly relevant if a chronic disabling arthritis has developed. Any history of sexually transmitted disease in the past is important, but non-specific urethritis and gonorrhoea are especially relevant.

Family history

Some diseases associated with chronic arthritis run in families. These include rheumatoid arthritis, gout and primary osteoarthritis, haemochromatosis, the seronegative spondyloarthropathies and inflammatory bowel disease. A family history of bleeding disorder may explain an acutely swollen tender joint in a boy (haemophilia).

joint in a boy (haemophilia).

Examination anatomy

Joint structures (Figure 9.4)

Inflammatory arthritis affects first the joint synovium. Thickening of this may be palpable and is called *pannus*. Later, destruction of surrounding structures including tendons, articular cartilages and the bone itself occur.

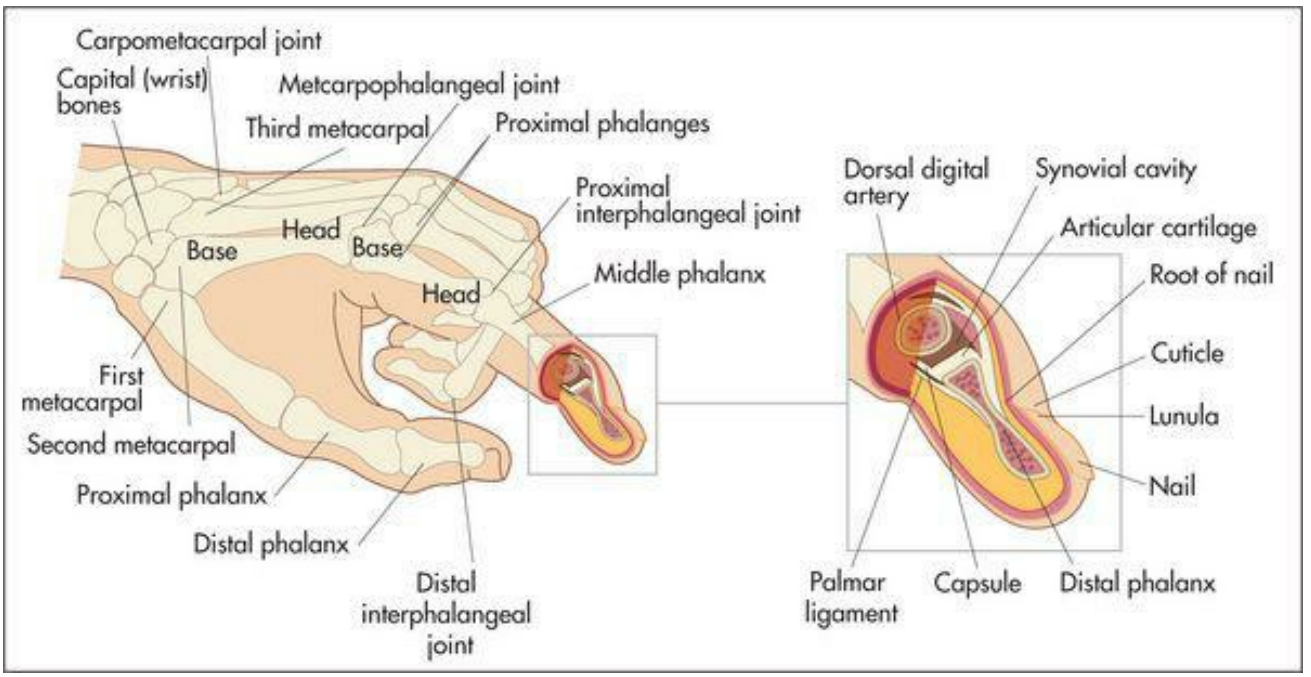


Figure 9.4 Hand bones and finger joint—a typical synovial joint

Joint pain may be well localised if there is inflammation close to the skin, but deeper joint abnormalities may cause pain to be referred. The areas where joint pain is felt correspond to the innervation of the muscle attached to that joint—the myotome. For example, the glenohumeral joint of the shoulder and the posterior scapular muscles are supplied from C5 and C6, so pain over the shoulder or scapula may arise from any structure supplied from these nerve roots—including the shoulder muscles and joints but also the C5 and C6 segments of the spine. Figure 9.1 shows a map of approximate referral patterns for important joints.

The extra-articular structures that surround a joint—the ligaments, tendons and nerves—may also be the source of joint pain. Disease of the joint itself tends to limit movement of the joint in all directions, both active

movement (moved by the patient) and passive movement (moved by the examiner). Extra-articular disease causes variable limitation of movement in different directions, and tends to cause more limitation of active than of passive movement.

The rheumatological examination

There are certain established ways of examining the joints and related structures[3] and it is important to be aware of the numerous systemic complications of rheumatological diseases. The actual system of examination depends on the patient's history and sometimes on the examiner noticing an abnormality on general inspection. Formal examination of all the joints is rarely part of the routine physical examination, but students should learn how to handle each joint properly and a formal examination is an important part of the evaluation of patients who present with joint symptoms or who have an established diagnosis and active symptoms. Diseases of the extra-articular soft tissues are particularly common.

General inspection

This is important for two reasons: first, it gives an indication of the patient's functional disability, which is essential in all rheumatological assessments; and second, certain conditions can be diagnosed by careful inspection. Look at the patient as he or she walks into the room. Does walking appear to be painful and difficult? What posture is taken? Does the patient require assistance such as a stick or walking frame? Is there obvious deformity, and what joints does it involve? Note the pattern of joint involvement, which gives a clue about the likely underlying disease (Tables 9.2 to 9.4).

For a more detailed examination the patient should be undressed as far as practical, usually to the underclothes. Depending on the patient's condition and the parts of the body to be examined, the examination may best be begun with the patient in bed, or sitting over the side of the bed or in a chair, or standing. The opportunity of watching the patient remove the clothes should not be lost because arthritis can interfere with this essential daily task.

Principles of joint examination

Certain general rules apply to the examination of all the joints and they can be summarised as: *look*, *feel*, *move*, *measure*, and *compare with the opposite side*.

Look

The first principle is always to compare right with left. Remember that joints are three-dimensional structures and need to be inspected from the front, the back and the sides. The skin is inspected for *erythema* indicating underlying inflammation and suggesting active, intense arthritis or infection, *atrophy* suggesting chronic underlying disease, *scars* indicating previous operations such as tendon repairs or joint replacements, and *rashes.* For example, psoriasis is associated with a rash and polyarthritis (inflammation of more than one joint). The psoriatic rash consists of scaling erythematous plaques on extensor surfaces. The nails are often also affected (page 252). Also look for a vasculitic skin rash (inflammation of the blood vessels of the skin), which can range in appearance from palpable purpura or livedo reticularis (bluish-purple streaks in a net-like pattern) to skin necrosis.

A small, firm, painless swelling over the back (dorsal surface) of the wrist is usually a synovial cyst—a ganglion.[c] A larger, localised, soft area of swelling of the dorsum of the wrist generally indicates tenosynovitis.

Note any *swelling* over the joint. There are a number of possible causes: these include effusion into the joint space, hypertrophy and inflammation of the synovium (e.g. rheumatoid arthritis), or bony overgrowths at the joint margins (e.g. osteoarthritis). It may also occur when tissues around the joints become involved, as with the tendinitis or bursitis of rheumatoid arthritis. Swelling of the lower legs may be due to fluid retention, which is painless and can occur in association with inflammation anywhere in the leg. Painful swelling may result from inflammation of the ankle joints or tendons, or of the fascia, or from inflammatory oedema of the skin and subcutaneous tissue.

Deformity is the sign of a chronic, usually destructive, arthritis, and ranges from mild ulnar deviation of the metacarpophalangeal joints in early rheumatoid arthritis to the gross destruction and disorganisation of a denervated (Charcot's[d]) joint (Figure 10.20, page 319). Deviation of the part of the body away from the midline is called *valgus* deformity, and towards the midline, *varus* deformity. For example, *genu valgum* means knock-kneed and *genu varum*, bow-legged.

Look for abnormal bone alignment. *Subluxation* is said to be present when displaced parts of the joint surfaces remain partly in contact. *Dislocation* is used to describe displacement where there is loss of contact between the joint surfaces.

Muscle wasting results from a combination of disuse of the joint, inflammation of the surrounding tissues and sometimes nerve entrapment. It tends to affect muscle groups adjacent to the diseased joint (e.g. quadriceps

tends to affect muscle groups adjacent to the diseased joint (e.g. quadriceps wasting with active arthritis of the knee) and is a sign of chronicity.

Feel

Palpate for skin *warmth*. This is done traditionally with the backs of the fingers where temperature appreciation is said to be better. A cool joint is unlikely to be involved in an acute inflammatory process. A swollen and warm joint may be affected by active synovitis (see below), infection (e.g. *Staphylococcus*) or crystal arthritis (e.g. gout).

Tenderness is a guide to the acuteness of the inflammation, but may be present over the muscles of patients with fibromyalgia. The patient must be told to let the examiner know if the examination is becoming uncomfortable. Tenderness can be graded as follows:

Grade I—patient complains of pain

Grade II—patient complains of pain and winces

Grade III—patient complains of pain, winces and withdraws the joint

Grade IV—patient does not allow palpation.

This may result from joint inflammation or from lesions outside the joints (periarticular tissues), including inflamed tendons, bursae, or attachments (entheses). Infected joints are extremely tender and patients will often not let the examiner move the joint at all. Palpation of a joint or area for tenderness must be performed gently, and the patient's face rather than the joint itself should be watched for signs that the examination is uncomfortable.

Palpate the joint deliberately now, if possible, for evidence of *synovitis*, which is a soft and spongy (boggy) swelling. This must be distinguished from an *effusion*, which tends to affect large joints but can occur in any joint. Here the swelling is fluctuant and can be made to shift within the joint. *Bony swelling* feels hard and immobile, and suggests osteophyte formation or subchondral bone thickening.

Move

Much information about certain joints is gained by testing the range of *passive* movement. (Passive movement is obviously contraindicated in cases of recent injury to the limb or joint, such as a suspected fracture.) The patient is asked to relax and let the examiner move the joint. This must be attempted gently and will be limited if the joint is painful (secondary to

attempted gently and will be limited if the joint is painful (secondary to muscle spasm), if a tense effusion is present, if there is capsular contraction or if there is a fixed deformity. The joints may have limited extension (called fixed flexion deformity) or limited flexion (fixed extension deformity). Passive movement of the spine is not a practical manoeuvre (unless the examiner is very strong), and active movement is tested here. *Active* movement is more helpful in assessing integrated joint function. Hand function and gait are usually applied as tests of *function*. *Pain on motion* indicates a joint or periarticular problem.

Stability of the joint is important and depends largely on the surrounding ligaments. This is tested by attempting to move the joint gently in abnormal directions to its usual limits, set by ligaments and muscular tone.

Joint crepitus, which is a grating sensation or noise from the joint, indicates irregularity of the articular surfaces. Its presence suggests chronicity.

Measure

Accurate measurement of the range of movement of a joint is possible with a goniometer, which is a hinged rod with a protractor in the centre. The jaws are opened and lined up with the joint. Measurement of joint movements is performed from the zero starting position. For most joints this is the anatomical position in extension—e.g. the straightened knee. Movement is then recorded as the number of degrees of flexion from this position. A knee with a fixed flexion deformity may be recorded as '30 to 60 degrees', which indicates that there is 30 degrees of fixed flexion deformity and that flexion is limited to 60 degrees. At some joints both flexion and extension from the anatomical position can be measured, as at the wrists. The goniometer is not routinely used by non-rheumatologists and there is a wide range of normal values for joint movement. Most clinicians estimate the approximate joint angles.

A tape measure is useful for measuring and following serially the quadriceps muscle bulk and in examination of spinal movements.

Assessment of individual joints

The hands and wrists (Figures 9.5 to 9.9)

Examination anatomy

The articulations between the phalanges are synovial hinge joints. The eight bones of the wrist (carpal bones) form gliding joints which allow wrist movements—flexion/extension and abduction/adduction as they slide over each other.

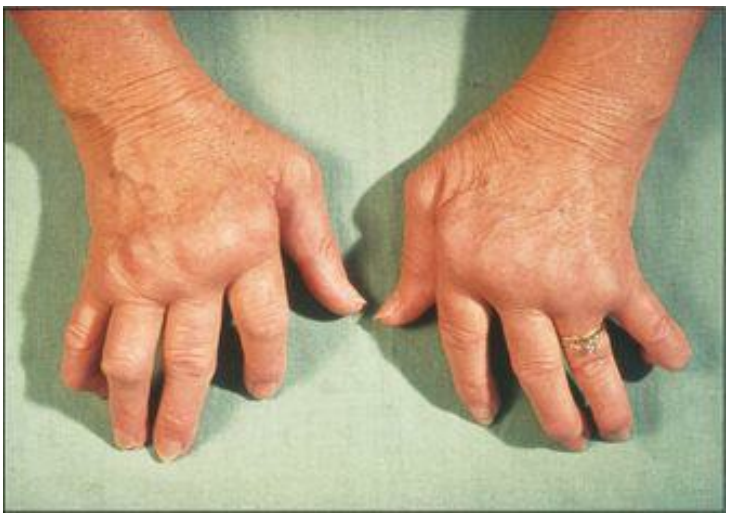


Figure 9.5 Examination of the hands and wrists
Sitting up (hands on a pillow)

1. **General inspection**
   Cushingoid
   Weight
   Iritis, scleritis, etc
   Obvious other joint disease

2. **Look**
   Dorsal aspect
   - Wrists
     Skin—scars, redness, atrophy, rash
     Swelling—distribution
     Deformity
     Muscle wasting
   - Metacarpophalangeal joints
     Skin
     Swelling—distribution
     Deformity—ulnar deviation, volar subluxation etc
   - Proximal and distal interphalangeal joints
     Skin
     Swelling—distribution
     Deformity—swan necking, boutonnière, Z, etc

- Nails

  Psoriatic changes—pitting, ridging, onycholysis, hyperkeratosis, discoloration

3. **Feel and move passively**

   Wrists
   - Synovitis
   - Effusions
   - Range of movement
   - Crepitus
   - Ulnar styloid tenderness

   Metacarpophalangeal joints
   - Synovitis
   - Effusions
   - Range of movement
   - Crepitus
   - Subluxation

   Proximal and distal interphalangeal joints
   - As above

   Palmar tendon crepitus

   Carpal tunnel syndrome tests

   Palmar aspect
   - Skin—scars, palmar erythema, palmar creases (anaemia)
   - Muscle wasting

4. **Hand function**

   Grip strength

   Key grip

   Opposition strength

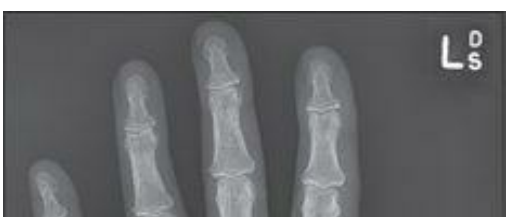
   Practical ability

5. **Other**

   Elbows—subcutaneous nodules—psoriatic rash

   Other joints

   Signs of systemic disease



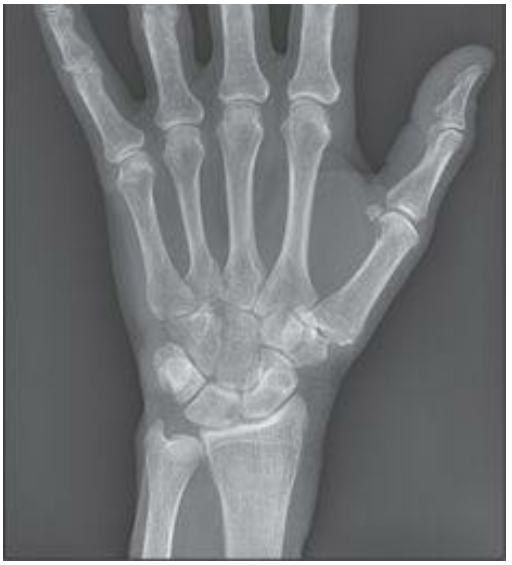


Figure 9.6 (right) X-ray of normal hand

Courtesy M Thomson, National Capital Diagnostic Imaging, Canberra.

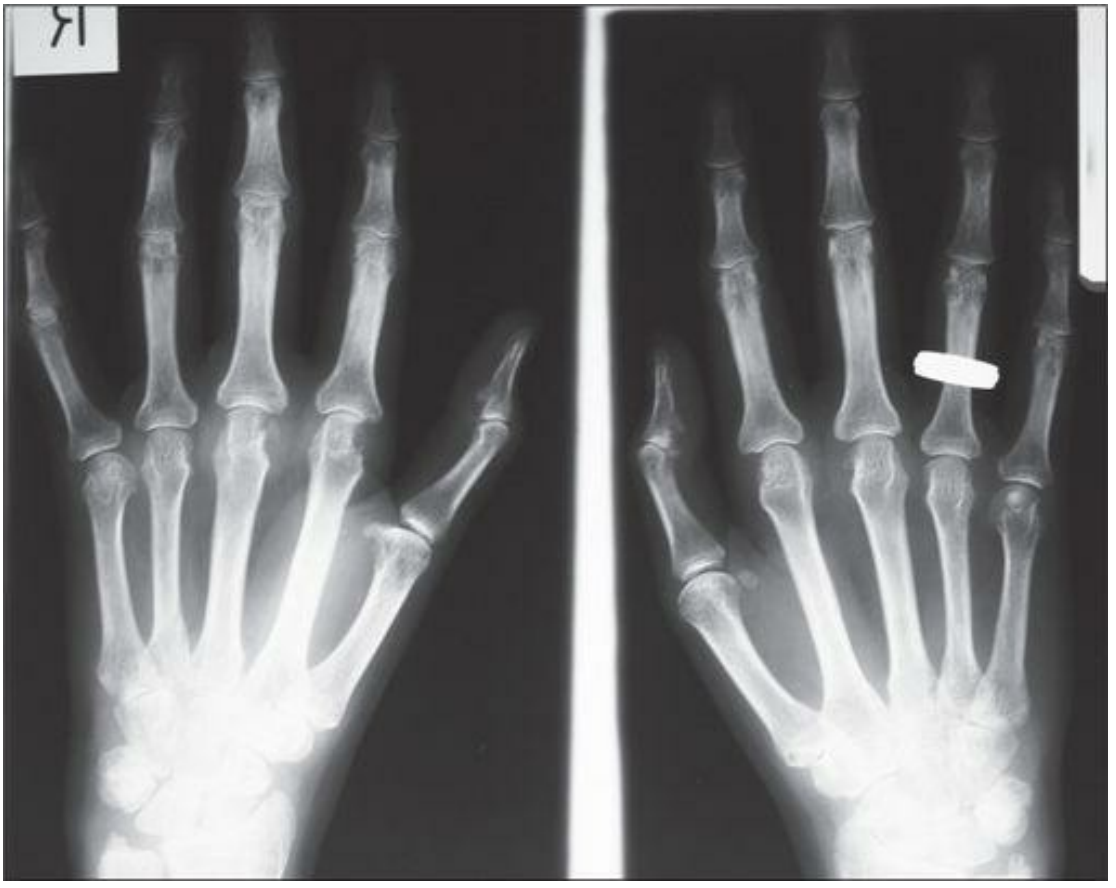


Figure 9.7 Rheumatoid arthritis, early findings
X-ray of the hands of a patient with early rheumatoid arthritis. Note erosions of the heads of the metacarpophalangeal joints and of the ulnar styloid, and reduced amounts of cartilage in the joint spaces.

Courtesy Canberra Hospital X-ray library.

Courtesy Canberra Hospital X-ray library.

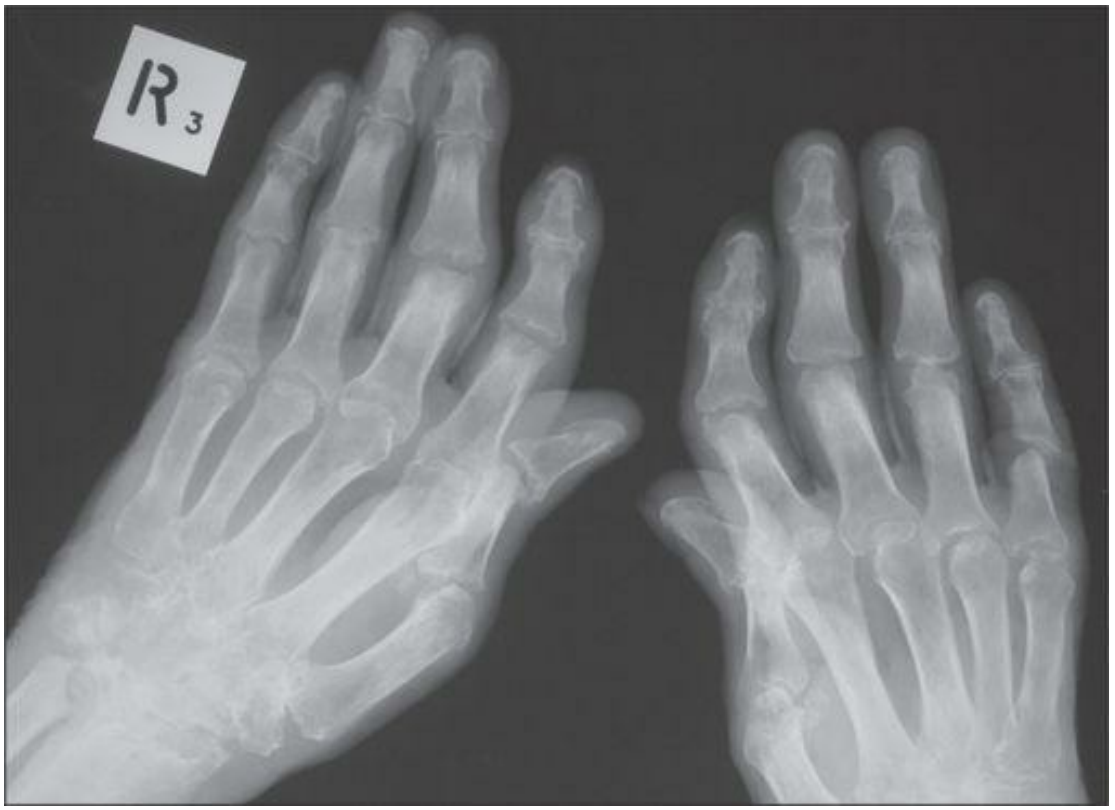
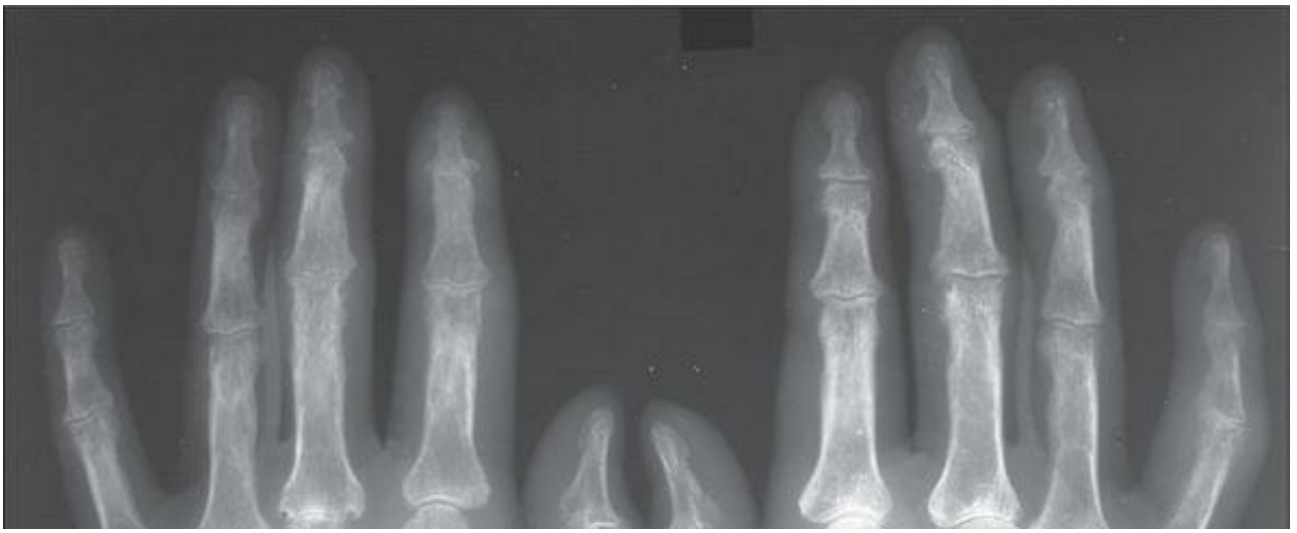


Figure 9.8 Rheumatoid arthritis, late findings
X-ray of the hands of a patient with advanced rheumatoid arthritis. Note loss of joint space and destruction of the right carpal joints, subluxation of metacarpophalangeal and poximal interphalangeal (PIP) joints, and Z deformity of the thumb. There are erosions of the PIP joints, a sign of active disease.

Courtesy Canberra Hospital X-ray library.



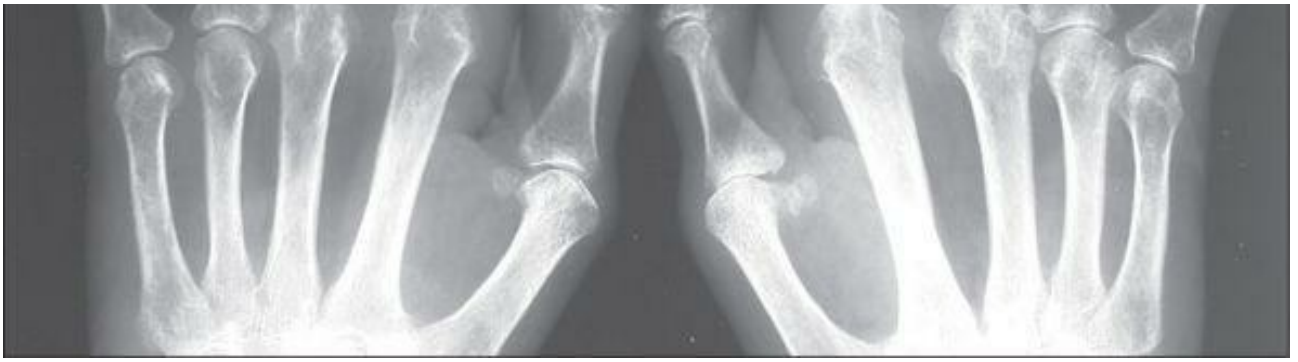


Figure 9.9 Osteoarthritis arthritis
X-ray of the hands showing the typical findings of osteoarthritis with joint-space narrowing and proliferative changes in the distal joints. Also note erosive and destructive changes at multiple proximal interphalangeal joints.

Courtesy Canberra Hospital X-ray library.

History

Pain may be present in some or all of the joints. It is more likely to be vague or diffuse if it has radiated from the shoulder or neck or is due to carpal tunnel syndrome, and to be localised if it is due to arthritis. *Stiffness* is typically worse in the mornings in rheumatoid arthritis. *Swelling* of the wrist may indicate arthritis or tendon sheath inflammation. Swelling of individual joints suggests arthritis. *Deformity* of the fingers and hand due to rheumatoid arthritis or of the fingers as a result of arthritis or gouty tophi may be the presenting complaint. The sudden onset of deformity suggests tendon rupture. *Locking or snapping* of a finger (trigger finger) is typical of inflammation of a flexor tendon sheath (tenovaginitis). *Loss of function* is a serious problem when it involves the numerous functions of the hand and wrist. The history should include an assessment of the difficulties the patient has in using the hands and wrists. *Neurological symptoms* as a result of nerve compression may cause paraesthesiae or limitation of strength or of complicated hand functions.

Examination

First sit the patient over the side of the bed and place the hands on the pillow with palms down. Often examination of the hands alone will give enough information for the examiner to make a diagnosis. As a result this is quite a popular test in viva voce examinations.

Start the examination at the *wrists and forearms*. Inspect the skin for erythema, atrophy, scars and rashes. Look for swelling and its distribution. Next look at the wrist for swelling, deformity, ulnar and hyloid prominence. Then look for muscle wasting of the intrinsic muscles of the hand. This results in the appearance of hollow ridges between the metacarpal bones. It is especially obvious on the dorsum of the hand.

Go on to the *metacarpophalangeal joints*. Again note any skin abnormalities, swelling or deformity. Look especially for ulnar deviation and volar (palmar) subluxation of the fingers. Ulnar deviation is deviation of the phalanges at the metacarpophalangeal joints towards the medial (ulnar) side of the hand. It is usually associated with anterior (Volar) subluxation of the fingers (Figure 9.10). These deformities are characteristic but not pathognomonic of rheumatoid arthritis (Table 9.9).

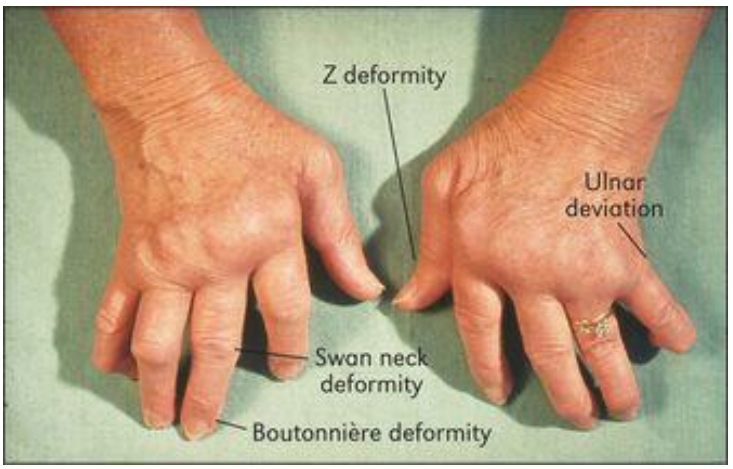


Figure 9.10 The hands in rheumatoid arthritis

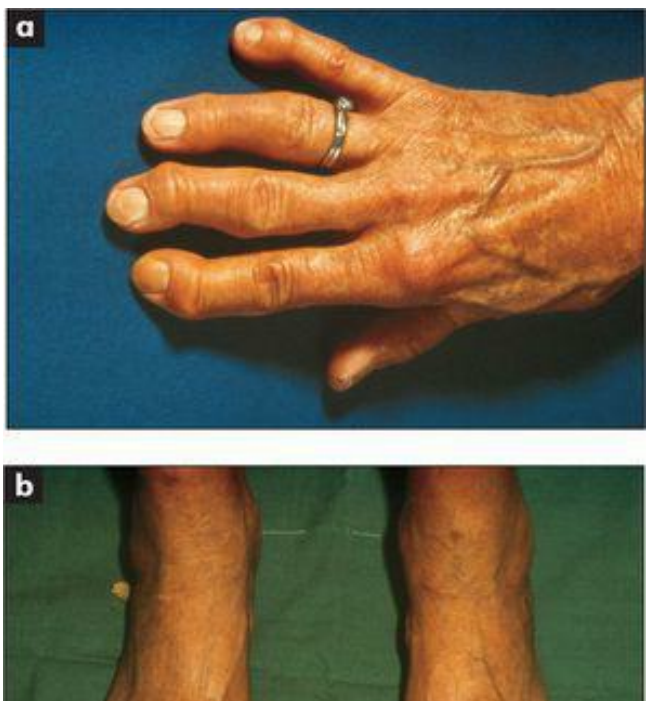
TABLE 9.9 Differential diagnosis of a deforming polyarthropathy

Rheumatoid arthritis
Seronegative spondyloarthropathy, particularly psoriatic arthritis, ankylosing spondylitis or Reiter's disease

Chronic tophaceous gout (rarely symmetrical)
Primary generalised osteoarthritis
Erosive or inflammatory osteoarthritis

Next inspect the *proximal interphalangeal* and *distal interphalangeal* joints. Again note any skin changes and joint swelling. Look for the characteristic deformities of *rheumatoid arthritis*. These include *swan neck* and *boutonnière* deformity of the fingers and *Z deformity* of the thumb (Figure 9.10). They are due to joint destruction and tendon dysfunction. The swan neck deformity is hyperextension at the proximal interphalangeal joint and fixed flexion deformity at the distal interphalangeal joint. It is due to subluxation at the proximal interphalangeal joint and tendon shortening at the distal interphalangeal joint. The boutonnière (buttonhole) deformity consists of fixed flexion of the proximal interphalangeal joint and extension of the distal interphalangeal joints. This is due to protrusion of the proximal interphalangeal joint through its ruptured extensor tendon. The Z deformity of the thumb consists of hyperextension of the interphalangeal joint and fixed flexion and subluxation of the metacarpophalangeal joint.

Now look for the characteristic changes of *osteoarthritis* (Figure 9.11). Here the distal interphalangeal and first carpometacarpal joints are usually involved. *Heberden's nodes*[e] are a common deformity caused by marginal osteophytes that lie at the base of the distal phalanx. Less commonly, the proximal interphalangeal joints may be involved and osteophytes here are called *Bouchard's*[f] nodes.



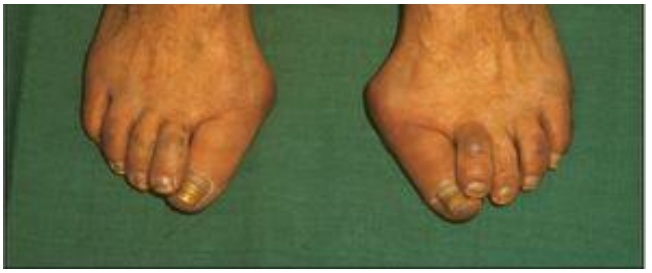


Figure 9.11 The hands (a) and the feet (b) of a patient with osteoarthritis
Showing Heberden's nodes (distal interphalangeal joints), Bouchard's nodes (proximal interphalangeal joints) and bunions.

Look also to see if the phalanges appear *sausage-shaped*. This is characteristic of psoriatic arthropathy, but can also occur in patients with Reiter's disease. It is due to interphalangeal arthritis and flexor tendon sheath oedema. Finger shortening due to severe destructive arthritis also occurs in psoriatic disease and is called *arthritis mutilans*. The hand may take up a *main en lorgnette* ('hand holding long-handled opera glasses') appearance due to a combination of shortening and telescoping of the digits.

Now examine the *nails*. Characteristic *psoriatic* nail changes may be visible: these include pitting (small depressions in the nail), onycholysis (Figure 9.12) and, less commonly, hyperkeratosis (thickening of the nail), ridging and discoloration. The presence of *vasculitic* changes around the nailfolds implies active disease. These consist of black to brown 1–2 mm lesions due to skin infarction and occur typically in rheumatoid arthritis (Figure 9.13). Splinter haemorrhages may be present in patients with systemic lupus erythematosus (and infective endocarditis) and are due to vasculitis. Unlike nailfold infarcts they are located under the nails in the nail beds. Periungual telangiectasiae occur in systemic lupus erythematosus, scleroderma or dermatomyositis.

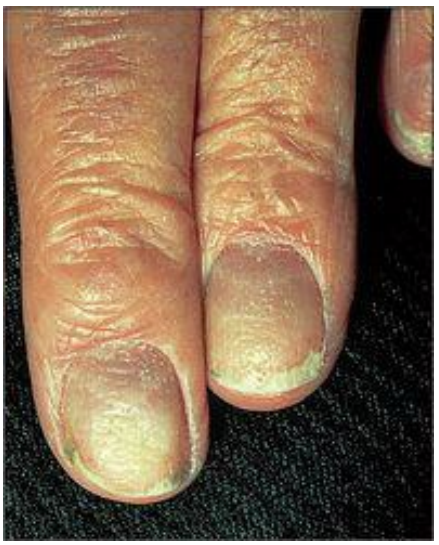


Figure 9.12 Psoriatic nails

Figure 9.12 Psoriatic nails
Showing onycholysis and discoloration, with typical pitting and ridging.

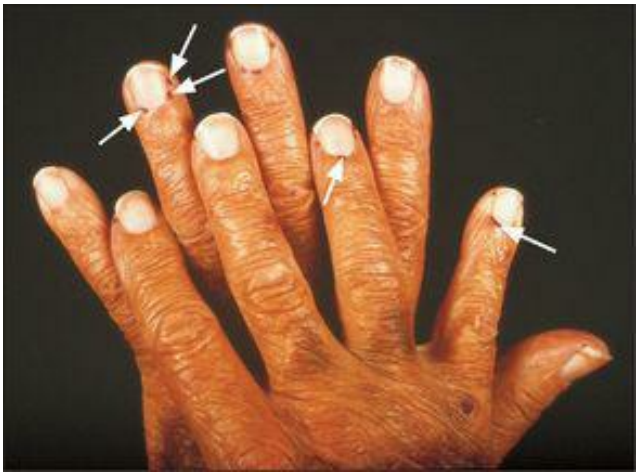


Figure 9.13 Rheumatoid vasculitis (arrows)

The hands should now be turned over and the *palmar surfaces* revealed. Look at the palms for scars (from tendon repairs or transfers), palmar erythema, and muscle wasting of the thenar or hypothenar eminences (due to disuse, vasculitis or peripheral nerve entrapment). Telangiectasia here would support the diagnosis of scleroderma.

Feel and move

Turn the hands back again to the palm-down position. Palpate the *wrists* with both thumbs placed on the dorsal surface by the wrists, supported underneath by the index fingers (Figure 9.14). Feel gently for synovitis (boggy swelling) and effusions. The wrist should be gently dorsiflexed (normally possible to 75 degrees) and palmar flexed (also possible to 75 degrees) with the examiner's thumbs. Then radial and ulnar deviation (20 degrees) is tested (Figure 9.15). Note any tenderness or limitation of movement or joint crepitus. Palpate the ulnar styloid for tenderness, which can occur in rheumatoid arthritis.

Figure 9.14 Palpating the wrist joint—approved method

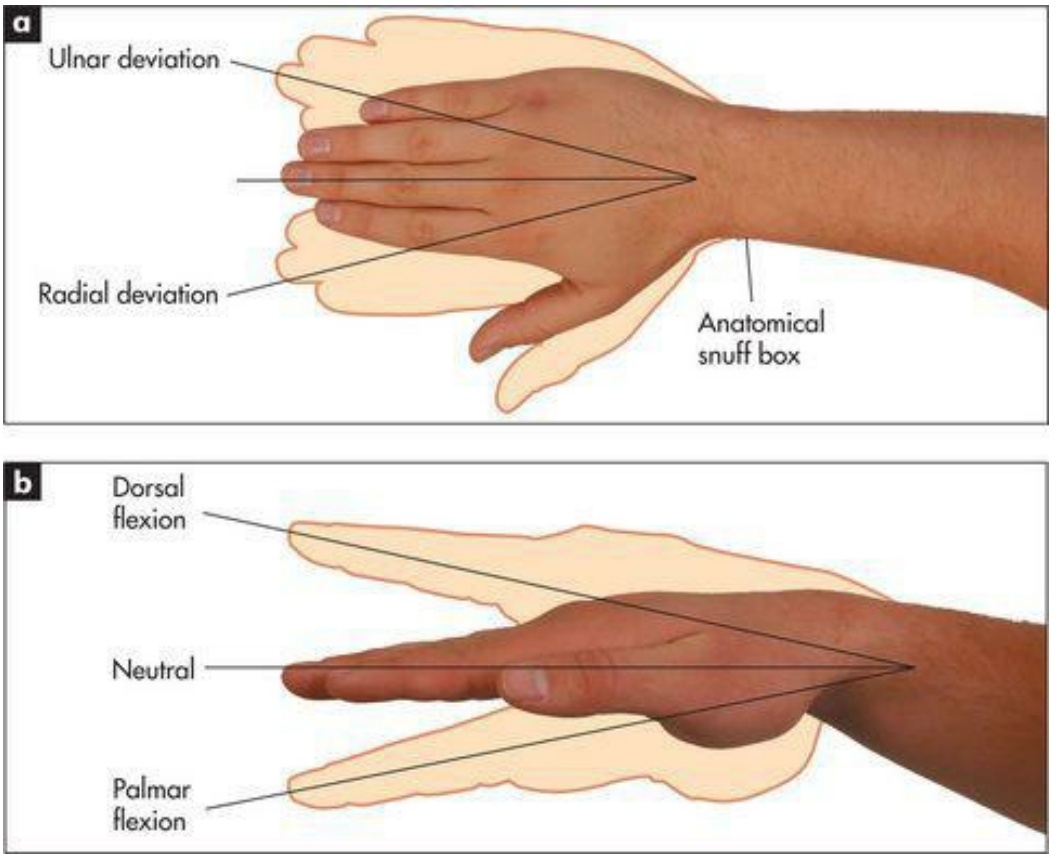


Figure 9.15 Movements of the wrist
(a) Ulnar and radial deviation. (b) Dorsal and palmar flexion.

Test for tenderness at the tip of the radial styloid. This suggests de Quervain's[g] tenosynovitis.

Feel for tenderness in the anatomical snuff box if scaphoid injury is suspected (Figure 9.15). Test for tenderness distal to the head of the ulna for extensor carpi ulnaris tendinitis.

Go on now to the *metacarpophalangeal joints*, which are palpated in a similar way with the two thumbs. Again passive movement is tested. Volar subluxation can be demonstrated by flexing the metacarpophalangeal joint with the proximal phalanx held between the thumb and forefinger. The metacarpophalangeal joint is then rocked backwards and forwards (Figure 9.16). Very little movement occurs with this manoeuvre at a normal joint. Considerable movement may be present when ligamentous laxity or

Considerable movement may be present when ligamentous laxity or subluxation is present.

Figure 9.16 Examination for volar subluxation at the metacarpophalangeal joints

Palpate the *proximal* and *distal interphalangeal* joints for tenderness, swelling and osteophytes.

Next test for *palmar tendon crepitus*. The palmar aspects of the examiner's fingers are placed against the palm of the patient's hand while he or she flexes and extends the metacarpophalangeal joints. Inflamed palmar tendons can be felt creaking in their thickened sheaths and nodules can be palpated. This indicates tenosynovitis.

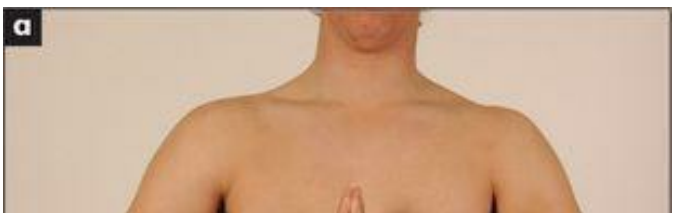
A *trigger finger* may also be detected by this manoeuvre. Here the thickening of a section of digital flexor tendon is such that it tends to jam when passing through a narrowed part of its tendon sheath. Rheumatoid arthritis is an important cause. Typically, flexion of the finger occurs freely up to a certain point where it sticks and cannot be extended (as flexors are more powerful than extensors). The application of greater force overcomes the resistance with a snap.

If the carpal tunnel syndrome is suspected, ask the patient to flex both wrists for 30 seconds—paraesthesiae will often be precipitated in the affected hand if the syndrome is present (*Phalen's*[h] wrist flexion test*)*. The paraesthesiae (pins and needles) are in the distribution of the median nerve (page 363), when thickening of the flexor retinaculum has entrapped the nerve in the carpal tunnel (Table 9.10). This test is more reliable than *Tinel's sign*,[i] in which tapping over the flexor retinaculum (which lies at the proximal part of the palm) may cause similar paraesthesiae.[4]

TABLE 9.10 Causes of carpal tunnel syndrome

Occupation-related: working with wrists and hands flexed Rheumatoid arthritis Hypothyroidism Acromegaly	Pregnancy Gout Obesity Amyloidosis Diabetes mellitus Idiopathic Carpal bone osteomyelitis

Now test active movements. First assess *wrist flexion and extension* as shown in Figure 9.17. Compare the two sides. Now go on to *thumb movements* (Figure 9.18). The patient holds the hand flat, palm upwards, and the examiner's hand holds the patient's fingers. Test *extension* by asking the patient to stretch the thumb outwards, *abduction* by asking for the thumb to be pointed straight upwards, *adduction* by asking him or her to squeeze the examiner's finger, and *opposition* by getting the patient to touch the little finger with the thumb. Look for limitation of these movements and discomfort caused by them. Next test *metacarpophalangeal and interphalangeal movements*. As a screening test, ask the patient to make a fist then to straighten out the fingers (Figure 9.19). Then test the fingers individually. If active flexion of one or more fingers is reduced, test the superficial and profundus flexor tendons (Figure 9.20). Hold the proximal finger joint extended and instruct the patient to bend it; the distal fingertip will flex if the flexor profundus is intact. Then hold the other fingers extended (to inactivate the profundus) and check finger flexion (inability indicates the superficialis is unable to work). The most common tendon ruptures are of the extensors of the fourth and fifth fingers.



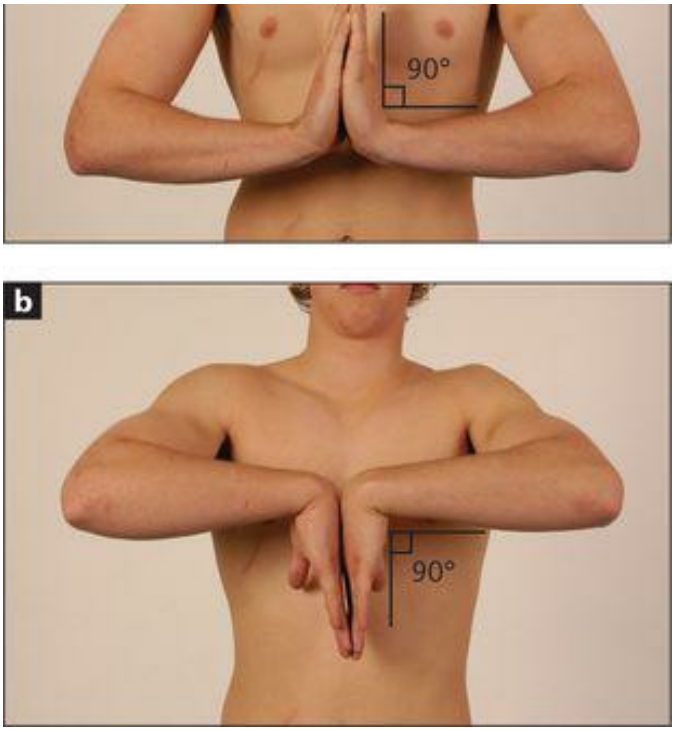


Figure 9.17 (a) Active wrist extension and (b) active wrist flexion

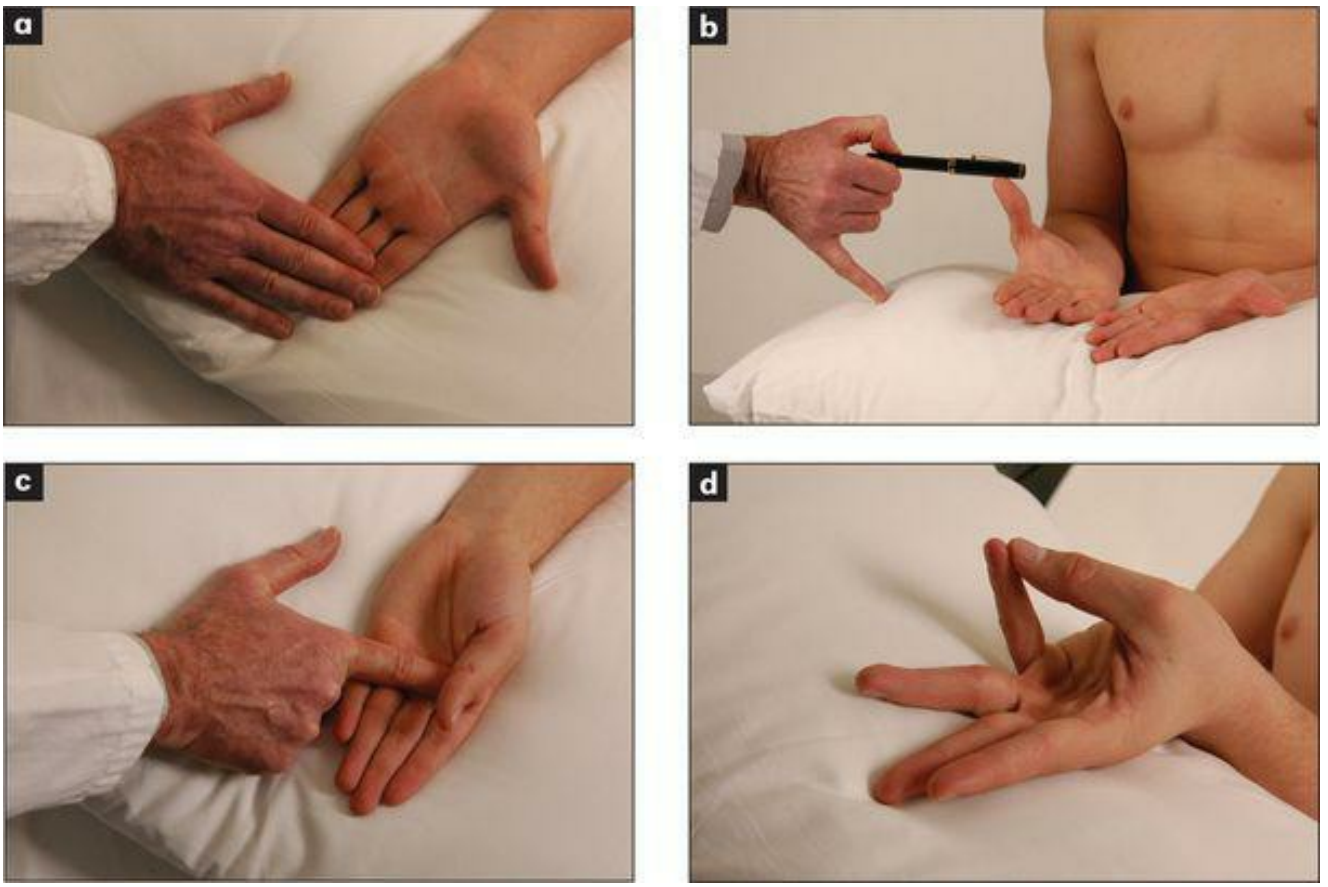


Figure 9.18 Thumb movements
(a) Extension. (b) Abduction. (c) Adduction. (d) Opposition.

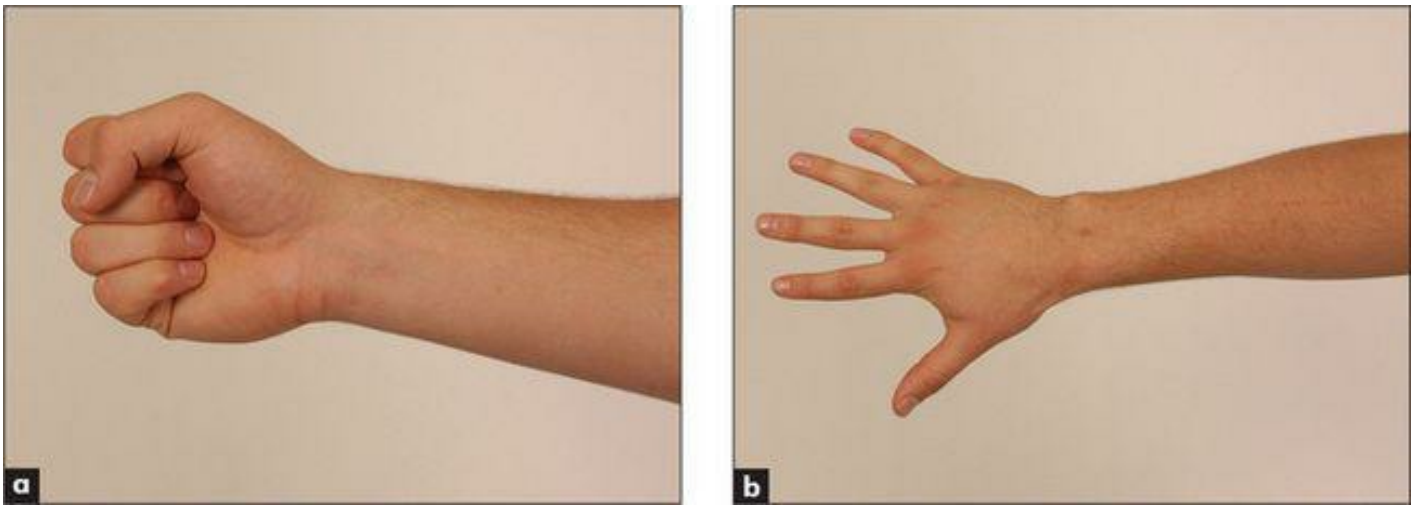


Figure 9.19 Screening metacarpophalangeal and interphalangeal movements
(a) Flexion. (b) Extension.

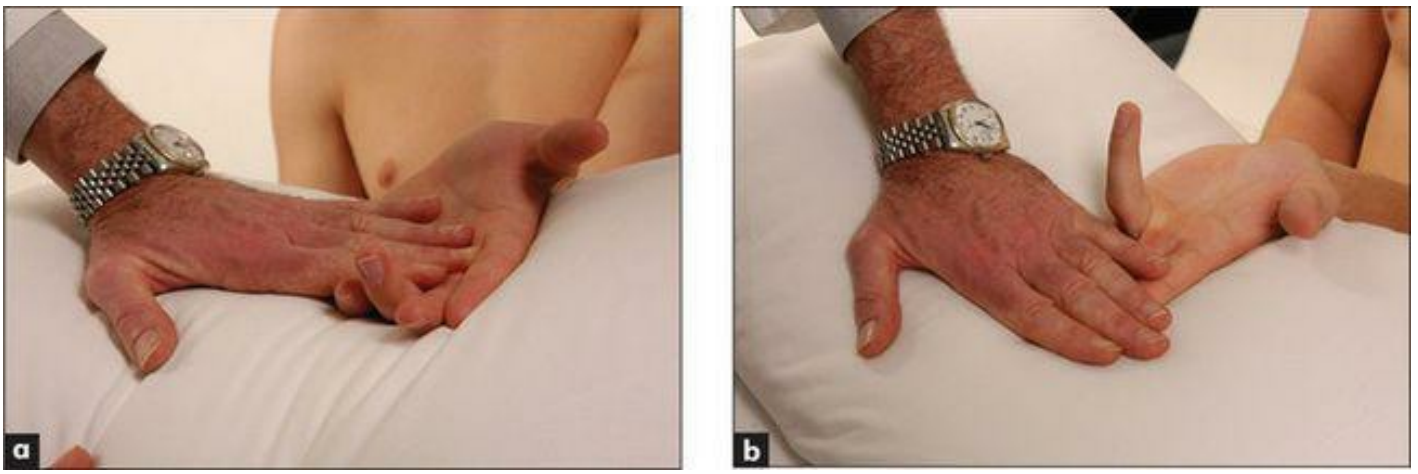


Figure 9.20 Testing the superficial and profundus flexor tendons
(a) Flexor profundus. (b) Flexor superficialis.

Function

It is important to test the function of the hand. *Grip strength* is tested by getting the patient to squeeze two of the examiner's fingers. Even an angry patient will rarely cause pain if given only two fingers. Serial measurements of grip strength can be made by asking the patient to squeeze a partly inflated sphygmomanometer cuff and noting the pressure reached. *Key grip* (Figure 9.21) is the grip with which a key is held between the pulps of the thumb and

forefinger. Ask the patient to hold this grip tightly and try to open up his or her fingers. *Opposition strength* (Figure 9.22) is where the patient opposes the thumb and individual fingers. The difficulty with which these can be forced apart is assessed. Finally, *a practical test*, such as asking the patient to undo a button or write with a pen, should be performed.

Figure 9.21 The key grip

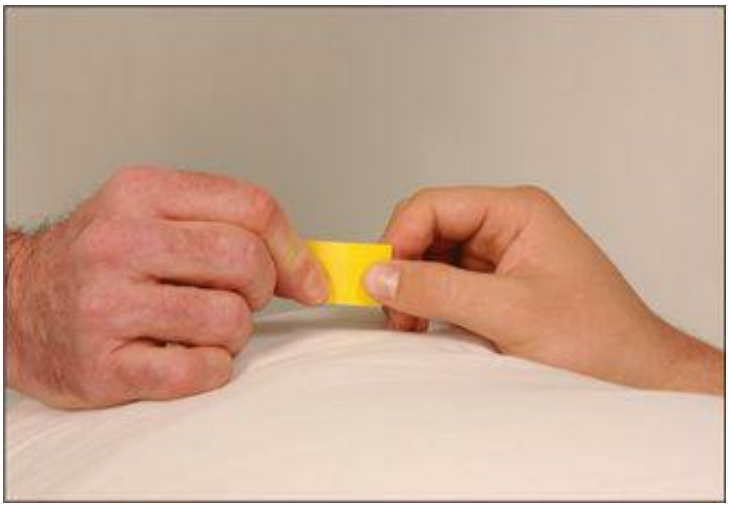


Figure 9.22 Testing opposition strength

Tests of hand function should be completed by formally assessing for neurological changes (page 362).

Examination of the hands is not complete without feeling for the *subcutaneous nodules* of rheumatoid arthritis near the elbows (Figure 9.23). These are 0.5–3 cm firm, shotty, non-tender lumps which occur typically over the olecranon. They may be attached to bone. They are found in rheumatoid-factor-positive rheumatoid arthritis. Rheumatoid nodules are

rheumatoid-factor-positive rheumatoid arthritis. Rheumatoid nodules are areas of fibrinoid necrosis with a characteristic histological appearance and are probably initiated by a small vessel vasculitis. They are localised by trauma but can occur elsewhere, especially attached to tendons, over pressure areas in the hands or feet, in the lung, pleura, myocardium or vocal cords. The combination of arthritis and nodules suggests the diagnostic possibilities listed in Table 9.11.

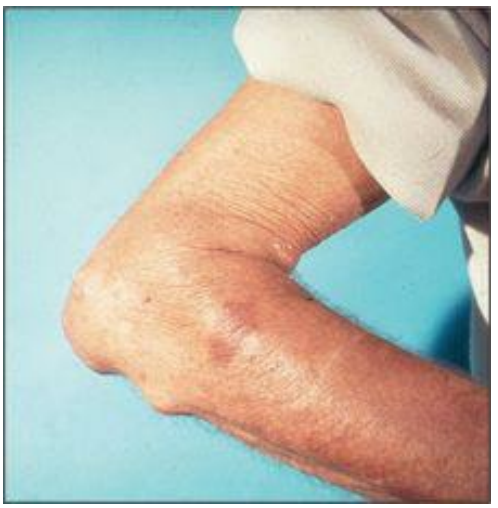


Figure 9.23 Subcutaneous nodules in rheumatoid arthritis

TABLE 9.11 Causes of arthritis plus nodules*

Rheumatoid arthritis
Systemic lupus erythematosus (rare)
Rheumatic fever (Jaccoud's† arthritis) (very rare)
Granulomas—e.g. sarcoidosis (very rare)

* Gouty tophi and xanthomata from hyperlipidaemia may cause confusion.

† François Jaccoud (1830–1913), professor of medicine, Geneva.

The elbows

Examination anatomy (Figure 9.24)

The humerus, radius and ulna meet at the elbow, which is a hinge and a pivot joint. Pivoting occurs between the radius and ulna, and the articulation between all three bones forms a hinge joint.

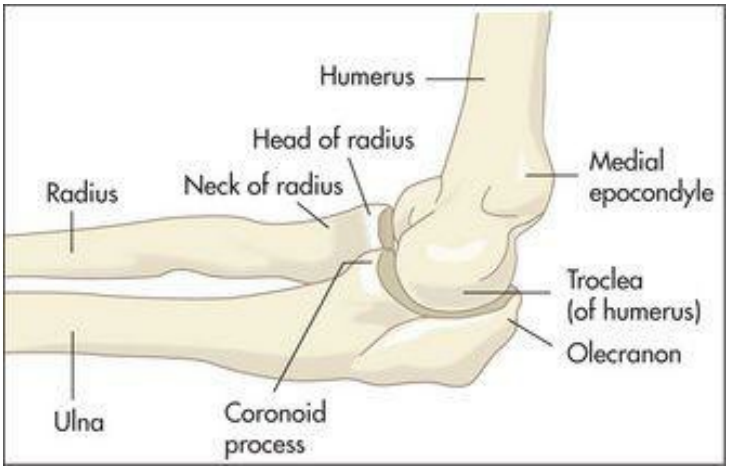


Figure 9.24 Anatomy of the elbows

History

Pain from the elbow is usually diffuse and may radiate down the forearm. It may occur over the lateral or medial epicondyle if the patient has tendinitis (tennis or golfer's elbow). The patient may have noticed some swelling as a result of inflammation. *Swelling* over the back suggests olecranon bursitis. *Stiffness* may interfere with elbow movements and the patient may complain of difficulty combing the hair. When supination and pronation are affected the patient may complain of difficulty with carrying and holding. If the patient is aware of the elbow moving abnormally this suggests *instability* of the joint and may be a result of rheumatoid arthritis or trauma. Ulnar nerve trauma at the elbow may lead to a complaint of *numbness* or *paraesthesiae* in the distribution of that nerve.

Examination

Watch as the patient undresses, for difficulty disentangling the arms from clothing. The upper arms should be exposed completely. Note any deformity or difference in the normal 5–10 degree valgus position (carrying angle) as