CHAPTER 1

MUSCULOSKELETAL SIGNS
Anterior drawer test

Mechanism/s
The ACL arises from the anterior aspect of the tibial plateau and inserts into the medial aspect of the lateral femoral condyle. It limits anterior movement of the tibia upon the femur. Loss of continuity of the ACL permits inappropriate anterior movement of the tibia and thus knee joint instability.

Sign value
A literature review of six studies reported variable sensitivity of 27–88%, specificity of 91–99%, positive LR of 11.5 and negative LR of 0.5.2 A literature review by Solomon DH et al. of nine studies reported a sensitivity of 9–93% and specificity of 23–100%.1

While a positive anterior drawer sign (+LR 11.5)2 has been suggested to be strong evidence of ACL injury, the results are not uniform, with another study reporting a +LR 2.0 (sensitivity 83%, specificity 57%, –LR 0.3).3 A negative anterior drawer sign cannot reliably exclude ACL injury (sensitivity 27–88%; –LR 0.5).2 When strong clinical suspicion persists, further diagnostic steps are necessary (e.g. interval re-examination, MRI, arthroscopy).

Description
With the patient lying supine, the knee at 90° flexion and the foot immobilised by the examiner, the proximal third of the tibia is pulled towards the examiner. In a positive test, there is anterior (forward) movement of the tibia without an abrupt stop.1

Condition/s associated with
• Anterior cruciate ligament (ACL) injury

FIGURE 1.1
Anterior drawer test for anterior cruciate ligament injury
Apley’s grind test

Description
With the patient lying prone and the knee at 90° flexion, the lower leg is passively internally and externally rotated while axial pressure is applied to the lower leg. The test is considered positive if tenderness is elicited.

The process can also be combined with or without distraction. If rotation plus distraction is more painful or there is increased rotation relative to the unaffected side, this is suggestive of a ligamentous lesion. If rotation plus compression is more painful or there is decreased rotation relative to the unaffected side, this is suggestive of meniscal injury.

Condition/s associated with
- Meniscal injury

Mechanism/s
Direct mechanical force upon the injured meniscus elicits tenderness.

Sign value
A review by Hegedus EJ et al. reported a pooled sensitivity of 60.7% and specificity of 70.2% with an odds ratio of 3.4. Significant heterogeneity in the data limits its accuracy. Overall, Apley’s grind test has limited diagnostic utility, limited supporting data and, in the acute setting, the manoeuvre produces severe pain.

McMurray’s grind test has more robust supporting data.
Condition/s associated with

Common
• Rotator cuff muscle injury
• Labral tear
• Anterior shoulder dislocation
• Bicipital tenosynovitis
• Adhesive capsulitis (frozen shoulder)
• Acromioclavicular joint injury

Mechanism/s
The shoulder joint is a complex structure. Its components include the humeral head, glenoid fossa, acromion, clavicle, scapula and surrounding soft tissue structures. Under normal circumstances the shoulder joint is capable of a vast range of movement. Apley’s scratch test assesses glenohumeral abduction, adduction, flexion, extension, internal rotation and external rotation. Tenderness or limited range of movement suggests injury to one or more components of the shoulder joint.

Sign value
Apley’s scratch test is a useful component of the general shoulder exam but has limited utility for a specific diagnosis. The position of the shoulder at which tenderness or limited range of movement occurs should be noted. In the patient with an abnormal Apley’s scratch test, further diagnostic manoeuvres should be performed to narrow the differential diagnosis.

Description
Apley’s scratch test is a general range of movement assessment of the shoulder joint (i.e. glenohumeral, acromioclavicular, sternoclavicular and scapulothoracic joints). The patient is instructed to touch the unaffected shoulder anteriorly and posteriorly (behind their head), and touch the inferior scapula posteriorly (behind their back). Tenderness and/or limited range of movement while performing these movements is considered an abnormal test.7

FIGURE 1.3
One of three manoeuvres of Apley’s scratch test

Apparent leg length inequality (functional leg length)

Description
A disparity between the relative distance from the umbilicus to the medial malleolus of each leg.8 By definition it implies asymmetry of the lower extremities in the absence of a bony abnormality. (See ‘True leg length inequality’ in this chapter.)

Condition/s associated with
- Altered foot mechanics
- Adaptive shortening of soft tissues
- Joint contractures
- Ligamentous laxity
- Axial malalignments

Mechanism/s
An apparent or functional leg length inequality may occur at any point from the pelvis to the foot.8

Ligamentous laxity
The ligaments on one side (e.g. in the hip joint) may be more flexible or longer than their counterparts, making the femur sit lower in the joint capsule.

Joint contracture
A joint contracture impairs full range of movement. If the knee joint is contracted in a flexed position, the length of the affected side will be less than the opposite leg during maximal attempted extension.

Altered foot mechanics
Excessive pronation of the foot eventuates in and/or may be accompanied by a decreased arch height compared to the ‘normal’ foot, resulting in a functionally shorter limb.8
Sign value
The distance (anywhere from 3–22 mm) at which apparent leg length inequality results in a clinically significant effect is controversial. The test should be interpreted in relation to the patient’s history and full gait assessment.
Apprehension test

- Rotator cuff muscle injury
- Glenoid labrum injury
- Glenoid defect (e.g. Bankart’s fracture)
- Humeral head defect (e.g. Hill–Sachs fracture)

Less common – atraumatic
- Connective tissue disorder: Ehlers–Danlos syndrome, Marfan’s syndrome
- Congenital absence of glenoid

Mechanism/s
Glenohumeral joint instability is caused by dysfunction of the bony and/or soft tissue structures that maintain joint stability: glenoid, humeral head, joint capsule, capsuloligamentous or glenohumeral ligaments, labrum, and rotator cuff muscles. The shoulder joint is susceptible to instability due to its inherent mobility and complex soft tissue structures responsible for stability.

In the apprehension test, the joint is placed into a position vulnerable to instability. It is the typical position precipitating traumatic anterior shoulder dislocation. For this reason, a significant number of healthy patients will experience apprehension during this manoeuvre.

Sign value
T’Jonck L et al. reported a sensitivity of 88.0%, specificity of 50%, positive likelihood ratio of 1.8 and negative likelihood ratio of 0.23.10

The apprehension test for glenohumeral joint instability is a moderately useful screening test. Based on available data, the test has limited utility to rule in the diagnosis. It is not used in the setting of acute anterior shoulder dislocation.

Description
The apprehension test is an assessment of glenohumeral joint instability. With the patient sitting or lying supine, the shoulder is placed into 90° abduction, 90° external rotation and 90° elbow flexion. The examiner applies pressure to the posterior aspect of the proximal humerus and attempts to move the humeral head anteriorly (see Figure 1.5). The test is positive if the patient experiences apprehension due to impending subluxation or dislocation of the glenohumeral joint.9

Condition/s associated with
More common – traumatic
- Recurrent glenohumeral joint subluxation or dislocation
Apprehension–relocation test (Fowler’s sign)

Description
The apprehension–relocation test is an assessment of glenohumeral joint instability. The relocation manoeuvre is typically performed following the apprehension test (see ‘Apprehension test’). With the patient sitting or lying supine, the shoulder is placed into 90° abduction, 90° external rotation and 90° elbow flexion. The examiner applies pressure to the anterior aspect of the proximal humerus and attempts to move the humeral head posteriorly. The test is positive if the patient experiences relief of apprehension (i.e. no longer feels impending shoulder dislocation).

Condition/s associated with
- Recurrent glenohumeral joint subluxation or dislocation
- Rotator cuff muscle injury
- Glenoid labrum injury
- Glenoid defect (e.g. Bankart’s fracture)
- Humeral head defect (e.g. Hill–Sachs fracture)

Less common – atraumatic
- Connective tissue disorder: Ehlers–Danlos syndrome, Marfan’s syndrome
- Congenital absence of glenoid

Mechanism/s
The underlying anatomy and causes of glenohumeral joint instability are outlined under ‘Apprehension test’ and apply here. In the apprehension–relocation test, symptomatic relief is due to restoration of the normal anatomical relationship of the humeral head in the glenohumeral joint.

Sign value
T’Jonck L et al. reported a sensitivity of 85%, specificity of 87%, positive likelihood ratio of 6.5 and negative likelihood ratio of 0.18.10 Lo et al. reported sensitivity of 32% and specificity of 100%.11 Speer KP et al. reported a sensitivity of 68% and specificity of 100%.12

The apprehension–relocation test is a useful screening manoeuvre for anterior glenohumeral joint instability. It appears to be more specific than the ‘apprehension test’ alone.
Bouchard’s and Heberden’s nodes

Mechanism/s
A number of studies have implicated bony osteophyte growth as the principal cause of Heberden’s and Bouchard’s nodes. Other contributing factors or theories include:

- genetic predisposition
- endochondral ossification of hypertrophied cartilage as a result of chronic osteoarthritic changes
- traction spurs growing in tendons in response to excessive tension and repetitive strain.

Sign value
Bouchard’s or Heberden’s nodes are a classical sign of interphalangeal osteoarthritis and are associated with generalised osteoarthritis. The presence of Bouchard’s and/or Heberden’s nodes is predictive of the radiographic changes of osteoarthritis.

Description
Bouchard’s nodes are bony outgrowths or nodules found over the proximal interphalangeal joints of the hands. Heberden’s nodes are similar but located over the distal interphalangeal joints.

Condition/s associated with
- Osteoarthritis
- Familial

FIGURE 1.7
Prominent Heberden’s nodes
Based on Ferri FF, Ferri’s Clinical Advisor, Philadelphia: Elsevier, 2011: Fig 1-223.
**Boutonnière deformity**

**FIGURE 1.8**
Digital extensor mechanism
A The proximal interphalangeal joint is extended by the central tendon slip (an extension of the hand’s dorsal extensor tendon); B the X is a functional representation of the fibrous interconnections between the two systems.


**FIGURE 1.9**
Pathoanatomy of boutonnière deformity
The sequence is: rupture of the central tendon slip, which then simultaneously pulls on the lateral bands, pulling the DIP joint into hyperextension and the PIP into flexion.

Description
Used to describe a deformity of the resting finger in which the proximal interphalangeal (PIP) joint is flexed and the distal interphalangeal (DIP) joint is hyperextended.

Condition/s associated with
- Inflammatory arthropathy (e.g. rheumatoid arthritis)
- Central slip extensor tendon injury

Mechanism/s
Disruption or avulsion of the central slip extensor tendon and volar migration of the lateral bands of the extensor tendon mechanism result in PIP flexion and DIP extension. The sign derives its name from the appearance of the central tendon slip, which was thought to resemble a buttonhole, or boutonnière in French, when torn.

The central tendon slip attaches to the dorsal aspect of the middle phalanx. Its main function is to maintain PIP extension and stabilise the extensor tendon apparatus. If the central tendon is disrupted or avulsed (torn off the base of the middle phalanx), the actions of the lateral bands and flexor digitorum profundus are unopposed, resulting in resting PIP flexion and DIP hyperextension.

Inflammatory arthropathy (e.g. rheumatoid arthritis)
Pannus in the PIP joint (which may be present in rheumatoid arthritis) can damage the central slip tendon. Chronic inflammation and synovitis of the joint may result in persistent PIP flexion and gradual elongation of the central slip tendon. Subsequent volar migration of the lateral bands results in the characteristic deformity.

Trauma
Forced flexion of an extended PIP joint, crush injury or penetrating injury may result in avulsion of the central tendon slip. Typically, the degree of deformity increases in the days following the injury. Acutely, the deformity may be subtle.

Sign value
A boutonnière deformity is classically associated with rheumatoid arthritis occurring in up to 50% of patients with the disease.

In a patient with blunt or penetrating trauma, the presence of a boutonnière deformity should be considered evidence of a central slip extensor tendon injury.
Bulge/wipe/stroke test

**More common**
- Osteoarthritis
- Rheumatoid arthritis
- Haemoarthrosis – trauma, coagulopathy
- Gout
- Infection – septic arthritis, gonococcal arthritis, transient synovitis

**Less common**
- Pseudogout (calcium pyrophosphate deposition disease)
- Tumour

**Mechanism/s**
Mechanical manipulation of excess fluid in the synovial joint capsule results in visible fluid shift. The wipe or bulge test displaces synovial fluid from one part of the synovial joint to another, thus suggesting the presence of a joint effusion as the cause of knee swelling.

**Sign value**
Limited evidence has been gathered on the value of this test as an individual sign. Some authors report that this test may pick up on as little as 4–8 mL of swelling. An effusion in the absence of acute traumatic injury or systemic disease is most commonly due to osteoarthritis.

Gogus F et al.27 reported the wipe test as having a sensitivity of 11–33% and specificity of 66–92% for identifying the presence of a knee effusion. Emphasis should be placed upon identifying a joint effusion in the setting of septic arthritis, an orthopaedic emergency.

**Description**
The bulge, wipe or stroke test is used to assess for knee joint effusion. With the patient supine and their knee extended, the examiner ‘swipes’ the medial aspect of the knee joint to displace fluid into the superolateral aspect of the synovial compartment, and then swipes the lateral side looking for a visible fluid shift. The test is positive if the examiner sees a wave of fluid.

**Condition/s associated with**
Any condition causing a knee effusion.
Butterfly rash (malar rash)

Mechanism/s
The exact mechanism is unclear. However, like the underlying disorder in SLE, it is thought to result from an autoimmune reaction caused by genetic, environmental and immunological factors.

Factors shown to be involved include:

- A genetic predisposition to ineffective or deficient complement, leading to a failure to clear immune complexes of apoptotic cells, which in turn increases the chance of the development of autoimmunity.
- Sunlight has been shown to damage and/or induce apoptosis of keratinocyte proteins in the epidermis and can stimulate autoantibody production. Sunlight may also increase the chance of keratinocytes being destroyed by complement and antibody-dependent mechanisms.
- Altered cellular and humoral immunity reactions have been seen in studies reviewing cutaneous manifestations of lupus. It is likely that a combination of these factors leads to immune deposition in the skin, damage, oedema and the characteristic malar rash.

Description
A red or purple, macular, mildly scaly rash that is seen over the bridge of the nose and cheeks. The shape of the rash can somewhat resemble a butterfly. The rash spares the nasolabial folds, which helps distinguish it from other rashes (e.g. rosacea). It is also photosensitive.

Condition/s associated with
Common
- Systemic lupus erythematosus (SLE)
- Drug-induced lupus erythematosus
- Dermatomyositis

FIGURE 1.11
Malar rash of SLE
Reproduced, with permission, from Goldman L, Ausiello D, Cecil Medicine, 23rd edn, Philadelphia: Saunders, 2007: Fig 287-3.
Butterfly rash (malar rash)

**FIGURE 1.12**
Mechanism of malar rash

**Sign value**
The malar rash is seen in approximately 40% of patients with SLE. It has a sensitivity of 57% and specificity of 96% for SLE. Its absence does not exclude the diagnosis.
Calcinosis/calcinosis cutis

-Calcinosis/calcinosis cutis

-Systemic sclerosis
-Burns

-Metastatic
-Due to hypercalcaemia or hyperphosphataemia of any cause
-Chronic renal failure – most common
-Excess vitamin D
-Primary hyperparathyroidism – rare
-Paraneoplastic hypercalcaemia
-Destructive bone disease (e.g. Paget’s disease)

-Iatrogenic
-Calcium gluconate injections
-Tumour lysis syndrome

-Idiopathic
-Calciphylaxis
-End-stage renal disease
-Altered calcium metabolism

Mechanism/s

Dystrophic calcinosis
-Dystrophic calcinosis occurs when crystals of calcium phosphate or hydroxyapatite are deposited in the skin secondary to inflammation, tissue damage and degeneration. Calcium and phosphate levels are usually normal. Proposed mechanisms include:

- High local levels of alkaline phosphatase break down a pyrophosphate that normally inhibits calcification.
- Tissue breakdown may lead to denatured proteins that bind to phosphate. These phosphate–protein compounds may react with calcium and thus provide a nidus for calcification.

Description
-Calcinosis refers to the formation or deposition of calcium in soft tissue. Calcinosis cutis more specifically refers to calcium deposits in the skin.

Condition/s associated with
-Conditions associated with calcinosis may be classified as dystrophic, metastatic, iatrogenic, idiopathic or calciphylaxis.
- Dystrophic calcinosis
  - Scleroderma
  - Dermatomyositis
  - SLE
**Metastatic calcinosis**

Abnormal calcium or phosphate metabolism with high levels of either or both is present. Excess calcium and/or phosphate allows for the formation and precipitation of calcium salts.

In chronic renal failure a number of mechanisms lead to altered phosphate and calcium metabolism:

- Decreased renal excretion of phosphate leads to hyperphosphataemia.
- Hyperphosphataemia results in a compensatory rise in parathyroid hormone (PTH) in an attempt to excrete phosphate. The rise in PTH results in an increase in phosphate absorption from the gut and also mobilises calcium from the bones, resulting in more calcium being available to precipitate with phosphate.

- Vitamin D deficiency owing to renal failure worsens initial hypocalcaemia and, therefore, further stimulates secondary hyperparathyroidism.

**Iatrogenic**

Intravenous administration of calcium or phosphate may cause local extravasation and precipitation of hydroxyapatite in surrounding tissue. Inflammation of the surrounding tissue secondary to the injection may also cause calcium and protein release, contributing to precipitation.

**Idiopathic**

Occurs in the absence of tissue injury or systemic metabolic disturbance.

**Sign value**

There is very limited evidence on this sign and it is rarely seen in isolation. If identified, further investigation is warranted.
Charcot foot

Description
A progressive destructive arthropathy of the ankle and foot. In its early stages, it may present as unilateral foot oedema following minor trauma. In advanced disease, significant destruction of bones and joints may occur (particularly in the midfoot), resulting in collapse of the plantar arch and development of ‘rocker-bottom foot’.

Condition/s associated with
Conditions resulting in sensory neuropathy:
- Diabetes mellitus – most common
- Syphilis – original description by Charcot

Mechanism/s
In neurotraumatic theory, peripheral neuropathy caused by diabetes leads to decreased pain sensation and impaired proprioception. Thus, if an acute injury occurs (e.g. microfracture, subluxation or fracture), the patient feels little or no pain and does not ‘guard’ the foot when mobilising. This leads to a destructive cycle of continued loading on the injured foot and progressive damage.

Under the inflammatory theory, when the same local insult occurs (microfracture, subluxation or fracture), inflammatory cytokines are released, including TNF-α and interleukin-1β. These two cytokines have been shown to increase activation of RANK ligand (RANKL), which in turn increases the transcription factor nuclear factor-κB (NF-κB). The net result of this is stimulation of the maturation of osteoclasts, which further eat away at bone. This predisposes the patient to engage in another vicious cycle of further fractures, inflammation, abnormal weight loading and osteolysis.

Regardless of underlying contributing factors, the RANKL/OPG (osteoprotegerin) pathway is thought to be a common denominator. RANKL is a member of the tumour necrosis factor (TNF) superfamily, and OPG is the competitive protein of RANKL. The process of bone

FIGURE 1.14
Charcot foot
A, B The classic rocker-bottom Charcot foot, with collapse and then reversal of the longitudinal arch; C loss of the normal calcaneal pitch, or angle relative to the floor, in patients with Charcot collapse of the arch.
Charcot foot

osteopenia due to unrestricted RANKL activity.\textsuperscript{35}

Other contributing factors include:

- Sympathetic denervation in distal limbs leads to increased peripheral blood flow – hyperaemia and more inflammation.\textsuperscript{36}

- Pre-existing osteopaenia has been seen in both type 1 and type 2 diabetes via a number of mechanisms,\textsuperscript{36} and this predisposes the diabetic patient to microfracture.

- Abnormal loading mechanics.
  - Oxidative stress and the formation of reactive oxidant species (ROS) with local dysregulation of immunoinflammatory processes.
  - AGE/RAGE pathway – hyperglycaemia generates AGEs. These promote change in both intra- and extracellular proteins, which become defective and lose functionality. Binding of AGE to receptors for AGE (RAGE) accelerates ROS production, activating NF-κB. An increase in AGE-modified collagen has been shown to affect osteoblastic cell differentiation and function in vitro. This may play a role in the pathogenesis of osteopaenia, which is present in patients with poorly controlled diabetes.
  - PI3 kinase pathway – PI3 kinase normally impedes inflammation via enhanced nitric oxide (NO) production. NO may cause apoptosis of osteoclast progenitors as well as inhibit the resorptive action of mature osteoclasts. The PI3 kinase and NO pathway may be compromised in diabetes.
  - Altered Wnt/β-catenin pathway (an important bone anabolic pathway).
FIGURE 1.16  
Detailed mechanism behind the development of Charcot foot 

- Altered fibroblast-like synoviocytes (FLS).
- OPG genetic polymorphisms.35

Sign value
Patients with Charcot foot are at higher risk of diabetic foot ulcers (affecting up to 50% of patients)37,38 and amputation.36

The presence of foot pain, heat and/or swelling in the diabetic patient needs immediate attention including referral to diabetic/high-risk foot clinics or orthopaedic and diabetic specialist services.

oxPTM: oxidative post-translational modifications  
BMD: bone mineral density  
FLS: fibroblast-like synoviocytes  
MMPs: matrix metalloproteinases  
DNOAP: diabetic neuropathic osteoarthropathy
Crepitus

Description
Grating, crunching, popping or crackling sounds heard and/or felt over joints during passive range of motion examination.

Condition/s associated with
- Arthropathy
  » Osteoarthritis
  » Rheumatoid arthritis
- Trauma
  » Cartilaginous injury – meniscal injury, labral injury
  » Ligamentous injury – anterior cruciate ligament
  » Fracture

General mechanism/s
Crepitus of the joints is caused when two rough surfaces grind against one another.

Rheumatoid/osteoarthritis
In both rheumatoid arthritis and osteoarthritis arthritis, degeneration of the articular cartilage of the joint surfaces occurs, creating erosions and irregularity. Two rough surfaces moving against each other produce crepitus.

In rheumatoid arthritis, the autoimmune response and subsequent inflammation, cytokine release and pannus formation cause destruction of cartilage.

In osteoarthritis, repetitive strain with loss of glycosaminoglycans and activation of matrix metalloproteinases (MMPs) is principally responsible for damage.

Sign value
Altman R et al. reported crepitus had a sensitivity of 89%, specificity of 58%, positive likelihood ratio of 3.0 and negative likelihood ratio of 0.2 for predicting osteoarthritis of the knee. Crepitus is common in patients with osteoarthritis. Crepitus alone has limited diagnostic value, due to its presence in other common disease states.
**Dropped arm test**

**Description**
With the patient upright, the examiner passively moves the patient’s arm to 90° of abduction. Then the patient is asked to slowly lower the arm to the anatomical position. A positive test occurs if the patient is unable to perform the action due to pain or if the arm just ‘drops’ to the side.

**Condition/s associated with**
- Rotator cuff muscle injury (e.g. supraspinatus muscle)
- Subacromial impingement
- Neurogenic weakness
- Suprascapular nerve palsy
- Axillary nerve palsy
- C5 radiculopathy

**Mechanism/s**
Abduction of the arm from 0° to 90° is dependent upon the supraspinatus and deltoid muscles. The supraspinatus is responsible for the first 15° of motion. The deltoid muscle is responsible for movement beyond 15°. Therefore, if a rotator cuff tear (e.g. supraspinatus muscle tear) or subacromial impingement is present, the ability of the arm to maintain abduction is impaired.

**Sign value**
Murrell GAC et al. and Dinnes J et al. reported a sensitivity of 10% and specificity of 98%, and a calculated positive likelihood ratio greater than 10 for rotator muscle tear. Park HB et al. reported a sensitivity of 27%, specificity of 88%, positive likelihood ratio of 2.3 and negative likelihood ratio of 0.8 for subacromial impingement.

When positive, the dropped arm test significantly increases the probability of rotator cuff muscle tear (supraspinatus muscle tear) or subacromial impingement. A negative test does not reliably exclude the diagnosis.
Eichhoff’s test

Description
The patient places their thumb within the palm of the examiner’s hand, who grasps it tightly. The hand is then abducted towards the ulna by the examiner (see Figure 1.18).

Condition/s associated with
- De Quervain’s tenosynovitis

Mechanism/s
De Quervain’s tenosynovitis is an inflammatory condition of the contents of the first extensor synovial compartment: the tendons of abductor pollicis longus and extensor pollicis brevis.

Repetitive strain injury or inflammatory disorders cause inflammation, leading to swelling over the radial aspect of the wrist. This narrows the space that the abductor pollicis longus and extensor pollicis brevis pass through on their way to the hand.

This manoeuvre and Finkelstein’s test involve generation of a passive distension and shear stress between the tendons and radius on its blunt styloid edge. In essence, the abductor pollicis longus and extensor pollicis brevis tendons are moved into the narrowed compartment and stretched, causing pain.

Sign value
There is limited data on the evidence of Eichhoff’s test’s diagnostic accuracy in diagnosing de Quervain’s tenosynovitis. One small study suggested Eichhoff’s test was associated with more false positives than Finkelstein’s test.44
Finkelstein’s test

Condition/s associated with

- De Quervain’s tenosynovitis

Mechanism/s

De Quervain’s tenosynovitis is an inflammatory condition of the contents of the 1st extensor synovial compartment: abductor pollicis longus and extensor pollicis brevis tendons.

Repetitive strain injury or inflammatory disorders cause inflammation that, in turn, causes swelling over the radial aspect of the wrist. This narrows the space through which the abductor pollicis longus and extensor pollicis brevis pass on their way to the hand. When performing this manoeuvre, the abductor pollicis longus and extensor pollicis brevis tendons are moved into the narrowed compartment and stretched, causing pain.45

Sign value

There is limited data on the evidence for Finkelstein’s test in diagnosing De Quervain’s tenosynovitis. De Quervain’s tenosynovitis is a clinical diagnosis.

Description

The examiner applies force at the patient’s thumb metacarpal, placing the wrist into forced ulnar deviation. Tenderness with the manoeuvre at the radial aspect of the wrist (at the abductor pollicis longus tendon or extensor pollicis brevis tendon) is considered a positive test result.

FIGURE 1.19

Finkelstein’s test

With the thumb inside the hand, the wrist is ulinarly deviated. Pain indicates a positive test.

### Gottron’s papules

**Description**
Violaceous (violet-coloured) papular rash on the dorsal aspect of the interphalangeal joints.

**Condition/s associated with**
- Dermatomyositis

**Mechanism/s**
One histological study demonstrated lymphocytic infiltration, epidermal atrophy and vacuoles in the basal layer of the skin, in addition to other findings. The mechanism is unknown.

**Sign value**
Gottron’s papules are said to be pathognomonic for dermatomyositis; however, they are not present in all patients with the disease.

**FIGURE 1.20**
Gottron’s papules
Found over bony prominences: fingers, elbows and knees. The lesions are slightly elevated, violaceous papules with slight scale.

*Reproduced, with permission, from Habif TP, Clinical Dermatology, 5th edn, Philadelphia: Mosby, 2009: Figs 17-20, 17-21.*
Hawkins’ impingement test

**Description**
With the patient upright, shoulder and elbow both flexed to 90°, the examiner internally rotates the shoulder joint. The sign is positive if tenderness is elicited (see Figure 1.22).

**Condition/s associated with**
- Rotator cuff muscle impingement
  - supraspinatus, teres minor, infraspinatus muscles
- Rotator cuff tendonitis

**Mechanism/s**
The tendons of the rotator cuff muscles pass through a narrow space between the acromion process of the scapula, bursa and the head of the humerus. Hawkins’ impingement test exacerbates narrowing in the coracoacromial space and will worsen pre-existing impingement of the tendons and muscles when present. The position advances the greater tuberosity towards or against the coracoacromial ligament. This manoeuvre will also elicit tenderness when rotator cuff tendonitis
is present, due to mechanical forces or compression on the injured tendon or muscle.\textsuperscript{49}

**Sign value**

Calis M et al. reported a sensitivity of 92\% and a specificity of 26–44\% for identifying rotator cuff tendonitis.\textsuperscript{50} Macdonald PB et al. reported a sensitivity of 83\% and a specificity of 51\% for NLR of 0.3 for rotator cuff tear.\textsuperscript{51}

A pooled analysis of 1029 patients across six studies by Alqunaee M et al.\textsuperscript{52} reported a sensitivity of 74\% and specificity of only 57\%. Given these results, the test is of little value to the examiner, while a negative test has moderate utility. Further physical examination and imaging should be used to confirm the diagnosis.
Heliotrope rash

**Condition/s associated with**
- Dermatomyositis
- Paraneoplastic syndrome

**Mechanism/s**
The mechanism is unknown but thought to be autoimmune in origin. Skin lesions demonstrate perivascular CD4 positive T-cell infiltration in the dermis.53

**Sign value**
Occurring in up to 83% of cases in a specific European region,54 the heliotrope rash is thought to be pathognomonic of dermatomyositis and should trigger further investigation.

Skin changes associated with dermatomyositis may precede muscle weakness, EMG abnormalities and elevations in creatinine phosphokinase by weeks or months.

**Description**
Usually described as a macular, confluent, purple or violaceous rash over both eyelids and periorbital tissue. It can present with or without oedema.

**FIGURE 1.23**
Heliotrope eruption seen in dermatomyositis
Reproduced, with permission, from Firestein GS, Budd RC, Harris ED et al., Kelley’s Textbook of Rheumatology, 8th edn, Philadelphia: WB Saunders, 2008: Fig 47-10.
Kyphosis

**Description**
Abnormally pronounced convex curvature of the thoracic spine as seen from the side. Kyphosis may be visible from any direction when severe. Often referred to in elderly females as the ‘dowager’s hump’.

**Condition/s associated with**

**More common**
- Osteoporosis/degenerative joint disease
- Traumatic – vertebral body fracture

**Less common**
- Ankylosing spondylitis
- Congenital
- Scheuermann kyphosis

**Mechanism/s**
Narrowing of the anterior aspect of the vertebral body is common in most forms of kyphosis.

**Osteoporosis/degenerative joint disease**
In degenerative or osteoporotic kyphosis, poor posture, mechanical straining and osteoporosis result in degeneration and/or compression fractures of the vertebrae. There is a relative loss of height of the anterior aspect of the vertebral body, leading to increased thoracic kyphosis.

**Congenital kyphosis**
Congenital kyphosis results from either a failure of formation or a failure of segmentation of the vertebral body elements. In failure of segmentation, the anterior part of the vertebral body fails to separate from the vertebral body below, resulting in anterior fusion of the anterior aspect of the vertebrae. The posterior aspect continues to grow, resulting in kyphosis.

**Scheuermann kyphosis**
Scheuermann kyphosis is a form of adolescent kyphosis. The mechanism behind Scheuermann kyphosis is multifactorial, including:
- herniation of vertebral disc material into the vertebral body, causing decreased vertebral height and increased pressure anteriorly, leading to abnormal growth and wedging of the vertebrae
- a thickened anterior ligament
- abnormal collagen matrix.
**Sign value**

Kyphosis in paediatric patients may be suggestive of congenital kyphosis, which can have serious complications and lead to significant disability if left untreated. Acute worsening in the degree of kyphosis in an elderly patient should prompt consideration of pathological fracture.
**Lachman’s test**

**FIGURE 1.25**
Lachman’s test of the anterior cruciate ligament (ACL)
With 20–30° knee flexion, the tibia is moved forward on the femur to test the integrity of the ACL.

**Description**
The patient lies supine with the knee at 20–30° flexion. The examiner immobilises the femur just above the knee with one hand and attempts to pull the proximal tibia anteriorly with the other hand; the thumb is placed upon the tibial tuberosity. The test is positive if there is anterior movement of the tibia without an abrupt stop.

**Condition/s associated with**
- Anterior cruciate ligament (ACL) injury

**Mechanism/s**
The ACL arises from the anterior aspect of the tibial plateau and inserts into the medial aspect of the lateral femoral condyle. It limits anterior movement of the tibia on the femur. If the ACL is intact, the tibia should not have significant forward movement; if it is ruptured, there will be inappropriate anterior movement of the tibia and knee joint instability.

**Sign value**
A review by McGee of five studies reported a sensitivity of 48–96%, a specificity of 90–99%, a positive likelihood ratio of 17.0 and a negative likelihood ratio of 0.2.2

A positive Lachman’s test is strongly predictive of ACL injury (+LR 17.0).2

In a patient with a high clinical suspicion of ACL injury despite a negative Lachman’s test (−LR 0.2),2 further evaluation is necessary (e.g. interval re-examination, MRI). In general, Lachman’s test is considered the better examination manoeuvre for ACL injury when compared with the anterior drawer sign and pivot-shift test.57 A more recent systematic review of six studies found a sensitivity of 81–89%, specificity of 91–100%, with a +LR of up to 42, but with wide confidence intervals.58
**Livedo reticularis**

**Description**
A macular, bluish/purple discolouration of the skin that has a lacy or net-like appearance.

**Condition/s associated with**

**More common**
- Primary or idiopathic livedo reticularis (LR)
- Hypothermia
- Elderly

**Less common**
- Secondary LR
  Present in numerous disorders including:
  - Hypercoagulable state
    - Antiphospholipid syndrome
    - Cryoglobulinaemia
    - Multiple myeloma
    - DVT
  - Microangiopathy/microangiopathic haemolytic anaemia (MAHA)
    - Thrombotic/thrombocytopenic purpura (TTP)
    - Haemolytic uraemic syndrome
    - Disseminated intravascular coagulation
  - Vasculitis/arteriopathy
    - Snedden’s syndrome
    - Calciphylaxis
  - Connective tissue disorders (e.g. SLE, dermatomyositis)
  - Embolisation (e.g. cholesterol embolisation syndrome)
  - Drug side effect
    - Amantadine
    - Quinine

**General mechanism/s**
Arterioles arising from the dermis divide to form a capillary bed. These capillaries then drain into the venules of the venous plexus. Livedo reticularis results from increased visibility of the venules of the skin. Venodilatation of superficial venules and deoxygenation of blood in the plexus are two main factors.59

In general, venodilatation is caused by altered autonomic nervous system function, circulating factors that cause
Livedo reticularis

Venodilatation or in response to local hypoxia. Venodilatation results in engorged venules, making them larger and thus easier to see through the skin.

Deoxygenation is principally caused by decreased cutaneous perfusion,\(^5^9\) which can be the result of decreased arteriolar inflow\(^6^0\) or decreased venous outflow. These are caused by:

- *decreased arteriolar inflow* – vasospasm due to cold, autonomic nervous system activity, arterial thrombosis or increased blood viscosity
- *decreased venous outflow* – venous thrombosis, increased blood viscosity.

**Primary or idiopathic livedo reticularis**

LR without the presence of underlying disease or hypothermia is associated with spontaneous arteriolar vasospasm, which decreases oxygenated blood inflow, causing tissue hypoxia and increased deoxygenation of venous blood.\(^6^1\)

### Hypothermia (autonomic nervous system)

The normal physiological response to hypothermia is arteriolar vasospasm. This decreases arteriolar blood flow, local tissue hypoxia and venous plexus dilatation.

### Elderly

The previous mechanisms apply to elderly patients, but with the added element of *thinning of the skin* that occurs with old age. This delicate and relatively translucent skin makes it more likely that the venous plexus will be visible.

### Anti-phospholipid syndrome

Anti-phospholipid syndrome is associated with arterial and venous thrombosis, resulting in increased tissue hypoxia and venule dilatation (due to venous stasis).

**FIGURE 1.27**

Mechanism of livedo reticularis
**Amantadine**
This is an antiviral and anti-Parkinson’s medication. Its involvement in LR is thought to be related to a combination of catecholamine-induced vasospasm and the effects of amantadine on N-methyl-D aspartic acid receptors in the skin.

**Cryoglobulinaemia**
Cryoglobulins are proteins that become insoluble and precipitate when the temperature drops. Increasing viscosity results in stasis and tissue hypoxia. In addition, cryoglobulinaemia is associated with microvascular thrombosis.

**Sign value**
Primary or idiopathic LR is a diagnosis of exclusion; a secondary cause should be sought.
- LR has been shown to have a significant relationship with anti-phospholipid syndrome, with up to 40% of patients presenting with LR as the first sign.\(^{62}\)
- Livedo reticularis in a patient with SLE is associated with the development of neuropsychiatric symptoms.
McMurray’s test

Condition/s associated with

- Meniscal injury

Mechanism/s

By extending the flexed knee while applying external or internal rotation of the leg, the femoral condyle is moved over the tibia and meniscus. Crepitus will be present when the femur moves over the torn meniscal fragment.

Sign value

In a review of two studies McGee reported a sensitivity of 17–29%, a specificity of 96–98%, a positive likelihood ratio of 8.0 and a negative likelihood ratio of 0.2 for detecting meniscal injury.\textsuperscript{2} In a meta-analysis Scholten RJPM et al. reported a sensitivity of 10–63% and specificity of 57–98%\textsuperscript{63}

In the setting of acute knee joint injury this manoeuvre is often very painful. These patients are often instructed to rest, ice, elevate and immobilise the affected knee, and return at a later date for repeat examination.

Description

This test begins with the patient lying supine and knee flexed to 90°. The medial meniscus is palpated with one hand on the posteromedial edge of the joint, while the other hand holds the ankle and performs external rotation. The lateral meniscus is assessed with one hand over the posterolateral aspect of the joint while the leg is internally rotated. The test is positive if ‘clunking’ is felt as the meniscal fragment is moved against the femur.

Physical examination of knee pathology can be challenging, especially in the presence of acute pain. A summary of the value of a number of tests and history is provided in Table 1.1. 

FIGURE 1.28
McMurray’s test
**TABLE 1.1**  
Key physical examination tests for knee pathology versus MRI

<table>
<thead>
<tr>
<th>Location</th>
<th>Thessaly test</th>
<th>McMurray's test</th>
<th>Apley's test</th>
<th>Joint line tenderness test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sensitivity</strong></td>
<td>0.62 (0.52 to 0.71)</td>
<td>0.63 (0.53 to 0.72)</td>
<td>0.43 (0.34 to 0.52)</td>
<td>0.83 (0.75 to 0.89)</td>
</tr>
<tr>
<td><strong>Specificity</strong></td>
<td>0.55 (0.44 to 0.66)</td>
<td>0.63 (0.53 to 0.73)</td>
<td>0.72 (0.61 to 0.80)</td>
<td>0.39 (0.29 to 0.49)</td>
</tr>
<tr>
<td><strong>LR+</strong></td>
<td>1.38 (1.05 to 1.81)</td>
<td>1.72 (1.26 to 2.33)</td>
<td>1.52 (1.04 to 2.21)</td>
<td>1.36 (1.14 to 1.62)</td>
</tr>
<tr>
<td><strong>LR−</strong></td>
<td>0.69 (0.51 to 0.93)</td>
<td>0.50 (0.44 to 0.78)</td>
<td>0.80 (0.65 to 0.97)</td>
<td>0.44 (0.28 to 0.69)</td>
</tr>
<tr>
<td><strong>OR</strong></td>
<td>2.00 (1.14 to 3.50)</td>
<td>2.93 (1.66 to 5.19)</td>
<td>1.91 (1.08 to 3.38)</td>
<td>3.08 (1.67 to 5.67)</td>
</tr>
<tr>
<td><strong>PPV</strong></td>
<td>0.55 (0.46 to 0.64)</td>
<td>0.57 (0.48 to 0.66)</td>
<td>0.59 (0.48 to 0.69)</td>
<td>0.50 (0.43 to 0.57)</td>
</tr>
<tr>
<td><strong>NPV</strong></td>
<td>0.52 (0.41 to 0.62)</td>
<td>0.57 (0.47 to 0.67)</td>
<td>0.49 (0.41 to 0.58)</td>
<td>0.64 (0.51 to 0.76)</td>
</tr>
</tbody>
</table>

\[ LR− = \text{likelihood ratio for negative test}; \ LR+ = \text{likelihood ratio for positive test}; \ OR = \text{odds ratio}. \]

\[ p\text{-values are based on a chi-squared distribution to assess whether or not the sensitivities, specificities, PPV or NPV are equal along the four physical tests and clinical history.}\]
Neer’s impingement test

Description
The patient’s shoulder is placed into 90° flexion and internal rotation, with the elbow in full extension. The examiner then stabilises the scapula with one hand and passively moves the shoulder joint to 180° flexion with the other hand. If tenderness is elicited at the anterolateral aspect of the shoulder joint, the test is positive.

Condition/s associated with
• Rotator cuff impingement/tendonitis
  » Supraspinatus
  » Infraspinatus
• Subacromial bursitis

Mechanism/s
The supraspinatus tendon and infraspinatus tendon transverse a narrow passage between the acromion, coracoacromial ligament and the humeral head before they insert into the proximal humerus. Narrowing of this space due to abnormalities of the acromion, acquired weakness in the posterior rotator cuff muscles (supraspinatus, infraspinatus, teres minor), or muscle hypertrophy in overuse, may cause impingement and inflammation.

In Neer’s test, passive shoulder flexion from 90° to 180° exacerbates underlying narrowing of the passage made up by the acromion, coracoacromial ligament and humeral head, resulting in compression of its contents (i.e. the supraspinatus and infraspinatus tendons).

Sign value
Calis M et al. reported a sensitivity of 88.7%, a specificity of 30.5%, a positive predictive value of 75.9% and a negative predictive value of 52.3%. Macdonald PB et al. reported a sensitivity of 75%, a specificity of 47.5%, a positive predictive value of 36% and a negative predictive value.
Neer’s impingement test

of 82.9% for the test to identify patients with subacromial bursitis. The same study reported a sensitivity of 83.3%, a specificity of 50.8%, a positive predictive value of 40.0% and a negative predictive value of 88.6% for the test to identify patients with rotator cuff tendon impingement. Neer’s impingement test is somewhat useful to exclude rotator cuff tendon impingement with a negative test. The test has limited potential to identify patients with rotator cuff impingement, because many painful shoulder conditions may result in a ‘positive’ test.
Patellar apprehension test

**Condition/s associated with**

- Patellofemoral instability

**Mechanism/s**

The patella normally rests in the patellofemoral groove, sliding up and down through this groove during knee flexion and extension. It is kept in place by the quadriceps tendon and patellar ligament, as well as other supporting structures. If these structures are damaged the patella is susceptible to lateral instability.

By displacing the patella laterally during attempted active knee extension, the examiner is deliberately attempting to displace the patella out of the groove to assess for patellofemoral instability.

**Sign value**

There is limited evidence supporting the use of this test. One small study reported a sensitivity of 39%. This is in contrast to another small study of 51 patients reporting a sensitivity of 100%, specificity of 88%, PPV 89%, NPV 100%, +LR 8.3 and −LR of 0 for predicting patellar instability.

**Description**

With the patient supine and knee slightly flexed (20–30°), the examiner applies pressure, attempting to displace the patella laterally, while the patient is instructed to straighten the knee. The test is positive if apprehension is elicited due to impending lateral patella instability/dislocation or tenderness.

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Reprinted, with permission, from DeLee JC, Drez D, Miller MD, DeLee and Drez’s Orthopaedic Sports Medicine, 3rd edn, Philadelphia: Saunders, 2009: Fig 22C1-5.
Patellar tap

• Gout
• Infection – septic arthritis, gonococcal arthritis, transient synovitis

Less common
• Pseudogout (calcium pyrophosphate deposition disease)
• Tumour

Mechanism/s
In the setting of a moderate-to-large joint effusion, the patella is displaced anteriorly relative to the distal femur at the knee joint. Application of pressure to the suprapatellar pouch accentuates anterior patellar displacement. When pushed or ‘tapped’, the patella can be felt to float down through the fluid and collide against the distal femur. In a normal knee, the patella and femur are in close contact and therefore cannot be made to click together.

Sign value
Gogus F et al. reported a sensitivity of 0–55% with specificity of 46–92%, depending on the clinician completing the examination. A larger study by Kastelein M et al., looking at effusions in traumatic knee injury, reported a sensitivity of 83%, a specificity of 49%, a positive likelihood ratio of 1.6 and a negative likelihood ratio of 0.3. The same study indicated that, although the bulge test may be able to detect a smaller effusion, the patellar test is more likely to be associated with a clinically important effusion.

The available data, limited by heterogeneity, suggests limited utility of the patellar tap. Emphasis should be placed upon the suspected aetiology of a joint effusion, such as septic arthritis (an orthopaedic emergency).

Description
With the patient lying supine with the leg extended, pressure is applied over the suprapatellar pouch, displacing synovial fluid forward towards the patella. With the other hand the patella is pushed or tapped downwards. A palpable click as the patella hits the underlying bone is a positive test. Occasionally the patella will also ‘bounce’ back up to the examiner’s fingers.

Condition/s associated with
Any condition causing a knee effusion:

More common
• Osteoarthritis
• Rheumatoid arthritis
• Haemarthrosis – trauma, coagulopathy

FIGURE 1.31
Patellar tap
Note that the left hand squeezes the suprapatellar pouch (A), while the other ‘taps’ the patella (B).
**Patrick’s test (FABER test)**

**Condition/s associated with**
Any cause of sacroiliitis including, but not limited to:

**More common**
- Osteoarthritis/degenerative joint disease
- Trauma

**Less common**
- HLA-B27 spondyloarthropathy
  - Ankylosing spondylitis
  - Psoriatic arthritis
  - Reactive arthritis
  - Enteropathic arthritis (associated with inflammatory bowel disease)
- Infectious sacroiliitis

**Description**
With the patient lying supine, the knee is flexed to 90° and the foot placed on the opposite knee. The flexed knee is then pushed down by the examiner to produce external rotation of the affected hip. If tenderness is elicited in the area of the buttocks, the test is considered positive for sacroiliitis, whereas tenderness in the groin suggests hip joint pathology.

FABER is a mnemonic for the movements of the hip during the test (i.e. Flexion, Abduction, External Rotation).

**Mechanism/s**
Manipulation of the hip with flexion, abduction and external rotation results in distraction of the inflamed sacroiliac joint,

**Sign value**
Limited sound methodological studies exist for the FABER test. Individual studies, however, have reported a sensitivity of 69–77% and a specificity of 100%. For detecting labral tears of the hip, the FABER test has poor diagnostic accuracy: sensitivity 65%, specificity 19% and positive likelihood ratio 0.80.
Phalen’s sign

Description
The patient puts their wrists into a position of 90° flexion and presses them into one another for 1 minute. The presence of paraesthesias and/or numbness in the distribution of the median nerve is a positive test.

Condition/s associated with
• Carpal tunnel syndrome (the most common causes of median nerve palsy)

Mechanism/s
In carpal tunnel syndrome, crowding within the carpal tunnel or repetitive strain injury results in chronic inflammation of the median nerve. When the wrist is flexed, the flexor retinaculum, which acts as a pulley on the digital flexor tendons, pulls them down onto the median nerve and acutely increases pressure on the nerve. This manoeuvre increases pressure within the carpal tunnel, further irritating the nerve, thus worsening neuropathic sensory abnormalities.

Sign value
D’Arcy CA et al. reported a wide range of a sensitivity of 10–91%, a specificity of 33–76%, a positive likelihood ratio of 1.1–2.1 and a negative likelihood ratio of 0.3–1.0. However, a more recent study, also comparing clinical tests with nerve conduction, reported a sensitivity of 74% across all severities of carpal tunnel syndrome, with a specificity of 74%. The sensitivity in identifying mild to moderate severity CTS was 92%.

FIGURE 1.33
Hand placement in Phalen’s test

FIGURE 1.34
Median nerve distribution of paraesthesias in the hand
Posterior drawer test

Description
With the patient and examiner in the same position as for the anterior drawer test, the examiner pushes the lower leg posteriorly, gently moving the proximal tibia backwards. If there is a tear in the posterior cruciate ligament, there will be significant abnormal posterior movement with a soft endpoint (see Figure 1.35).

Condition/s associated with
- Posterior cruciate ligament (PCL) tear

Mechanism/s
The PCL attaches the posterior aspect of the tibia to the lateral medial condyle (see Figure 1.36). It is a short ligament which restricts anterior movement of the femur on the tibia and posterior movement of the tibia. When the posterior cruciate is damaged or deficient, abnormal movement is possible. The posterior drawer test is used to assess the integrity of the PCL.

Sign value
The posterior drawer test is a useful sign, with reported sensitivity of 90–95%, specificity of 99% and a +LR of 97.8 if present.76
FIGURE 1.36
Anatomy of the knee showing posterior cruciate ligament attachments and relationships
Proximal weakness/proximal myopathy

Description
Proximal myopathy is a muscle disorder which results in proximal muscle group weakness (e.g. shoulder: pectoralis major, deltoid, biceps; hip: gluteal, quadriceps, iliopsoas, adductor). Proximal weakness is rapidly assessed by asking the patient to rise from a seated position and/or perform the motion of hanging washing on a clothesline. A complete assessment of power should be performed.

Condition/s associated with
• Inflammatory myopathy
  » Polymyositis
  » Dermatomyositis
• Endocrine myopathy
  » Hyperthyroidism – see Chapter 7, ‘Endocrinological signs’
  » Hypothyroidism – see Chapter 7, ‘Endocrinological signs’
  » Hyperparathyroidism – see Chapter 7, ‘Endocrinological signs’
• Systemic disorders
  » Systemic lupus erythematosus (SLE)
  » Rheumatoid arthritis
• Genetic
  » Myotonic dystrophy
  » Spinal muscular atrophy
• Other
  » Myasthenia gravis
  » Polymyalgia rheumatica

TABLE 1.2
Mechanisms of inflammatory myopathies

<table>
<thead>
<tr>
<th>Disease</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polymyositis</td>
<td>T-cell (in particular CD8) and macrophage destruction of muscle fibres</td>
</tr>
<tr>
<td>Dermatomyositis</td>
<td>Complement and antibody destruction of microvasculature; the deposition of complement and antibody complexes leads to inflammation and destruction of muscle fibres and hence weakness</td>
</tr>
</tbody>
</table>

Mechanism/s
Inflammatory myopathies
Inflammatory myopathies result in immunologically mediated inflammation and destruction of skeletal muscle, causing weakness (see Table 1.2).

Systemic disorders
Proximal myopathy may present in a number of systemic rheumatological disorders such as SLE and RA. It is thought that circulating antibody complexes, deposited in tissues and/or targeted at muscles, damage muscle fibres, resulting in weakness.

Sign value
Patients with gradual-onset progressive symmetric proximal muscle weakness should be evaluated for a myopathy.
Psoriatic nails/psoriatic nail dystrophy

**FIGURE 1.37**
Nail dystrophic changes
A Nail pitting; B onycholysis; C severe destructive change with nail loss and pustule formation.

*Reproduced, with permission, from Firestein GS, Budd RC, Harris ED et al., Kelley’s Textbook of Rheumatology, 8th edn, Philadelphia: WB Saunders, 2008: Fig 72-3.*

**Description**
Psoriatic nail changes refer to a number of different abnormalities seen in the nails rather than just one sign. Changes include:

- Pitting of the nail plate
- Subungual hyperkeratosis under the nail plate
- Onycholysis (nail lifting) and changes in nail shape
- ‘Oil drops’ and ‘salmon patches’
- Splinter haemorrhages

**Condition/s associated with**
- Psoriasis
- Psoriatic arthritis

**Mechanism/s**
The mechanism is poorly understood. It is likely that a combination of...
Psoriatic nails/psoriatic nail dystrophy

Psoriatic nails/psoriatic nail dystrophy

Psoriasis is thought to be a disease of abnormal immunology in which an atypical T-cell response occurs, part of which results in an aberrant proliferation of T cells which migrate to the skin and activate and release various cytokines (e.g. IFN-γ, TNF-α and IL-2). These cytokines induce changes in keratinocytes and are also associated with the development of the characteristic psoriatic skin lesions.77

**Nail pitting**
Nail pitting is the result of multifocal abnormal nail growth. The nail matrix is made up of keratinocytes, which generate the keratin that results in production of the nail plate. As new cells are produced, the older cells are pushed forwards and ‘grow’ the nail.

In psoriatic nails, there are parakeratotic cells that disrupt normal keratinisation and nail production. These abnormal cells group together and then get sloughed off as the nail grows, leaving a depression in the nail plate.76,78

**Subungual keratosis**
Excessive proliferation of keratinocytes under the nail plate leads to the accumulation of keratotic cells. This often leads to a raised and thickened nail plate.77

**Oil drops**
Thought to be caused by the accumulation of neutrophils that become visible through the nail plate.

**Salmon patches**
Focal hyperkeratosis of the nail bed and altered vascularisation.76

**Splinter haemorrhages**
See Chapter 3, ‘Cardiovascular signs’.

**Sign value**
Studies report psoriatic nail changes may be present in up to 15–50% of cases of psoriasis and have a lifetime prevalence of 80–90%.79,80 Several studies report a higher incidence of psoriatic nail changes (75–86%) in patients with psoriatic arthritis.81–84
Raynaud’s syndrome/phenomenon

**Description**

Raynaud’s syndrome/phenomenon occurs in the digits from various stimuli, resulting in peripheral hypoperfusion followed by hyperaemia. It has three ‘colour’ phases:

1. white – blanching associated with vasoconstriction of the blood vessels
2. blue – cyanosis
3. red – when blood flow is restored and hyperaemia results.
Condition/s associated with

Common
• Raynaud’s phenomenon

Less common
• Vasculitis
  » Buerger’s disease
• Autoimmune/connective tissue disorders
  » Scleroderma (systemic sclerosis)
  » Systemic lupus erythematosus
  » CREST syndrome
  » Sjögren’s syndrome
  » Dermatomyositis
  » Polymyositis
  » Rheumatoid arthritis
• Drugs
  » Beta blockers

Mechanism/s
Raynaud’s syndrome occurs due to an exaggerated vasoconstrictive response causing transient cessation of blood flow to the digits.\textsuperscript{85–88} The cause of this abnormal vasoconstrictive response is multifactorial:

1 Increased sympathetic nerve activation (centrally and peripherally mediated) – in response to cold temperatures or stressful situations, enhanced sympathetic nerve activation leads to vasoconstriction of the arterioles in the digits. Larger numbers of alpha-2-adrenoreceptors may result in more pronounced vasoconstriction.\textsuperscript{85–88} The increase in alpha-2-adrenoreceptors involves reactive oxygen species, Rho/Rho kinase and the actin cytoskeleton. Cold can induce Rho/Rho kinase, causing more adrenoreceptors to move to the cell membrane and interact with its ligand.\textsuperscript{89}

2 Impaired habituation of the cardiovascular response to stress is also thought to contribute. Habituation is the gradual extinction of a response to a stimulus over time. In normal individuals, ongoing exposure to a stress results in habituation, and decreasing incidence and duration of the response.\textsuperscript{85,86}

3 Local vascular factors – an imbalance between local vasoconstrictive factors (endothelin, 5-HT, thromboxane [TXA] and other cyclo-oxygenase [COX] pathway products) and vasodilatory factors (nitric oxide [NO])\textsuperscript{85,86} may also exist in Raynaud’s syndrome.

» Local endothelin may not produce enough NO for vasodilatation.\textsuperscript{86}
» Repeated vasospasm causes oxidative stress and reduced NO production, thus decreasing vasodilatation.\textsuperscript{85}
» Inappropriately greater production of endothelin and thromboxane (TXA\textsubscript{2}) in response to cold also occurs, leading to marked vasoconstriction.\textsuperscript{85,86}
» In some studies, a higher than normal endothelin-1, a potent vasoconstrictor, was seen in patients with primary Raynaud’s syndrome.\textsuperscript{86}

4 Other factors. Some of these include:
» oestrogen – causing sensitisation of vessels to vasoconstriction\textsuperscript{85,86}
» increased blood viscosity\textsuperscript{86}
» decreased amounts of calcitonin gene-related peptide (CGRP) neurons – impairing normal nerve sensitivity, activation and vasodilatation\textsuperscript{86}
» endothelial damage
Secondary Raynaud’s syndrome

Structural vascular abnormalities (in addition to the factors outlined above) are thought to play a role in Raynaud’s phenomenon occurring secondary to an underlying disease process.

In scleroderma (systemic sclerosis), abnormal proliferation of intimal cells results in endothelial cell damage. Abnormal endothelial cells then exacerbate vasospasm by:86,88

- perturbing smooth muscle cells, causing them to proliferate and contract
- enhancing pro-coagulant activity and inhibitors of fibrinolysis, thus promoting microthrombi
- promoting inflammation through release of adhesion factors.

Other factors thought to contribute in systemic sclerosis include:86

- raised levels of angiotensin II – a vasoconstrictor
- lack of compensatory angiogenesis to meet the demands of proliferated intima – leading to ischaemia.

Sign value

Although not always associated with other rheumatological complaints, a thorough history and examination of a patient presenting with Raynaud’s syndrome needs to be undertaken to assess for other underlying causes. It is said that the majority of patients with scleroderma recall these symptoms many years before the onset of skin induration. Further, Raynaud’s phenomenon is present in 90–99% of patients with diffuse or limited scleroderma.90
Saddle nose deformity

Description
Collapse of the middle section of the nose relative to the tip and dorsum, like a saddle.

Condition/s associated with

More common
• Trauma
• Iatrogenic – nasal surgery

Less common
• Wegener’s granulomatosis
• Relapsing polychondritis

Mechanism/s
Destruction of the nasal septum or support cartilage results in the deformity. Direct trauma or prior surgery is the most common aetiology.

Wegener’s granulomatosis
Wegener’s granulomatosis is an autoimmune vasculitic disorder characterised by necrotising granulomas affecting the small blood vessels of the upper and lower airways. It is thought that immune complex deposition or an autoimmune response results in inflammation and damage/destruction of the vessels and their surrounding structures. In immune disease, the cartilaginous structures of the outer nose and septum are generally more severely involved than the bony nasal dorsum. Severe disease may cause progressive loss of septal support, leading to enlarged anterior septal perforations, resulting in significant collapse of nasal cartilage.

Relapsing polychondritis
Relapsing polychondritis is an autoimmune chronic inflammatory disorder resulting in the destruction of cartilage – in particular auricular and nasal cartilage.

Sign value
Saddle nose deformity occurs in up to 65% of relapsing polychondritis, and 9–29% of patients with Wegener’s granulomatosis.
**Sausage-shaped digits (dactylitis)**

**Description**
Fusiform swelling of multiple digits such that it is difficult to visualise the individual joints (e.g. PIP, DIP). Or, more simply, fingers or toes that are so swollen they look like sausages. Dactylitis typically affects multiple digits, whereas flexor tenosynovitis is a distinct entity usually only present in one digit.

**Condition/s associated with**

**More common**
- HLA-B27 spondyloarthritis
  - Psoriatic arthritis
  - Ankylosing spondylitis
  - Reactive arthritis
  - Enteropathic arthritis (associated with inflammatory bowel disease)
- Sickle cell anaemia – paediatric

**Uncommon**
- Tuberculosis
- Gout
- Sarcoidosis
- Disseminated gonorrhea

**Mechanism/s**

**Spondyloarthropathies**
Irritation of the flexor tendons, flexor tendon sheath and surrounding soft tissues due to pro-inflammatory cytokines results in pronounced diffuse inflammation of the digits. Recent high-resolution MRI studies in psoriatic arthritis reported flexor tendon pulley and sheath-related enthesitis as the more specific cause.

**Tuberculosis dactylitis**
A variant of tuberculous osteomyelitis whereby TB granulomas in the short tubular bones of the hands and feet and then the surrounding tissues, causing inflammation and swelling.

**Syphilitic dactylitis**
A manifestation of congenital syphilis where the syphilitic spirochetes invade perichondrium, bone, periosteum and marrow and thus inhibit osteogenesis. Inflammation from the invasion is another contributing factor to pain and swelling of the digits.

**Sarcoid dactylitis**
Sarcoid non-cascating granulomas invade bone and soft tissue, causing swelling and inflammation.

**Sickle cell dactylitis**
In sickle cell anaemia, a haemoglobin S-gene mutation results in rigid and ‘sickle’-shaped red blood cells under hypoxic conditions. Acute sickling in...
Sausage-shaped digits (dactylitis)

the peripheral circulation results in digital ischaemia and painful fusiform digital swelling. It typically occurs in the paediatric population.

**Sign value**

In regards to patients with seronegative spondyloarthropathy, sausage-shaped digits have a sensitivity of 17.9% and a specificity of 96.4%.96 The development of dactylitis may be a marker for progression of psoriatic arthritis,97 being present in 16–24%97 of reported cases, with lifetime incidence and prevalence of 48% and 33%, respectively.98 It is seen in only 4% of tuberculosis93 cases.

Identification of sausage-shaped digits or dactylitis in an adult should prompt an evaluation for a seronegative spondyloarthropathy. Development of dactylitis in a child of African or Mediterranean descent should prompt evaluation for sickle cell disease.
Sclerodactyly

Condition/s associated with

• Scleroderma (systemic sclerosis)
• CREST syndrome (i.e. Calcinosis, Raynaud’s phenomenon, Oesophageal dysmotility, Sclerodactyly, Telangiectasia)

Mechanism/s

In scleroderma, T cells infiltrate the skin and set in motion a cascade of events including abnormal fibroblast and growth factor stimulation. This in turn leads to increased production of extracellular matrix, fibrillin and type 1 collagen and other factors. Ultimately this results in fibrosis and thickening of the skin.

Sign value

Skin thickening is seen more often in diffuse scleroderma (27%) than in limited disease (5%).

Description

Thickening and tightening of the skin covering the digits.

---

Genetic factors  Environmental  Other factors

Immunological reaction – mononuclear cells and cytokines infiltrate layers of skin

Vascular inflammation, fibroblasts stimulated, TGF-β released, growth factors, other factors released

Collagen, fibrillin, fibronectin, extracellular matrix synthesis and deposition

Fibrosis, skin thickening and tightening

Sclerodactyly

---

FIGURE 1.42
Sclerodactyly with flexion contractures
Reproduced, with permission, from Firestein GS, Budd RC, Harris ED et al., Kelley’s Textbook of Rheumatology, 8th edn, Philadelphia: WB Saunders, 2008: Fig 47-12.

FIGURE 1.43
Proposed mechanism of sclerodactyly


**Description**
A confluent, violaceous, macular rash over the posterior shoulders and neck.

**Condition/s associated with**
- Dermatomyositis

**Mechanism/s**
Complement and antibody mediated microvascular injury likely results in the development of the rash. Dermatomyositis is a systemic inflammatory disorder primarily of muscle and skin characterised by microvascular damage due to antibody complex and complement deposition. Genetic predisposition, viruses and UV light are all thought to play a role.

The cutaneous changes in dermatomyositis include: hyperkeratosis, epidermal basal cell vacuolar degeneration, pathological apoptosis of epidermal basal and suprabasal cells, dyskeratosis, a focally thinned epidermis, and increased dermal mucin deposition. Factors contributing to these changes include:

- Dysregulated cytokine production and cell-mediated processes. Histopathology of dermatomyositic lesions have shown the principal infiltrating cell in the skin is the CD4+ T lymphocyte, distributed mainly in the perivascular upper dermis.
- In skin biopsies, a significantly increased number of both CD-40+ cells (including keratinocytes and mononuclear cells in the dermis), as well as infiltrating CD4+ CD-40L+ T-lymphocytes, are found. Activation of the CD-40/CD-40L system may cause upregulation of several pro-inflammatory molecules, including IL-6, IL-15, IL-8 and MCP-1.
- Dysregulated apoptosis of keratinocytes. The exact mechanism for keratinocyte apoptosis is unclear, but includes UVB light, Fas-FasL, TNFα, and CD8+ T-cell-mediated activation of the apoptosis pathway.
- Photosensitivity is an important clinical feature. Exposure to sunlight and specifically UVA and UVB radiation may serve a central role in disease onset and persistence. Exposure to UVB radiation has also been shown to upregulate the pro-inflammatory cytoking TNF-α.
- Excess mucin deposition has been hypothesised to occur secondary to increased hyaluronic acid production by dermal fibroblasts following immunological stimulation.

*Reproduced, with permission, from Hochberg MC et al., Rheumatology, 5th edn, Philadelphia: Mosby, 2010: Fig 144-7.*
Sign value

Although not pathognomonic, the shawl sign is strongly associated with dermatomyositis. In up to 30% of cases of dermatomyositis, skin manifestations occur. However, skin manifestations may occur without the onset of muscle symptoms.¹⁰²
Simmonds–Thompson test

(absence of plantarflexion) can be elicited.

**Condition/s associated with**
- Achilles tendon rupture

**Mechanism/s**
Normally, squeezing the gastrocnemius and soleus muscles results in shortening of the distance between the Achilles tendon insertion site and distal femur, causing plantarflexion.\(^{103}\) If the Achilles tendon is ruptured, no movement occurs.

**Sign value**
With a sensitivity of 96%, specificity of 93%, +LR 13.5 and −LR of 0.05,\(^{104}\) it is a worthwhile test to perform if an Achilles rupture if suspected. A positive test almost certainly suggests significant damage to the tendon, but does not always indicate complete rupture.\(^{105}\)

**Description**
With the patient lying prone on the exam table with their ankles hanging over the end, the examiner squeezes the calf muscle. The test is considered positive if no movement in the ankle

*FIGURE 1.45*
Simmonds–Thompson test
The calf muscles are squeezed, and the test is positive if there is no ankle plantarflexion.
Speed’s test

The test is positive if tenderness is elicited.

Condition/s associated with

- Biceps tendinitis
- SLAP lesion (Superior Labral tear from Anterior to Posterior) – an injury of the glenoid labrum

Mechanism/s

Traction on an inflamed biceps tendon or pressure on a labral tear will result in tenderness.

Sign value

Holtby R et al., in predicting biceps pathology and SLAP lesions, reported a sensitivity of 32%, a specificity of 75%, a positive likelihood ratio of 1.28 and a negative likelihood ratio of 0.91. This test has limited value.
Subcutaneous nodules (rheumatoid nodules)

Sign value

Seen in 20–25% of seropositive rheumatoid arthritis. They are the most common extra-articular manifestation of the disease. Frequency of development of nodules is associated with elevated rheumatoid factor titres. The presence of nodules is associated with a more severe disease phenotype.

Description

Visible and palpable subcutaneous nodules typically occur over bony prominences and/or extensor surfaces.

Condition/s associated with

• Rheumatoid arthritis

Mechanism/s

Thought to be mediated via Th-1 inflammatory response. Trauma over bony prominences causes local vessel damage that leads to new blood vessel growth and granulomatous tissue formation. Endothelial injury results in accumulation of immune complexes and stimulates monocytes to secrete IL-1, TNF, TGF-β, prostaglandins and other factors, including proteases, collagenases and fibronectin. This ultimately leads to angiogenesis, fibrin deposition and formation of the characteristic rheumatoid nodule.
Sulcus sign

Mechanism/s
In the setting of anterior shoulder dislocation, the head of the humerus moves inferiorly relative to the glenohumeral joint. This causes traction of the skin overlying the glenohumeral joint, and a dimple over the space between the acromion and the humeral head may be seen.

Sign value
Anterior shoulder dislocation is often apparent on inspection with the arm held anteriorly and internally rotated. Radiographs should be obtained to confirm the diagnosis. Testing for glenohumeral head instability, Tzannes and Murrell\textsuperscript{110} found a sulcus sign of 2 cm or more to have a specificity of 97% for multidirectional instability; however, the corresponding sensitivity was only 28%. If positive, the sign supports glenohumeral head instability and/or a subluxation, but should be confirmed with imaging.

Description
With the patient’s arm relaxed and hanging by the side, the examiner looks at the shoulder area. If chronic subluxation is suspected, the examiner may apply traction to the arm to elicit the sign. Dimpling of the skin between the acromion and humeral head is a positive test.

Condition/s associated with
• Anterior shoulder dislocation
• Anterior shoulder subluxation
• Glenohumeral joint instability

**FIGURE 1.49**
Sulcus sign
Note the slight dimple under the acromion.
*Reproduced, with permission, from DeLee JC, Drez D, Miller MD, DeLee and Drez’s Orthopaedic Sports Medicine, 3rd edn, Philadelphia: Saunders, 2009: Fig 17H2-16.*
**Supraspinatus test (empty-can test)**

**Mechanism/s**
The supraspinatus muscle works in concert with the deltoid muscle during shoulder abduction and stabilises the humeral head in the glenoid fossa. Mechanical strain upon an injured supraspinatus muscle or tendon will result in tenderness and/or weakness during this manoeuvre.

**Sign value**
McGee, in a review of two studies in patients with rotator cuff muscle tears with painful supraspinatus tests, reported a sensitivity of 63–85%, a specificity of 52–60% and a positive likelihood ratio of 1.7.75 A review of five studies of patients with rotator cuff muscle tears with weakness during testing, reported a sensitivity of 41–84%, a specificity of 58–70%, a positive likelihood ratio of 2.0 and a negative likelihood ratio of 0.5.2

The supraspinatus test has limited utility and may be positive in several other shoulder conditions. Detection of weakness has more diagnostic utility than tenderness alone.

**Description**
The patient sits or stands with the shoulder in 90° flexion, 30° abduction, with the elbow extended and the thumbs pointing towards the ground, as if emptying two cans. The examiner applies downward pressure as the patient attempts to lift the arms up. The test is positive if the patient experiences tenderness or is unable to hold up their arm.

**Condition/s associated with**
- Supraspinatus tear
- Supraspinatus tendinitis
- Supraspinatus impingement
Swan-neck deformity

A relative imbalance of flexor and extensor tendons of the digit due to chronic synovial inflammation. A variety of changes may result in this deformity, whose basis is inflammatory disruption of the collateral ligaments, volar plates, joint capsule or invasion of the flexor tendons. The resulting changes may be:

Description
A deformity characterised by distal interphalangeal (DIP) joint flexion and proximal interphalangeal (PIP) joint hyperextension in the resting digit, to some extent resembling a swan’s neck.

Condition/s associated with
Common
• Rheumatoid arthritis

FIGURE 1.51
Swan-neck deformity pathoanatomy
A Terminal tendon rupture may be associated with synovitis of DIP joint, leading to DIP joint flexion and subsequent PIP joint hyperextension. Rupture of flexor digitorum superficialis tendon can be caused by infiltrative synovitis, which can lead to decreased volar support of PIP joint and subsequent hyperextension deformity; B lateral-band subluxation dorsal to axis of rotation of PIP joint. Contraction of triangular ligament and attenuation of transverse retinacular ligament are depicted.

Swan-neck deformity

• attenuation or disruption of the extensor tendon on the distal phalanx, leading to unopposed flexion – and thus the flexed DIP joint
• disruption of the retinacular ligament (which helps hold the finger in flexion), leading to unopposed extensor forces at the PIP joint and PIP joint hyperextension.

Sign value
Swan-neck deformity is classically associated with rheumatoid arthritis. In patients with acute trauma with forced DIP flexion during active extension, mallet finger (i.e. extensor tendon avulsion distal to DIP joint) should be considered.

FIGURE 1.52
Swan-neck deformity
Telangiectasia

**General mechanism/s**

Telangiectasias are predominantly persistently dilated small capillaries and venules. The exception to this is hereditary haemorrhagic telangiectasia, as these lesions are arteriovenous (AV) malformations.

**Hereditary haemorrhagic telangiectasia (HHT)**

HHT is an autosomal dominant disorder causing development of AV malformations, due to a genetic abnormality of the TGF-β receptor. The TGF-β pathway is known to modulate vascular architecture, matrix formation and basement membrane development.¹¹³

**Scleroderma**

The underlying mechanism for telangiectasia in scleroderma is unknown. It is presumed that there is endothelial injury, leading to aberrant angiogenesis and the development of new vessels. It has been suggested that the TGF-β pathway may be involved.¹¹³

**Description**

Permanent dilatation of superficial peripheral vessels resulting in blanching red lesions on the skin. Telangiectasia may present as a fine red line or a punctum (dot) with radiating lines.⁶⁶

**Condition/s associated with**

There are numerous conditions associated with telangiectasia, including but not limited to those listed in Table 1.3.

---

**TABLE 1.3**

Telangiectasia-associated conditions

<table>
<thead>
<tr>
<th>Systemic diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carcinoid syndrome</td>
</tr>
<tr>
<td>Ataxia–telangiectasia</td>
</tr>
<tr>
<td>Mastocytosis</td>
</tr>
<tr>
<td>Dermatomyositis</td>
</tr>
<tr>
<td>Scleroderma – especially periungual telangiectasia</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
</tr>
<tr>
<td>Hereditary haemorrhagic telangiectasia</td>
</tr>
<tr>
<td>Liver cirrhosis</td>
</tr>
</tbody>
</table>

---

**FIGURE 1.53**

Telangiectasia associated with systemic sclerosis (scleroderma)

Note the skin tightening around the lips.

Reproduced, with permission, from Habif TP, Clinical Dermatology, 5th edn, Philadelphia: Mosby, 2009: Fig 17-30.
**Spider naevus**
See ‘Spider naevus’ in Chapter 6, ‘Gastroenterological signs’.

**Sign value**
Location and characteristics of telangiectasia can assist in diagnosis.

- Periungual telangiectasia (telangiectasia next to the nails) is said to be highly suggestive of SLE, scleroderma or dermatomyositis.\(^{114}\)
- Broad macules with a polygonal or oval shape, known as *mat telangiectasias*, are associated with CREST syndrome.\(^{114}\)
- Telangiectasias in adulthood that are located around the mucous membranes, extremities and under the nails are associated with hereditary haemorrhagic telangiectasia.
- The presence of increased numbers of telangiectasias may indicate the development of pulmonary vascular disease in scleroderma. Telangiectasia may be a clinical marker of more widespread microvascular disease in scleroderma.\(^{115}\)
The test is performed with the knee at 5° and 20° of flexion. The examiner supports the patient by the hands as they stand flatfooted. The patient then rotates their knee and body, internally and externally, three times, keeping the knee in slight flexion (5°). The same movement is repeated with the knee flexed to 20°. The test is always performed first on the normal knee so that the patient is comfortable with performing the manoeuvre (i.e. how to keep the knee in 5° and then 20° of flexion). See Figure 1.54.

**Condition/s associated with**
- Meniscal tears

**Mechanism/s**
The knee meniscus (lateral and medial) is fibrocartilaginous tissue that separates the femur from the tibia. The menisci provide rotational stability and act as shock absorbers, among other functions. The Thessaly test creates loading conditions on the meniscus and a rotational stress that may result in reproduction of pain, catching or other reported symptoms.

**Sign value**
Blyth M et al.\(^1\) compared the accuracy of physical tests for meniscal tears to MRI-based diagnosis (see Table 1.1 on page 35). The Thessaly test had only moderate value in assessment when compared to MRI. The tests may be useful initially, but realistically, definitive imaging is required.
Thomas’ test

Description
With the patient lying supine, the knee and hip on the ‘normal’ side are flexed, with the knee held against the chest. A positive test occurs if the opposite leg rises off the table.

Condition/s associated with
- Hip flexion contracture – fixed flexion deformity
- Iliotibial band syndrome
- Normal ageing/stiffness

Mechanism/s
Drawing up the knee and flexing one side of the hip rotates the pelvis. In order to keep the alternate leg flat on the bed, the hip flexors and rectus femoris must stretch enough to allow the leg to lie flat. In other words, if the hip flexors are contracted, the affected leg will rise as the pelvis rotates.

Sign value
A systematic review of diagnostic tests for hip pathology found minimal scientific evidence for Thomas’ test due to methodological flaws. There is limited value in this sign.
Tinel’s sign

Description
Paraesthesias in a median nerve distribution occur when the examiner taps with a finger at the distal wrist over the median nerve. It should be noted that Tinel’s original description was not specific for the median nerve but rather for the sensation of ‘pins and needles’ arising from any injured nerve tested in this way.

Condition/s associated with
• Carpal tunnel syndrome

Mechanism/s
In carpal tunnel syndrome, there is increased pressure in the carpal tunnel and resulting damage to the median nerve. It is thought that this damage results in altered mechanosensitivity of the median nerve, possibly due to an abnormally excitable membrane. So, when lightly struck through the skin, the inflamed nerve functions abnormally.

Sign value
D’Arcy CA et al. reported Tinel’s sign had limited or no value in distinguishing people with carpal tunnel syndrome from those without. A review of several studies reported a sensitivity of 25–60%, a specificity of 64–80%, a positive likelihood ratio of 0.7–2.7 and a negative likelihood ratio of 0.5–1.1. Neither Tinel’s sign nor Phalen’s sign reliably rule in or rule out carpal tunnel syndrome.
Trendelenburg’s sign

**Condition/s associated with**
- Gluteus medius muscle weakness
  - Superior gluteal nerve palsy – iatrogenic
  - Lumbar radiculopathy
  - Sequelae of hip joint pathology
    - Osteoarthritis
    - Slipped femoral capital epiphysis (SCFE) – paediatrics
    - Legg–Calve–Perthes disease – paediatrics

**Mechanism/s**
The gluteus medius muscle originates from the iliac crest and inserts into the greater trochanter of the femur. Normally when we stand on one leg, the gluteus medius muscle abducts the hip joint to maintain normal alignment of the pelvis. With gluteal medius weakness, the sound side (the side opposite to the stance leg) sags, or tilts downwards.

**Sign value**
Given the number of potential causes, a positive Trendelenburg sign is fairly non-specific; however, it is never normal and should be investigated.

**Description**
The patient is asked to stand on one leg while bending the other knee so the foot is held off the ground. For the sign to be present, the pelvis must be seen to ‘drop’ on the unsupported side. Confusingly, the pathology is *not* located on the ‘dropped’ side, but in the opposite leg, hence the saying ‘the sound side sags’.

**FIGURE 1.58**
Trendelenburg test
Note that the positive test on the right indicates a problem with the left hip abductors – remember ‘the sound side sags’.

True leg length inequality (anatomic leg length inequality)

Description
The leg length is measured from the anterior iliac spine to the medial malleolus. There is no clear definition as to what constitutes a significant discrepancy. Some authors suggest that it is not clinically relevant until there is more than 20 mm difference between legs.\(^{119}\)

Condition/s associated with
- Fracture – hip, femur, tibia
- Dislocation – hip, knee
- Post-surgical shortening
- Congenital disorders

Mechanism/s
True, or anatomic, leg length equality relates to the actual length of the bones and anatomical structures making up the hip and the lower limb. Therefore, any problem in the anatomy that constitutes the leg length (from the head of the femur down to the ankle) may cause a discrepancy. For example, abnormalities in growth plates during development may lead to one leg being longer than the other. Aberrant healed fractures can also lead to a shortened leg.

Sign value
A leg length discrepancy is a non-specific sign. It should be interpreted in the context of the patient’s history.
Ulnar deviation

FIGURE 1.59
Ulnar deviation and subluxation
The hand shows typical manifestations of end-stage erosive changes around the metacarpophalangeal joints, with volar and ulnar drift of the fingers.
Reproduced, with permission, from Firestein GS, Budd RC, Harris ED et al., Kelley’s Textbook of Rheumatology, 8th edn, Philadelphia: WB Saunders, 2008: Fig 66-5.

Description
Displacement of the metacarpophalangeal and/or radiocarpal joint towards the ulnar aspect of the wrist.

Condition/s associated with
- Rheumatoid arthritis
- Pigmented villonodular synovitis

Mechanism/s
Metacarpophalangeal (MCP) joint
MCP joints are condylar and are able to move in two planes. They are less stable than interphalangeal joints. Progressive inflammatory changes from rheumatoid arthritis result in stretching of the joint capsule and ligaments, causing instability. Extrinsic forces on the joints tend to pull in a direction of ulnar deviation. Possible factors include:108,111
- the normal tendency of fingers to move towards the ulnar side on flexion
- inflammation of the carpometacarpal (CMC) joints in the 4th and 5th fingers causes further spread of the metacarpals in flexion, producing an ‘ulnarly’ directed force on the extensor tendons
- stretching of the collateral ligaments of the MCP joints, accessory collateral ligaments or flexor tunnels that permits volar displacement of the proximal phalanges.

Radiocarpal ulnar deviation
Progressive inflammatory changes lead to progressive synovitis of the wrist joint and carpal bones, including the scaphoid. Abnormal wrist mechanics develop due to translocation of the carpal bones relative to the radius, and imbalance of mechanical forces.108

Sign value
Ulnar deviation of the MCP joints is classically associated with rheumatoid arthritis, although other causes should be considered.
**FIGURE 1.60**
Irregular patchy erythema with associated prominent telangiectasias in a woman with dermatomyositis
Reproduced, with permission, from Shields HM et al., Clin Gastroenterol Hepatol 2007; 5(9): 1010–1017.

**Description**
A confluent, macular, violet/red rash seen over the anterior neck and upper chest. Often found in a V-shape similar to the neck of a shirt.

**Condition/s associated with**
- Dermatomyositis

**Mechanism/s**
Complement- and antibody-mediated microvascular injury likely results in the development of the rash. Dermatomyositis is an inflammatory myopathy characterised by microvascular damage and destruction of muscle by antibody complex and complement deposition. Genetic predisposition, viruses and UV light are all thought to play a role. See Heliotrope rash and Shawl sign.

**Sign value**
Although not pathognomonic, the V-sign is highly suggestive of dermatomyositis. In up to 30% of cases, skin manifestations including the V-sign may occur before development of the characteristic muscle weakness.
Valgus deformity

**Description**
Angulation of the distal bone of a joint away from the midline of the body.

**Condition/s associated with**
Associated conditions are given in Table 1.4.

**Mechanism/s**
**Hallux valgus**
Anatomical, biomechanical and pathological factors contribute to the formation of hallux valgus. Some of those identified include:

- Limited soft tissue stabilising the 1st MTP joint results in forces pushing the toe laterally being relatively unrestrained.
- Owing to the anatomy of the metatarsocuneiform joint, increased pressure under the first metatarsal (e.g. from excessive pronation) will tend to displace the first metatarsal.
- Inflammatory joint disease may precipitate the formation of hallux valgus by damaging ligaments and altering normal joint alignment.
TABLE 1.4
Valgus deformity-associated conditions

<table>
<thead>
<tr>
<th>Hip</th>
<th>Knee</th>
<th>Ankle</th>
<th>Toe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteochondrosis</td>
<td>Cerebral palsy</td>
<td>Paralytic</td>
<td>Biomechanical</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Osteochondrosis</td>
<td></td>
<td>Congenital</td>
</tr>
<tr>
<td>Blount’s disease</td>
<td>Osteochondrosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rickets</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paralytic</td>
<td></td>
<td>Multiple sclerosis</td>
<td></td>
</tr>
<tr>
<td>Osteochondrosis</td>
<td></td>
<td>Cerebral palsy</td>
<td></td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td></td>
<td>Rheumatoid arthritis</td>
<td></td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td></td>
<td>Intra-articular damage</td>
<td></td>
</tr>
</tbody>
</table>

Anatomical absence of muscle stabiliser – from metatarsal to proximal great toe

Excessive pronation

Inflammatory disease – destruction of ligaments and normal joint integrity

FIGURE 1.62
Factors involved in the mechanism of hallux valgus

Anatomical absence of muscle stabiliser – from metatarsal to proximal great toe

Excessive pronation

Inflammatory disease – destruction of ligaments and normal joint integrity

Medial displacement of proximal toe, lateral displacement of distal toe

Chronic stress on medial ligaments – eventual disruption of medial ligaments

Unopposed adductor ligament action

Chronic stress – eventual hallux valgus deformity

Knee valgus (genu valgum)
Genu valgum may be caused by a number of disorders. Basic mechanisms for a number of these conditions are shown in Table 1.5.

Sign value
Valgus deformity has limited utility. Aetiology differ largely depending upon the site of the deformity.
### TABLE 1.5
Genu valgum mechanism/s

<table>
<thead>
<tr>
<th>Condition</th>
<th>Basic mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D deficiency</td>
<td>A lack of vitamin D leads to abnormal bone mineralisation, softer-than-normal bones, abnormal bone regrowth and bowing of the legs. Mechanical forces play a role in bone regrowth</td>
</tr>
<tr>
<td>Paget’s disease</td>
<td>Invasion with paramyxovirus leads to abnormal activation of osteoclasts and aberrant osteoblast activity. Deformation of the bone and knee can lead to anatomical changes and valgus deformity</td>
</tr>
<tr>
<td>Osteochondrosis</td>
<td>Interrupted blood supply, especially to the epiphysis, leads to necrosis and then later bone regrowth – resulting in abnormal formation of femur and knee joint – and eventually a valgus deformity</td>
</tr>
<tr>
<td>Neuromuscular disorders</td>
<td>Weak quadriceps, gastrocnemius and hip abductors may cause knees to enter valgus position(^5^6)</td>
</tr>
</tbody>
</table>

Valgus deformity
Varus deformity

Description
Angulation of the distal bone of a joint towards the midline.

Condition/s associated with
Associated conditions are given in Table 1.6.

Mechanism/s

Coxa vara
Present if the angle between the femoral neck and shaft is less than 120°.

Congenital
Congenital coxa vara may present in infancy or later in childhood. It is often bilateral and characterised by progressive bowing of the femur and a defect in the medial part of the neck of the femur.56

Rickets
Pressure placed on the femoral neck of abnormally mineralised bone distorts its normal architecture.

FIGURE 1.63
Bowing of both legs in infantile Blount’s disease
**Perthes’ disease**
Although the underlying cause of Perthes’ disease is unknown, there is a loss of blood supply to the femoral head. Avascular necrosis of the femoral head results in distortion of the normal bony alignment of the femur.

**Genu varum**
Genu varum or ‘bow-leggedness’ is normal in many children up to 2 years.\(^{122,123}\) It should be differentiated from Blount’s disease.

**Blount’s disease**
The underlying mechanism of genu varum in Blount’s disease is unknown. Abnormal growth of the medial tibial epiphyseal growth plate causes progressive varus deformity at the knee joint.\(^{123}\)

**Hallux varus**
Hallux varus is comprised of medial deviation of the first metatarsophalangeal (MTP) joint, supination of phalanx and interphalangeal flexion or claw toe. It results from an imbalance between osseous, tendon and capsuloligamentous structures at the first MTP joint.\(^{124}\)

**Sign value**
Aetiologies differ largely depending upon the site of the deformity.

### TABLE 1.6
Varus deformity-associated conditions

<table>
<thead>
<tr>
<th>Hip</th>
<th>Knee</th>
<th>Ankle</th>
<th>Toe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital disorders (e.g. cleidocranial dysplasia, Gaucher’s disease)</td>
<td>Physiological – common</td>
<td>Trauma</td>
<td>Complication from bunion surgery</td>
</tr>
<tr>
<td>Perthes’ disease</td>
<td>Blount’s disease</td>
<td>Iatrogenic</td>
<td>Trauma</td>
</tr>
<tr>
<td>Development dysplasia of hip</td>
<td>Rickets</td>
<td>Congenital</td>
<td>Burn injury with contracture</td>
</tr>
<tr>
<td>Slipped capital femoral epiphysis (SCFE)</td>
<td>Trauma</td>
<td></td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td>Rickets</td>
<td>Infection</td>
<td></td>
<td>Psoriatic arthritis</td>
</tr>
<tr>
<td>Osteomyelitis</td>
<td>Tumour</td>
<td></td>
<td>Charcot–Marie–Tooth (CMT) disease</td>
</tr>
<tr>
<td>Paget’s disease</td>
<td>Skeletal dysplasia</td>
<td></td>
<td>Avascular necrosis</td>
</tr>
<tr>
<td>Trauma</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
Yergason’s sign

Description
The examiner stands in front of the patient, who has their arms flexed to 90° at the elbow and the palms facing downwards (pronated). The patient then tries to supinate the forearm against resistance from the examiner.

Condition/s associated with
- Biceps tendonitis
- SLAP lesion (Superior Labral tear from Anterior to Posterior) – an injury of the glenoid labrum
- Rotator cuff injuries

Mechanism/s
The long head of biceps is the main supinator of the arm. With resistance against supination, the muscle and tendon are stressed and any inflammation or damage is exacerbated, resulting in tenderness.

The long head of biceps travels in the bicipital groove of the humerus and originates on the lip of the glenoid labrum. The fibrous extension of the subscapularis muscle covers the long head of the biceps tendon and holds it in place. If this fibrous extension is ruptured, the biceps tendon is susceptible to subluxation. Continuous subacromial impingement can wear away the capsule above the long head.

of the biceps tendon. This causes ongoing injury.\textsuperscript{75}

**Sign value**

Holtby R et al., in predicting biceps tendon pathology and SLAP lesions, reported a sensitivity of 43\%, a specificity of 79\%, a positive likelihood ratio of 2.05 and a negative likelihood ratio of 0.72.\textsuperscript{106} A more recent meta-analysis and systematic review reported a specificity of 95\%, with significant heterogeneity between studies.\textsuperscript{126}

In detecting rotator cuff tendonitis, the reported sensitivity was 37\% and specificity 87\%, with a +LR of 2.8 and −LR of 0.7.\textsuperscript{127}
References


References


References


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References


