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4TH EDITION

MECHANISMS OF **CLINICAL SIGNS**

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Mechanisms of CLINICAL SIGNS

4th Edition

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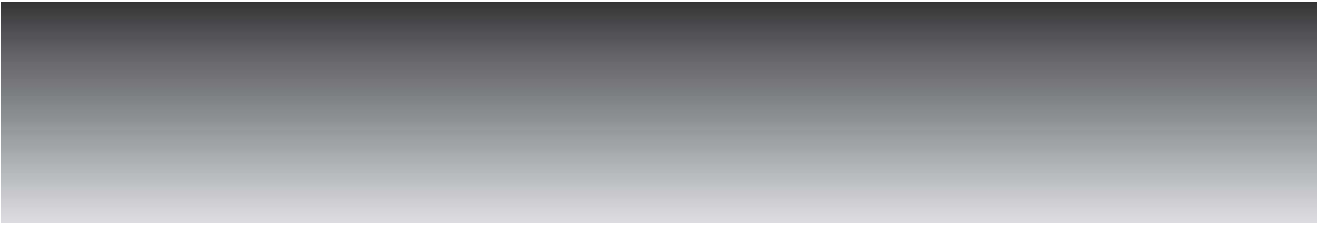
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Preface 4th edition

The authors are pleased to present the latest edition of this textbook. As with previous editions, refinements have been made in response to comments from clinicians, health workers and medical students from around the world. This edition updates current knowledge with new evidence, where available, and the latest concepts in the mechanisms of pathology. Many of these changes have resulted directly from reader feedback, and we are grateful to everyone who has taken the time to share their thoughts with us.

The authors recognise that clinical examination is changing, and its place may be questioned in an era of rapidly advancing technology. There is an increasing reliance on imaging techniques for diagnosis and, more recently, artificial intelligence. Despite the vast amount of information now available at our fingertips, we believe more strongly than ever that the best assessment of a patient begins with a thorough history and physical examination. This approach not only assists clinicians in reaching an accurate diagnosis but also contributes to effective patient care. We encourage readers not to underestimate the knowledge and skills required to perform a comprehensive physical examination. There is no true substitute for these skills and, in many respects, they remain an important part of the art of medicine.

We hope you find the fourth edition of *Mechanisms of Clinical Signs* a valuable resource in your work and training, and we look forward to bringing you future editions.



CHAPTER 1

MUSCULOSKELETAL SIGNS

Anterior drawer test

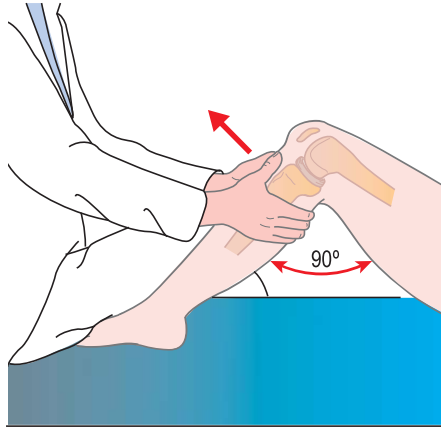


FIGURE 1.1
Anterior drawer test for anterior cruciate ligament injury

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 (Figure 1.1). In a positive test, there is anterior (forward) movement of the tibia without an abrupt stop.¹

Associated condition

- 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. Loss of continuity of the ACL allows

inappropriate anterior movement of the tibia and therefore knee joint instability.

Sign value

A systematic review and meta-analysis of 22 studies of isolated ACL injury reported overall sensitivity of 83% (95% CI 77–88) and specificity of 85% (95% CI 64–95).² Previous literature reviews had only assessed multi-ligament injury, which may overestimate positive findings.²

When comparing acute and chronic ACL injury, the anterior drawer test has variable reliability. Evidence from 28 studies indicates that the anterior drawer test is less dependable for diagnosing ACL injuries in the acute phase, with a sensitivity of 49% and specificity of 58%.³ In chronic ACL injury or where concurrent injuries may be present the anterior drawer test is more reliable for identification of ACL injury, with a sensitivity of 92% (95% CI 88–95) and specificity of 91% (95% CI 87–94).⁴

While a positive anterior drawer sign (PLR 11.5)³ has suggested strong evidence of ACL injury, the results are not uniform, with another study reporting a PLR 2.0 (sensitivity 83%, specificity 57%, NLR 0.3).⁵ A negative anterior drawer sign cannot reliably exclude ACL injury (sensitivity 27–88%; NLR 0.5).³

When strong clinical suspicion persists, further diagnostic steps are necessary (e.g. interval re-examination, MRI, arthroscopy).

Apley's grind test

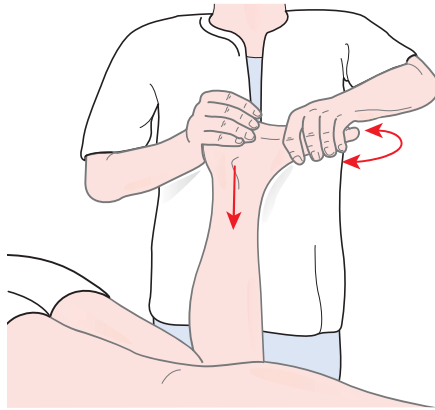


FIGURE 1.2
Apley's grind test

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 (Figure 1.2). The test is positive when it produces tenderness.

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 suggests a ligamentous lesion. If rotation plus compression is more painful or there is decreased rotation relative to the unaffected side, this suggests meniscal injury.⁶

Associated condition

- Meniscal injury

Mechanism(s)

Direct mechanical force on the injured meniscus elicits tenderness.

Sign value

A review by Hegedus et al. reported a pooled sensitivity of 60.7% and specificity of 70.2% with an odds ratio of 3.4.⁷ The wide variation in the data reduces its accuracy. Overall, Apley's grind test has limited diagnostic use, limited supporting data and, in the acute setting, the manoeuvre produces severe pain.⁸

McMurray's grind test has more robust supporting data.

Apley's scratch test

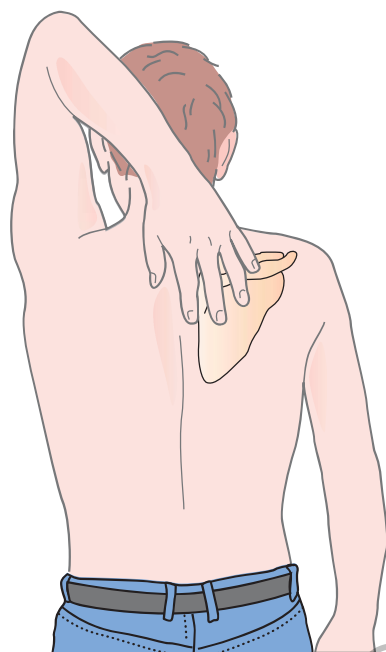


FIGURE 1.3

One of three manoeuvres of Apley's scratch test

Based on Woodward T, Best TM. The painful shoulder: part 1, clinical evaluation. Am Fam Physician 2000;61(10):3079–88.

Description

Apley's scratch test is a general range of movement assessment of the shoulder joint (i.e. glenohumeral, acromioclavicular, sternoclavicular and scapulothoracic joints) (Figure 1.3).

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.⁹

Associated conditions

Common

- Rotator cuff muscle injury
- Labral tear
- Anterior shoulder dislocation
- Bicipital tendonitis
- 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 parts of the shoulder joint.

Sign value

Apley's scratch test is a useful component of the general shoulder exam but has limited value for a specific diagnosis. Note the position of the shoulder at which tenderness or limited range of movement occurs. In a patient with an abnormal Apley's scratch test, conduct further diagnostic manoeuvres to narrow the differential diagnosis. It should be noted that there is limited evidence for the reliability of the Apley's scratch test as an isolated examination technique.

Apparent leg length inequality (functional leg length)

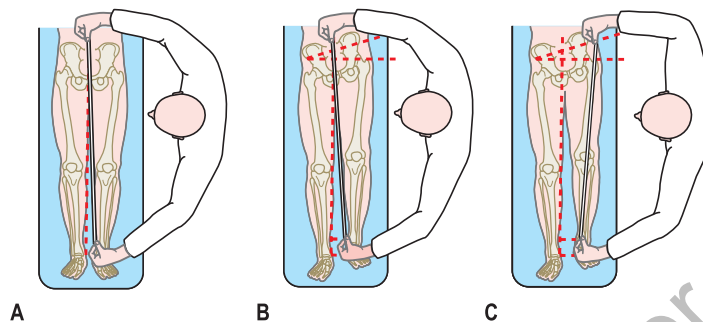


FIGURE 1.4

Measurement of leg lengths

A The apparent leg length is the distance from the umbilicus to the medial malleolus; **B** pelvic rotation causing an apparent leg length discrepancy; **C** the true leg length is the distance from the anterior superior iliac spine to the medial malleolus.

Based on Firestein GS, Budd RC, Harris ED, et al. Kelley's Textbook of Rheumatology, 8th edn, Philadelphia: WB Saunders, 2008, Fig 42-24.

Description

A disparity between the relative distance from the umbilicus to the medial malleolus of each leg¹⁰ (Figure 1.4). By definition it implies asymmetry of the lower extremities in the absence of a bony abnormality. (See 'True leg length inequality' in this chapter.)

Associated conditions

- 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.¹⁰

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.

Apprehension test

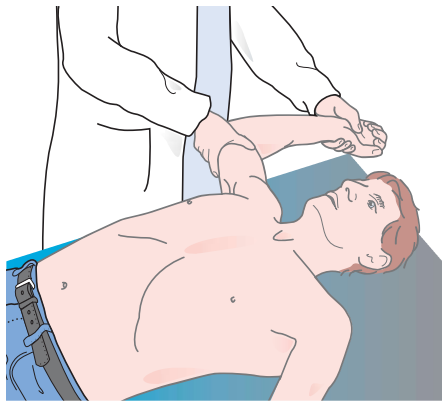


FIGURE 1.5

Apprehension test

The arm is abducted and placed in an externally rotated position. Note the right arm of the examiner is providing anterior traction on the humerus, pulling the posterior part of the humeral head forward. The same test can be done from the back, with the patient sitting up and the examiner pushing forward on the posterior head of the humerus.

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 (Figure 1.5).

The test is positive if the patient experiences *apprehension* due to impending subluxation or dislocation of the glenohumeral joint.¹¹

Associated conditions

More common – traumatic

- 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)

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

Early studies reported a sensitivity of 88.0%, specificity of 50%, positive likelihood ratio (PLR) of 1.8 and negative likelihood ratio (NLR) of 0.23.¹² However, a systematic review of two studies on the apprehension test reported the test as highly specific for anterior instability of the shoulder if apprehension was used as the end

Bouchard's and Heberden's nodes



FIGURE 1.7

Prominent Heberden's nodes

Based on Ferri FF. Ferri's Clinical Advisor, Philadelphia: Elsevier, 2011, Fig 1-223.

Description

Bouchard's nodes are bony outgrowths or nodules found over the *proximal* interphalangeal joints of the hands (Figure 1.7).

Heberden's nodes are similar but located over the *distal* interphalangeal joints.

Associated conditions

- Osteoarthritis
- Familial

Mechanism(s)

A number of studies have implicated *bony osteophyte growth* as the principal

cause of Heberden's and Bouchard's nodes.¹⁵ Other contributing factors or theories include:

- genetic predisposition
- endochondral ossification of hypertrophied cartilage as a result of chronic osteoarthritic changes¹⁶
- traction spurs growing in tendons in response to excessive tension and repetitive strain¹⁷
- chronic enthesitis with inflammatory tissue bulging at known weak points between collateral ligaments and extensor tendons. This leads to subsequent ossification and can be seen on high-resolution MRI.¹⁸

Sign value

Bouchard's or Heberden's nodes are a classic sign of interphalangeal osteoarthritis^{17,19} and are associated with generalised osteoarthritis.^{20,21}

The presence of Bouchard's and/or Heberden's nodes is predictive of the radiographic changes of hand osteoarthritis but is not a reliable surrogate for *generalised* osteoarthritic disease.^{22,23}

Boutonnière deformity

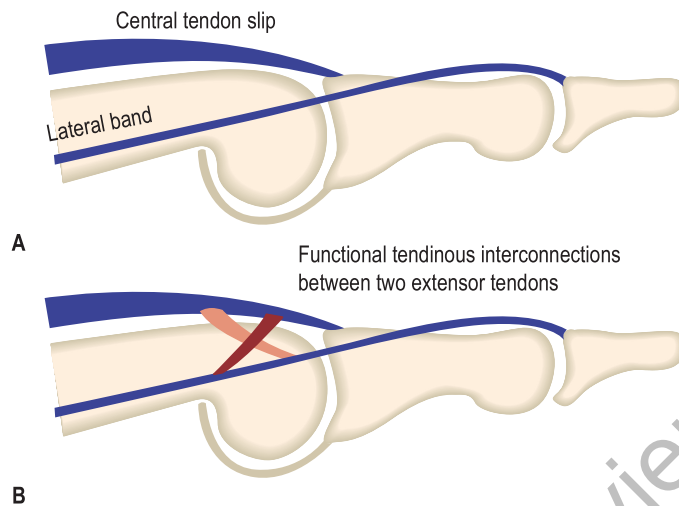


FIGURE 1.8

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.

Based on DeLee JC, Drez D, Miller MD. DeLee and Drez's Orthopaedic Sports Medicine, 3rd edn, Philadelphia: Saunders, 2009, Fig 20B2-27.

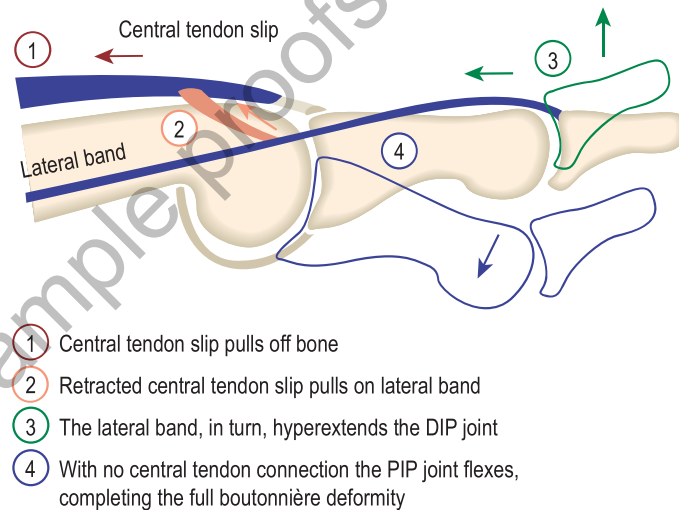


FIGURE 1.9

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.

Based on DeLee JC, Drez D, Miller MD. DeLee and Drez's Orthopaedic Sports Medicine, 3rd edn, Philadelphia: Saunders, 2009, Fig 20B2-28.

Bulge/wipe/stroke test

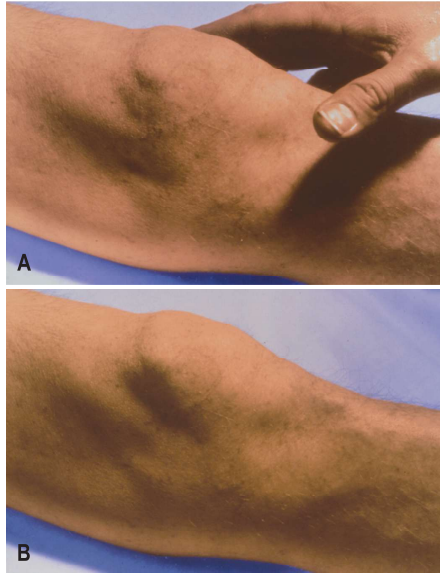


FIGURE 1.10

Demonstration of the bulge test for a small synovial knee effusion. The medial aspect of the knee has been stroked to move the synovial fluid from this area (shaded depressed area in **A**); **B** shows a bulge in the previously depressed area after the lateral aspect of the knee has been tapped.

Based on Firestein GS, Budd RC, Harris ED, et al. Kelley's Textbook of Rheumatology, 8th edn, Philadelphia: WB Saunders, 2008, Figs 35-9A and B.

Description

The bulge, wipe or stroke test is used to assess for knee joint effusion (Figure 1.10). 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.

Associated conditions

Any condition causing a knee effusion.

More common

- Osteoarthritis
- Rheumatoid arthritis
- Haemarthrosis – trauma, coagulopathy
- Traumatic soft tissue knee injury (i.e. acute or chronic ACL tear)
- 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 a visible fluid shift. The wipe or bulge test displaces synovial fluid from one part of the synovial joint to another, suggesting the presence of a joint effusion as the cause of knee swelling.

Sign value

An effusion in the absence of acute traumatic injury or systemic disease is most commonly due to osteoarthritis.²⁸ Limited evidence has been gathered on the diagnostic reliability of this test and ability to identify chronic effusions,²⁹ although some authors report that this test may pick up on *as little as 4–8 mL of swelling*.³⁰ It should be noted that there is low significant inter- and

Butterfly rash (malar rash)



FIGURE 1.11

Malar rash of systemic lupus erythematosus
 Reproduced, with permission, from Goldman L, Ausiello D. Cecil Medicine, 23rd edn, Philadelphia: Saunders, 2007, Fig 287-3.

Description

A red or purple, macular, mildly scaly rash that is seen over the bridge of the nose and cheeks and can be flat or raised³³ (Figure 1.11). 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. It can be painful or intensely pruritic, lasting from days to weeks in most cases.³³

Associated conditions

Common

- Systemic lupus erythematosus (SLE)
- Drug-induced lupus erythematosus (particularly antihypertensives and proton-pump inhibitors)³⁴
- Dermatomyositis

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 (Figure 1.12).

Factors shown to be involved include the following:^{34,35}

- A genetic predisposition to ineffective or deficient complement can lead to a failure to clear immune complexes of apoptotic cells, which in turn increases the chance of developing autoimmunity.
- Sunlight can damage and/or induce apoptosis of keratinocyte proteins in the epidermis and 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

Butterfly rash (malar rash)

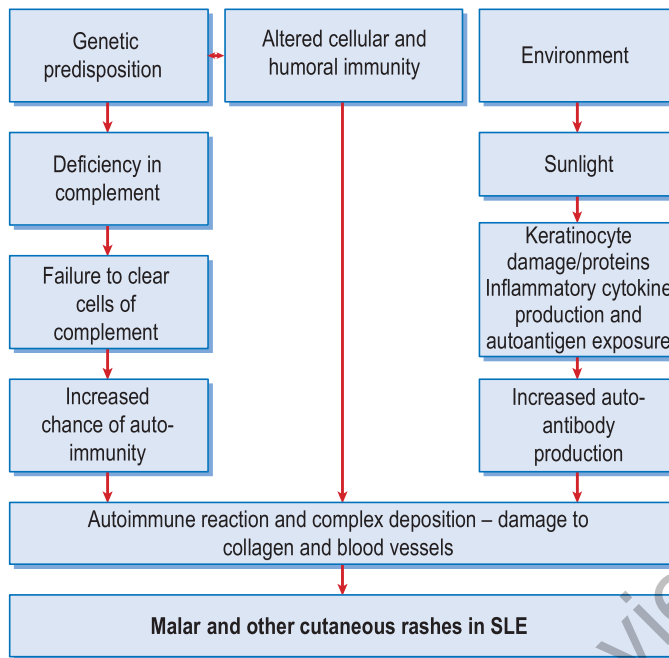


FIGURE 1.12

Mechanism of malar rash

in studies reviewing cutaneous manifestations of lupus.

It is likely that a combination of these factors leads to immune deposition in the skin, tissue damage, oedema and the characteristic malar rash.

Sign value

The malar rash is seen in approximately 40% of patients with SLE.³⁵ It has a sensitivity of 57% and specificity of 96% for SLE.³³ Its absence does not exclude the diagnosis of SLE.

Calcinosis/calcinosis cutis



FIGURE 1.13

Calcinosis
Hard, whitish nodules of the digit, representing dystrophic calcinosis in this patient with dermatomyositis.

Reproduced, with permission, from James WD, Berger T, Elston D. Andrews' Diseases of the Skin: Clinical Dermatology, 11th edn, Philadelphia: Saunders, 2011, Fig 26-12.

Description

Calcinosis refers to the formation or deposition of calcium in soft tissue (Figure 1.13). Calcinosis cutis more specifically refers to calcium deposits in the skin.

Associated conditions

Conditions associated with calcinosis may be classified as dystrophic, metastatic, iatrogenic, idiopathic or calciphylaxis.³⁶

- Dystrophic calcinosis
 - » Scleroderma
 - » Dermatomyositis
 - » SLE

- » Systemic sclerosis
- » Burns
- Metastatic
 - » Chronic renal failure – most common
 - » Due to hypercalcaemia or hyperphosphataemia of any cause
 - » 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.³⁶ Proposed mechanisms include the following:

- High local levels of alkaline phosphatase break down a pyrophosphate that normally inhibits calcification.³⁷
- Tissue breakdown may lead to denatured proteins that bind to phosphate. These phosphate–protein compounds may react with calcium, providing a nidus for calcification.³⁸

Charcot foot

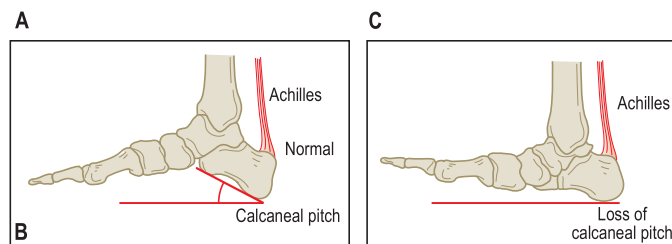


FIGURE 1.14

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.

Reproduced, with permission, from Mann JA, Ross SD, Chou LB, Chapter 9: Foot and ankle surgery. In: Skinner HB, Current Diagnosis & Treatment in Orthopedics, 4th edn, Fig 9-8. Available: <http://proxy14.use.hcn.com.au/content.aspx?aID=2321540> [10 Mar 2011].

Description

Charcot foot is a progressive destructive arthropathy of the soft tissues, bones and joints of the ankle and foot^{40,41} (Figure 1.14). In its early stages, it may present as unilateral foot oedema following minor trauma with an associated increase in temperature ($> 2^\circ$ from contralateral side) and erythema.⁴⁰ It is commonly associated with peripheral neuropathy, typically with diabetes. It can be difficult to differentiate from an infective process.

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'.

Associated conditions

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. So, 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 damage⁴² (Figure 1.15).

Under the *inflammatory theory*, when the same local insult occurs (microfracture, subluxation or fracture), inflammatory cytokines are released, including TNF- α and interleukin-1 β . These two cytokines have been shown to increase activation of RANK ligand (RANKL),⁴⁰ which in turn increases the transcription factor nuclear factor- κ B (NF- κ B).⁴¹ The net result of this is *stimulation of the maturation of osteoclasts*, which further eat away at bone (Figure 1.16). This predisposes the patient to engage in another vicious cycle of further

Lachman test

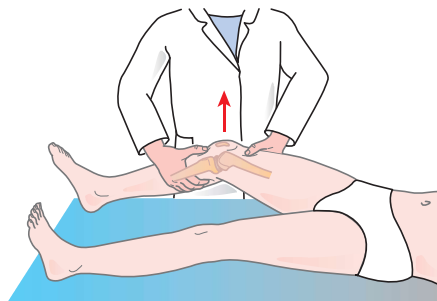


FIGURE 1.26

Lachman test of the 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 (Figure 1.26). The test is positive if there is anterior movement of the tibia without an abrupt stop.

Associated condition

- 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.

Livedo reticularis



FIGURE 1.27

Livedo reticularis – a net-like pattern, often erythematous or violaceous in colour

Reproduced, with permission, from Floege J, Johnson RJ, Feehally J. Comprehensive Clinical Nephrology, 4th edn, Philadelphia: Saunders, 2010, Fig 64-13.

Sign value

Lachman test is a reliable test for ACL injury but did not demonstrate superior validity to the anterior drawer sign in chronic ACL injury.² A high-quality meta-analysis of Lachman test reported a pooled sensitivity of 85% (95% CI 83–87), specificity of 94% (95% CI 92–95) and PLR of 5.³ This was supported by a subsequent meta-analysis, which reported sensitivity of 85% and specificity of 93% for Lachman test without concomitant ligamentous injury.²

However, in complete ACL tear and post-acute injury Lachman test is less reliable. In a sub-group analysis for complete tear the sensitivity was 68% (95% CI 54–79) and specificity was 79% (95% CI 51–93), with sensitivity of 79% (95% CI 57–80) and specificity of 77% (95% CI 53–91) with post-acute injuries,² thought to be due to increased discomfort with the examination. The pivot shift test is more reliable in these settings. It is important to note that multiple ligament knee injury may change the diagnostic accuracy of all ACL tests but is yet to be sufficiently studied.

Description

A macular, bluish/purple discolouration of the skin that has a lacy or net-like appearance (Figure 1.28).

Associated conditions

More common

- Primary or idiopathic livedo reticularis (LR)
- Hypothermia

Less common

- Secondary LR
 - Present in numerous disorders including:
 - Hypercoagulable state
 - » Antiphospholipid syndrome
 - » COVID-19 (thought to be due to hypercoagulable state given transient nature)
 - » Cryoglobulinaemia
 - » Multiple myeloma
 - » Deep vein thrombosis (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 effects
 - » Amantadine
 - » Quinine

plexus. LR 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.⁸⁴

In general, venodilatation is caused by altered autonomic nervous system function, circulating factors that cause 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,⁸⁴ which can be the result of decreased arteriolar inflow⁸⁵ 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.

General mechanisms

Arterioles arising from the dermis divide to form a capillary bed (Figure 1.28). These capillaries then drain into the venules of the venous

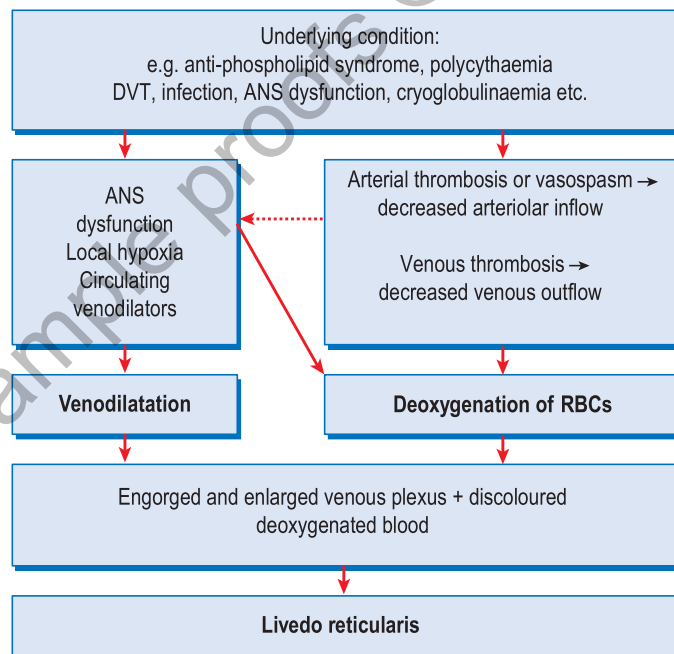


FIGURE 1.28

Mechanism of livedo reticularis

Varus deformity

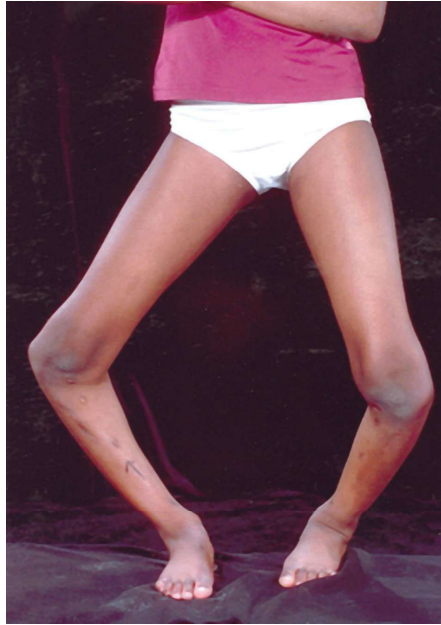


FIGURE 1.65

Bowing of both legs in infantile Blount's disease

Reproduced, with permission, from Harish HS, Punushottam GA, Wells L, Chapter 674: Torsional and angular deformities. In: Kliegman RM, Behrman RE, Jenson HB, et al. Nelson Textbook of Pediatrics, 18th edn, Philadelphia: Saunders, 2007, Fig 674-8.

Description

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

Associated conditions

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 110° (Figure 1.66).

Congenital

Congenital coxa vara may present in infancy or later in childhood (when it may be described as developmental coxa vara). 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.¹⁸³

Rickets

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

Perthes' disease

Although the underlying cause of Perthes' disease is unknown, there is a loss of blood supply to the femoral head with both biological and mechanical factors believed to play a role.¹⁸⁴ 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, although it can persist for up to 36 months.¹⁸⁵ 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 (tibia vara)^{185,186} (Figure 1.65). Obesity and ethnicity are thought to be risk factors for developing Blount's.^{186,187}

Hallux varus

Hallux varus is comprised of medial deviation of the first metatarsophalangeal (MTP) joint, supination of phalanx and

TABLE 1.6

Varus deformity-associated conditions

Hip	Knee	Ankle	Toe
Congenital disorders (e.g. cleidocranial dysplasia, Gaucher's disease)	Physiological – common	Trauma	Complication from bunion surgery
Perthes' disease	Blount's disease	Iatrogenic	Trauma
Developmental dysplasia of hip	Rickets	Congenital	Burn injury with contracture
Slipped capital femoral epiphysis (SCFE)	Trauma		Rheumatoid arthritis
Rickets	Infection		Psoriatic arthritis
Osteomyelitis	Tumour		Charcot–Marie–Tooth disease
Paget's disease	Skeletal dysplasia		Avascular necrosis
Trauma			

**FIGURE 1.66**

Metaphyseal chondrodysplasia, type Schmid
There is bilateral coxa vara, the metaphyses are splayed and irregular, and there is lateral bowing of the femora.

Reproduced, with permission, from Adam A, Dixon AK (eds). Grainger & Allison's Diagnostic Radiology, 5th edn, New York: Churchill Livingstone, 2008, Fig 67.13.

interphalangeal flexion or claw toe. It results from an imbalance between osseous, tendon and capsuloligamentous structures at the first MTP joint.¹⁸⁸

Hallux varus can be congenital or acquired with the latter being more common. The most common presentations include rheumatoid arthritis, trauma, burn with contracture and iatrogenic (classically secondary to surgery for hallux valgus).¹⁸⁸

Sign value

Aetiologies differ largely depending on the site of the deformity.

Wrist Hyperflexion and Abduction of the Thumb (WHAT) test

Description

The patient's wrist is moved into full flexion and ulnar abduction of the thumb. The examiner then asks the patient to resist pressure applied to the abducted thumb. The test is positive if pain is elicited during the action.

Associated condition

- De Quervain's tenosynovitis

Mechanism(s)

De Quervain's tenosynovitis (DCT) is an inflammatory condition and stenosis of the associated tendon sheaths of the contents of the first extensor synovial compartment: the tendons of abductor pollicis longus and extensor pollicis brevis.⁵⁸

Repetitive strain injury (particularly associated with gaming or repetitive typing/tool use in daily activities) 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.

There are several contributing factors to the development of DCT including:⁵⁸

- Anatomical variations
- Inflammation: increased expression of inflammatory mediators including neutrophil esterase, macrophages, TNF- α , IL-20 and

COX-2 have been identified in patients with DCT, and Kuo et al. identified a correlation with increased expression of these mediators and severity of disease, suggesting an inflammatory component.^{59,60} Shen et al. showed an increase in the expression of IL-1 α , IL-6 and VEGF, suggesting promotion of angiogenesis and chronic inflammation.⁶¹

- Oestrogen-binding receptors (EBRs) an inducer of COX-2 are also expressed in greater quantities in DCT, which may suggest the prominence in women of child-bearing age.⁶¹
- Myxoid degeneration: Examination of tendon sheaths associated with DCT demonstrates increased vascularity and mucopolysaccharide deposition, with only some showing lymphoid infiltration. This may potentially suggest that chronic inflammation overlays or interacts with an underlying myxoid degeneration of the tendon sheaths.⁵⁸
- Genetic factors
- Somatotropin hormone therapy increasing growth hormone and insulin-like growth factor 1 increasing thickening of flexor tendons and synovial oedema⁵⁸

Sign value

In a prospective study Goubau et al. reported the WHAT test had greater sensitivity and specificity when compared with Eichhoff's.⁵⁷ WHAT

Yergason's sign

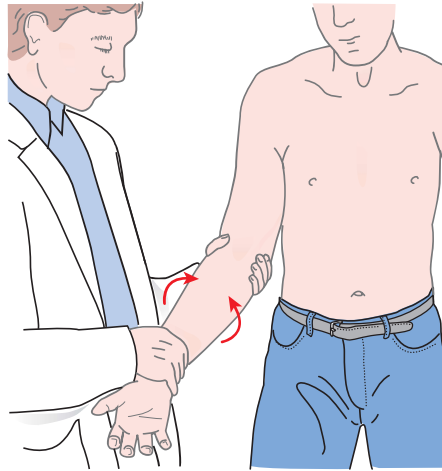


FIGURE 1.67
Yergason's sign

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 (Figure 1.67).

Associated conditions

- Biceps tendonitis
- SLAP lesion (**S**uperior **L**abral tear from **A**nterior to **P**osterior) – 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

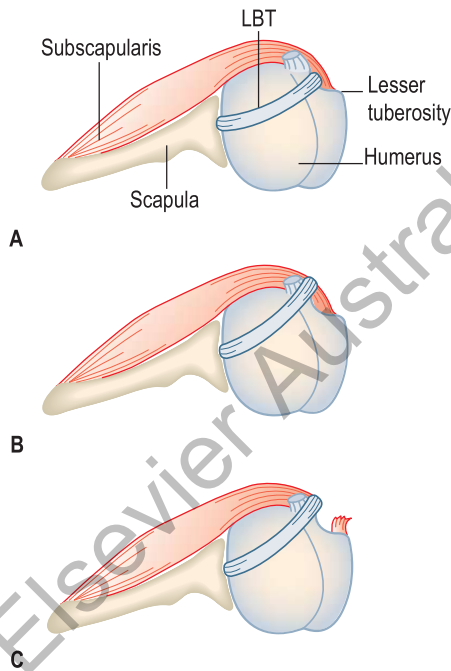


FIGURE 1.68

Yergason's sign pathoanatomy

Overhead view of the subscapularis muscle, long head of the biceps tendon (LBT) and bicipital groove

A Intact structure depicting normal anatomy; **B** partial tear of the subscapularis tendon from the attachment on the lesser tubercle, with the LBT subluxed over the lesser tubercle into the subscapularis muscle; **C** complete tear of the subscapularis tendon from the attachment on the lesser tubercle, with the LBT subluxed over the lesser tuberosity and the subscapularis tendon.

Based on Pettit RW, Sailor S.R, Lentell G, et al., Yergason's test: Discrepancies in description and implications for diagnosing biceps subluxation. Athletic Training Edu J 2008;3(4):143–7.

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 place¹⁵³ (Figure 1.68). If this fibrous extension is ruptured, the biceps tendon is susceptible to subluxation.

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