Chapter 4
Osteoarthritis

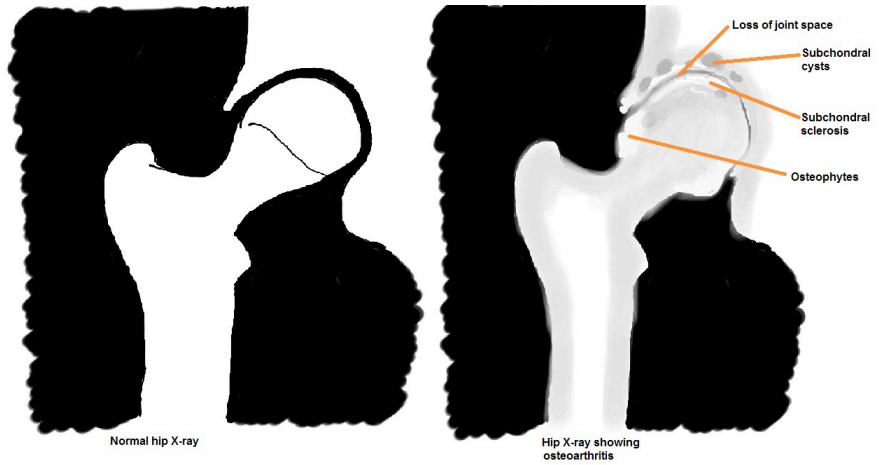
Degenerative joint disease is a mechanical abnormality of the joints leading to wear of the articular cartilage with eventual full thickness loss with involvement of the subchondral bone. Estimates indicate that about 5 million people in the UK have X-ray evidence of moderate to severe osteoarthritis of the hands, knees and hips, with 36 million working days lost in 1999-2000 in Great Britain, representing £3.2 billion lost production. The cost to health and social services during this time period for treatment of all types of arthritis (including inflammatory) was £5.5 billion with prescription costs totalling £341 million and hip and knee replacements totalling £405 million.

Symptoms and signs include pain, reduced mobility of the joint, joint line tenderness, reduced range of motion and an effusion. Commonly affected joints include the knees (most commonly affected joint) and hips, hands and feet although any cartilaginous joint could be involved. Osteoarthritis is rarely symmetrical and is not as deforming or destructive as inflammatory arthopathies.

In the hands, bony enlargements on the distal interphalangeal joints (Heberden's nodes) and proximal interphalangeal joints (Bouchard's nodes) may be noted.

Diagnosis is made through history, clinical examination and radiographs. The characteristic findings on X-rays (which can be remembered with he mnemonic "**LOSS**"), are:

L - Loss of joint space
O - Osteophytes
S - Subchondral cysts
S - Subchondral sclerosis



X-rays are usually all that is required for investigation. However, if there is complex deformity, other imaging may include CT to determine the degree of bone loss and any malalignement, MRI to determine the degree of cartilage loss and ligament integrity, and isotope bone scan to detect any underlying bone abnormality (tumour or infection).

Osteoarthritis may be *primary* or *secondary*.

Primary Osteoarthritis

This is a chronic degenerative disorder that is related to age (usually over 45 years of age), although there is now emerging evidence also linking it to a genetic predisposition. Reduced proteoglycan content in the articular cartilage and an increase in water content leads to the cartilage being more vulnerable to wear. The wear process includes degradation of the collagen fibres, leading to further degeneration of the cartilage.

With deterioration and loss of the joint bearing surface, subchondral bone may be exposed. As weight-bearing occurs on the exposed bone, it will become hardened, eburnated and sclerotic. The cartilage loss also leads to formation of osteophytes and subchondral cysts.

Secondary Osteoarthritis

The findings are similar to primary osteoarthritis but caused by mechanical or structural abnormalities within the limb or joint. Examples include:

1. Intra-articular fractures
2. Limb malalignment following a fracture distant from the joint
3. Inflammatory joint disease (rheumatoid arthritis, gout)
4. Septic arthritis (infective process rapidly destroys cartilage)
5. Ligamentous instability (joint instability leading to rapid cartilage wear)

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Management

Non-operative

1. Life-style modification with weight-loss, graded exercise, mobility aids (walking stick or frame), physiotherapy and occupational therapy with home modifications.

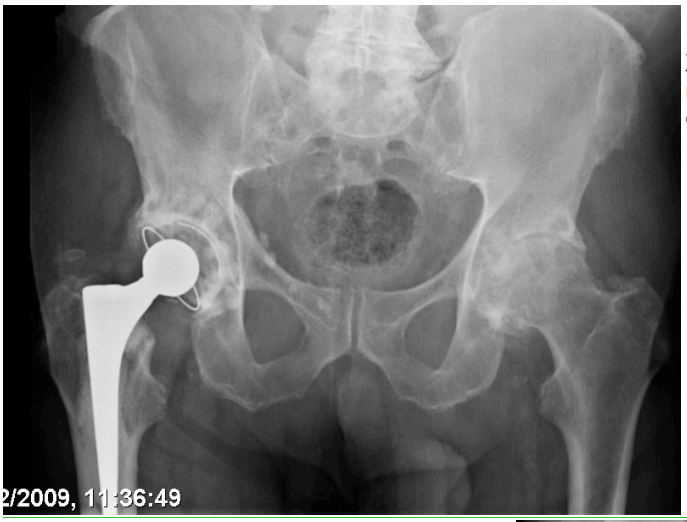
2. Analgesia usage, including paracetamol, NSAIDs and opioids, escalating according to the WHO pain ladder.

3. Joint injections, including steroid and hyaluronic acid injections, may give some short-term benefit but are associated with risks and are falling out of favour.

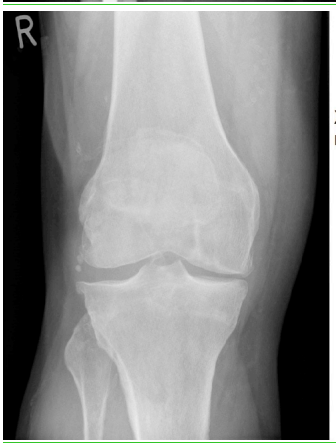
Operative
There are no procedures proven to lead to cartilage regeneration. Evidence shows that arthroscopic debridement for knee arthritis gives no prolonged benefit.

Surgery involves excision of the arthritic joint and replacement with an artificial joint articulation. The most common procedures are total hip arthroplasty (59,000 performed in UK per year) and total knee arthroplasty (62,000 performed in UK per year) which give

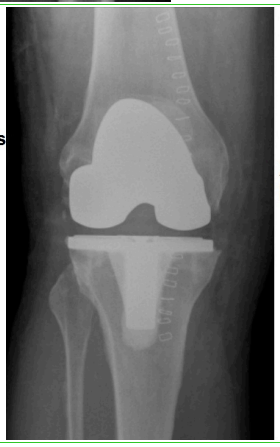
predictably good pain relief and mobility, with excellent survival rates up to 10-20 years after surgery.



X-ray: Total hip replacement right hip, osteoarthritis left hip



X-ray: right knee medial osteoarthritis



X-ray: right total knee replacement

Chapter 5
Inflammatory and infective arthritis

Rheumatoid arthritis (RA)

This is the most common form of inflammatory arthritis, affecting 3% of women and 1% of men. RA is a symmetrical, deforming polyarthritis. The disease process is a cell-mediated (mononuclear cells) immune response within the synovium of joints, leading to inflammation and destruction initially of the peri-articular soft tissues and later cartilage and bone. Inflammatory mediators, including cytokines (particularly IL-1 and TNF-alpha) and lymphokines, initiate a destructive cascade within the joint, degrading the cartilage and

damaging surrounding tissues. Inflamed synovium has a proliferation of blood vessels forming the ‘pannus’ which invades the joint and further damages the cartilage.

Clinically, this manifests as insidious onset of joint swelling and nodules with associated stiffness, followed by joint pain with radiographic changes of erosive joint narrowing and destruction with periarticular osteopenia +/- subluxation or dislocation. X-ray changes are commonly seen at MCPJs, PIPJs and carpal bones of hands, wrists, cervical spine, knees and hips (the latter commonly with protrusio = medial deepening of acetabulum).

Laboratory tests include:

Raised CRP and ESR
Rheumatoid factor (Immunoglobulin M) - +ve in 80% of patients
HLA-DR4 - +ve in 70% of patients

Systemic manifestations include:

Vasculitis
Pericarditis
Pulmonary fibrosis
Felty's syndrome (RA with splenomegaly and leukopenia)
Still's disease (acute onset juvenile RA with fever, rash and splenomegaly)
Sjögren's syndrome (autoimmune exocrinopathy often associated with RA leading to lymphoid proliferation and decreased salivary and lacrimal secretion - keratoconjunctivitis sicca complex)

Diagnostic criteria from the American College of Rheumatology include morning stiffness, joint swelling, arthritis of joints of the fingers lasting >6 weeks, nodules, positive laboratory results and specific X-ray findings (see above).

Common hand signs include:

Synovitis leading to attenuation of finger stabilising structures, causing:
- **Boutonnière** deformity - proximal interphalangeal joint (PIPJ) flexion with distal interphalangeal (DIPJ) hyperextension
- **Swan-neck** deformity - PIPJ hyperextension with DIPJ flexion

- **Metacarpo-phalangeal joints (MCPJ) ulnar and volar deviation and subluxation**

Tenosynovitis of hands and wrist tendon sheaths (leading to swelling, pain and stiffness and eventually tendon rupture)

Rheumatoid wrist with tenosynovitis eventually leading to tendon subluxation, combined with joint destruction causing:
- **Carpus subluxation in a volar and ulnar direction**
- **Dorsal subluxation of the ulnar head (caput ulnae)**

Management

The goals are:

Control pain and synovitis
Maintain joint function
Prevent deformity

Treatment is in a multidisciplinary setting and includes therapeutic drugs, physiotherapy (to maintain joint range of motion and strength), occupational therapy (for splints and functional aids) and occasionally surgery.

Drug therapy used to start with NSAIDs and then progress to antimalarials, remittent agents (sulfasalazine, gold and penicillamine), steroids and cytotoxic drugs. Recently, rheumatologists have shown the benefit from treating much earlier in the disease process with new **disease-modifying anti-rheumatic drugs (DMARDs)**. These include methotrexate, azothioprine and TNF-alpha inhibitors (including infliximab and etanercept).

Surgery, in the form of synovectomy and joint replacement, has become increasingly rare due to this more aggressive therapeutic regime. Patients requiring surgery under general anaesthetic require evaluation of cervical spine stability with pre-operative radiographs. Most DMARDs are halted a few weeks prior to surgery. Surgery now is predominantly for hand tendon repair or re-routing, finger and wrist joint replacement or fusion and total knee or hip replacement.

Juvenile Rheumatoid Arthritis - see paediatric chapter

Systemic Lupus Erythatosus (SLE)

This is a chronic inflammatory disease of unknown origin probably related to immune complexes. It is common in Afro-Caribbean women. 75% patients have joint involvement with similar findings to rheumatoid arthritis, with acutely swollen, tender PIPJs, MCPJs, wrists and other joints.

Systemic findings:

Fever
Butterfly malar rash
Pancytopenia
Pericarditis
Nephritis
Polyarthritis

Laboratory tests:

Raised CRP and ESR
Antinuclear antibody (ANA) - usually +ve
HLA-DR2 & 3 - +ve in ~50% of patients
Rheumatoid factor - may be +ve

Management

Same as for rheumatoid arthritis, using the same multidisciplinary approach and drugs.

Polymyalgia rheumatica

Aching and joint stiffness, particularly around the shoulders and hips. Cause unknown. Associated with headache, malaise and anaemia. May have raised ESR, anaemia and raised alkaline phosphatase.

Treated symptomatically with NSAIDs and analgesia with steroids for severe cases.

Associated with temporal arteritis (requires urgent temporal artery biopsy and steroids to reduce risk of blindness).

Seronegative arthropathies

Characterised by -ve rheumatoid factor and +ve HLA-B27. Typically occur at ligament insertions into bone (enthesopathy) or spine (spondyloarthropathy).

Ankylosing spondylitis (AS)

AS typically presents with insidious onset of back pain and morning stiffness with hip pain in males between 30-50 years of age. Bilateral sacroiliitis +/- anterior uveitis with a positive HLA-B27 is diagnostic. The condition progresses over several years with increasing spinal rigidity and kyphotic deformity, eventually leading to the ‘chin on the chest’ position. Spine X-rays demonstrate obliteration of sacroiliac joints with squaring and fusion of vertebrae. The spine becomes one rigid, brittle column and is prone to fracture after trivial falls with a high risk of epidural haemorrhage and neurological impairment.

Systemic findings:

Iritis
Aortitis
Colitis
Amyloidosis
Sarcoidosis
Pulmonary fibrosis and restricted chest excursion
Heart disease

Management

Hip pain and stiffness with spinal deformity often improve with bilateral total hip replacement. Clinicians must maintain a high degree of suspicion for spine fracture after injury - if in doubt, CT scan the whole spine. Spine fractures usually require fixation by a spinal surgeon in a specialist centre.

Reiter's syndrome

This presents in young men with conjunctivitis, urethritis and oligoarthritis ("can't see, pee or bend the knee"!). It usually causes acute single joint swelling and pain (commonly knee), which may recur. Most are HLA-B27 positive and many chronic patients have sacroiliitis. Rheumatoid factor is negative.

Treatment involves physiotherapy and NSAIDs/analgesia.

Psoriatic arthritis

This commonly causes swelling and pain in the small joints of hands and feet, usually in young men. It affects between 5-10% patients with psoriasis and about half are HLA-B27 positive. Pitting is seen in fingernails with swollen "sausage" digits and DIPJ deformity and erosion.

Treatment is similar to rheumatoid arthritis.

Crystal deposition disease

This presents with recurrent attacks of exquisitely painful single joint arthritis. It commonly affects the lower extremity, especially the great toe. Razor-sharp crystals form within the synovium, leading to inflammation and pain with eventual joint destruction due to mechanical wear.

Gout

A disorder of nucleic acid metabolism leading to hyperuricaemia and monosodium urate crystal deposition. Serum urate may be elevated but this is not diagnostic. Joint fluid microscopy shows intracellular **crystals that are strongly negatively birefringent**.

Treatment during acute attack is with indomethacin or other NSAIDs. Allopurinol (a xanthine oxidase inhibitor) is used to lower serum urate levels in chronic gout but stopped during acute flares.

Pseudogout

Calcium pyrophosphate crystal deposition due to a disorder of pyrophosphate metabolism can be mistaken for gout. Crystals are **weakly positively birefringent** under microscopy. Pseudogout may lead to chondrocalcinosis (deposition of calcium in cartilage) within joints, especially the knee. Treatment is with NSAIDs symptomatically.

Septic arthritis

This must be suspected in any acutely swollen, painful and stiff joint and must be ruled out as an emergency. Infection within a joint rapidly kills cartilage, which if left to continue, can completely destroy a joint within hours.

Septic arthritis occurs following haematogenous spread or by extension of osteomyelitis and is common in children. Risk factors in adults include immunocompromise, diabetes mellitus, rheumatoid arthritis, intravenous drug users and previous invasive procedures into an infected joint.

Clinically the joint is hot, swollen, red with an effusion and the patient is in extreme pain with any active or passive movements. Suspicion is raised with fever, increased CRP, white cell count (specifically neutrophils in bacterial infection) and ESR. X-rays may be normal. Confirmation of diagnosis is by joint aspiration (under sterile conditions) with urgent Gram stain and microscopy (for organisms and crystals).

The most common organisms are *Staphylococcus aureus,* streptococci and Gram negative bacilli.

Management

This is with rapid diagnosis followed by urgent surgical joint debridement and washout. At the time of surgery, multiple tissue samples are sent for microscopy and culture prior to starting empirical antibiotic therapy dependent on local hospital antibiotic policy (usually a third generation cephalosporin +/- penicillinase resistant penicillin).

Once culture and sensitivities have been finalised, antibiotics may be altered as needed. Further joint washout and debridement may be required at 48-72 hours if joint pain and swelling is not improving. Antibiotics may continue for up to 6-8 weeks, dependent on the joint remaining quiescent and the return of all inflammatory markers and clinical signs to normal levels.

Chapter 6 - Other joint disorders

There are a number of common disorders of the joints that do not fit into the categories in this book. They are discussed in this chapter to cover the pathology that is regularly seen in musculoskeletal clinics.

Shoulder impingement

The tendon of supraspinatis, one of the rotator cuff muscles, runs under the acromion and the acromioclavicular joint (ACJ) and functions to abduct the shoulder. The tendon is protected from the undersurface of the acromion by a bursa that allows it to glide smoothly in this tight space. Anatomical abnormality in the shape of the acromion or degeneration with formation of spurs on the underside of the ACJ can lead to the tendon rubbing painfully against these structures, leading to inflammation within the bursa and tendon and possibly to eventual tendon damage and rupture. Young patients presenting with impingement must have shoulder instability ruled out as a cause of their impingement.

The patient presents with pain upon initiating abduction and worsening with overhead movements. The impingement test will be positive with limited abduction of the shoulder. X-rays may show a downward sloping acromion or ACJ spurring. MRI shows inflammation within the tendon and bursa and will determine if there is overt damage to the tendon.

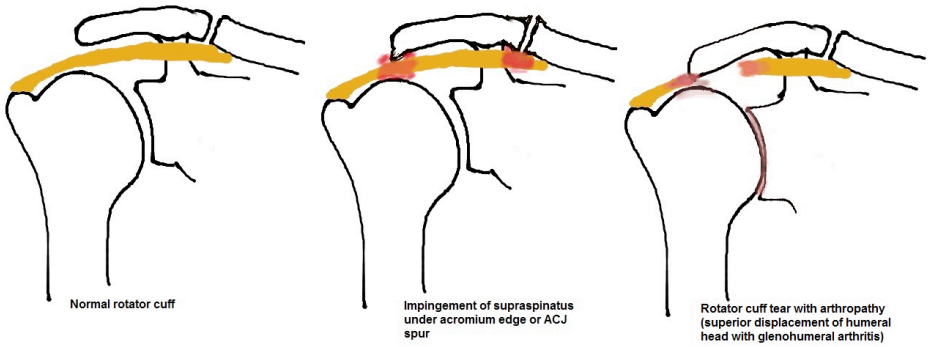
Treatment is with analgesia and physiotherapy. Steroid injections into the subacromial bursa may help relieve symptoms enough for physiotherapy to progress. Continued painful symptoms despite these conservative measures may be treated with arthroscopic subacromial decompression in which the undersurface of the acromion and ACJ are shaved and the inflamed bursa excised. After this procedure, symptoms may take up to 3 months to resolve.

Rotator cuff tear

There are 2 major causes of rotator cuff tears: failure due to repetitive stresses seen in young athletes (rare and treated with physiotherapy) and degeneration thought to occur due to continued subacromial impingement, with a partial tear progressing to a full-thickness tear and eventually rotator cuff arthropathy (secondary arthritis of the shoulder joint) in elderly patients. A third of people over the age of 60 will have some degree of rotator cuff tear, some more symptomatic than others.

Patients describe an insidious onset of pain and weakness with pain worsened with overhead movements. Examination will reveal reduced range of motion and weakness on testing of the specific rotator cuff muscles. X-rays may be normal but chronic disease may show superior migration of the humeral head (due to the unresisted pull of deltoid) or evidence of glenohumeral arthritis.

Treatment depends on the presentation. Chronic cuff tears are treated initially with analgesia and physiotherapy with an aggressive rotator cuff strengthening programme, possibly with subacromial steroid injections to help control pain. Chronic, symptomatic tears that have had a full rehabilitation programme may benefit from surgical repair. Acute rotator cuff tears should have an early surgical repair. Repair is either open or arthroscopic and relies on suture anchors to reattach the rotator tendons to their normal position. This is usually combined with subacromial decompression. Following surgery, immobilisation and rehabilitation follows a protracted course of around 6-9 months but results are generally very good.



Shoulder dislocation

The shoulder has an extensive range of motion, relying predominantly on soft tissue structures and the rotator cuff muscles for stability. Because of this, it is at risk of becoming unstable and is the most frequently dislocated joint in the human body. The most common direction of dislocation is anterior (95%), usually occurring with a sudden force applied to an abducted, externally rotated arm. Posterior dislocation is rare and easy to miss, sometimes occurring due to epileptic seizure, electroconvulsive therapy or electrocution.

Treatment requires urgent reduction utilising analgesia and sedation with various traction manoeuvres described. Assessment of axillary nerve function (sensation in the regimental badge region – known as the stars and stripes area in the US - and deltoid motor function) must be performed before and after reduction as this nerve may be damaged during dislocation or reduction. The shoulder is immobilised in a sling (with some controversy regarding a sling in external rotation versus internal rotation), followed by physiotherapy led rotator cuff strengthening exercises.

A dislocation will damage the soft tissues and capsule that are so vital to the stability of the shoulder joint. Repeated dislocations may also lead to injury to the bony structures of the glenoid and humeral head, making reduction and subsequent stability more difficult to achieve. The younger the age of first dislocation, the higher the risk of subsequent dislocations. Multiple dislocations, particularly in young patients, may warrant surgical stabilisation to prevent recurrence and this may be performed as open or arthroscopic surgery. If there is bony involvement, this needs to be dealt with at the time of surgery or the resulting stability may be compromised.

Tennis elbow

Repetitive rotation with the forearm in extension may result in a micro-tear of the common extensor origin at the elbow (lateral epicondyle) with a deranged healing response, leading to a chronic inflammatory response. This leads to pain and localised tenderness, worsened with resisted wrist extension.

Initial treatment involves activity modification, compression braces, stretching exercises and ultrasonic massage, and NSAIDs. Localised steroid injections combined with these other conservative measures may be beneficial in up to 95% of cases. Unremitting symptoms may require open surgical debridement.

Carpal tunnel syndrome

Median nerve compression in the carpal tunnel is the most common entrapment neuropathy in the upper limb, causing pain and numbness in the distribution of the nerve. The carpal tunnel has 3 sides which are rigid bony structures, its floor being the proximal carpal row, with the side walls of the scaphoid and trapezium radially and the pisiform and hook of hamate ulnarly. The roof is made of the transverse carpal ligament, another rigid structure. Through this tunnel runs the median nerve, accompanied by 9 flexor tendons.

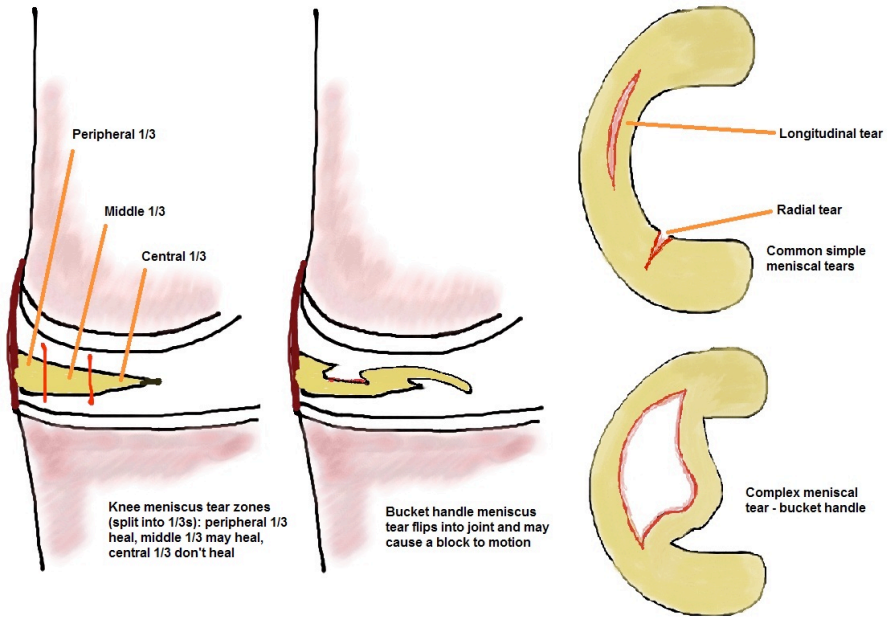
Any change in the volume of the tunnel, for example, due to swelling (rheumatoid arthritis), trauma (distal radius fracture and haematoma) or mass lesion (lipoma or ganglion) will compress the nerve, leading to symptoms, although the majority of carpal tunnel syndrome cases are idiopathic.

Patients complain of intermittent numbness with pins and needles affecting the thumb, index, middle and radial half of the ring finger. There may be associated weakness and wasting of the thenar eminence muscles. Physical tests include Phalen's test (holding wrist in flexion for 60 seconds to bring on symptoms) and Tinel's test (tapping over carpal tunnel to exacerbate symptoms) and may be reinforced with nerve conduction testing.

Treatment may be attempted with splints or steroid injections but, with continued symptoms or if there is evidence of nerve denervation (thenar wasting or weakness), surgical decompression should be offered. This is performed under local anaesthetic, which allows complete division of the transverse carpal ligament, relieving the nerve compression. Symptoms may not improve but should not worsen and it may take months for some recovery to be seen. Risks include recurrence, nerve damage and scar sensitivity.

Knee meniscal tear

Menisci are C-shaped medial and lateral cartilages that play an important role in knee mechanics, providing load-transmission, joint lubrication, stability and possibly proprioceptive function to the knee. Damage may occur in a young sports person during a twisting injury, often associated with an ACL tear, or may be seen in older patients as a result of degeneration of the biomechanical structure of the meniscus. Tears may be simple radial or longitudinal tears or more complex "bucket-handle" tears with a portion of the damaged meniscus flipping into the joint. The peripheral 1/3 of the meniscus is vascular and tears involving this region have a chance of healing whilst the central 2/3 are unlikely to heal due to avascularity.



A patient with an acute meniscal tear will describe a twisting injury with sudden onset of pain on either the medial or lateral side of the joint with swelling developing over 12-24 hours. The patient will present with pain and a limited range of motion and an effusion. Investigate initially with X-rays to rule out a fracture but continued symptoms are investigated with MRI. MRI will show the extent of the tear and associated ligament disruption or bony injury.

Treatment may be conservative if the pain settles and the patient can achieve a full range of motion (particularly full extension) once the effusion has resolved. If there is evidence of a peripheral (repairable) tear or there is a block to full extension (due to a flipped bucket handle tear), a knee arthroscopy with meniscal repair (via sutures) or partial excision may be performed. Symptomatic degenerate, chronic meniscal tears (usually seen in patients over 40 years of age), often involving the posterior horn of the medial meniscus, may be treated with arthroscopic meniscal debridement. Total excision of a meniscus leads to early development of osteoarthritis and is never performed.

Anterior Cruciate Ligament (ACL) injury

The ACL is an important stabilising structure of the knee, providing anterior translational and rotational stability. Rupture occurs in sportsmen with a non-contact twisting injury resulting in sudden pain and immediate large swelling/haemarthrosis.

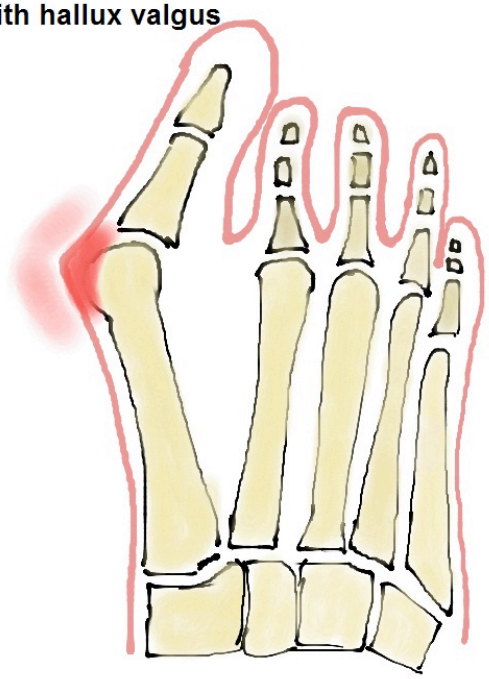
Examination will reveal positive anterior drawer and Lachman's tests. MRI will show the extent of associated meniscal and bony injuries and confirm ACL rupture. Treatment commences with achieving full range of motion in the knee. Any attempt at surgery prior to this may result in arthrofibrosis with severe chronic stiffness and limited motion of the knee. Having achieved full motion, the patient will commence ACL rehabilitation under the guidance of physiotherapists, working on quadriceps strengthening and hamstring control. If the patient suffers knee instability despite this rehabilitation, ACL reconstruction surgery may be considered.

ACL reconstruction is classically performed arthroscopically and may utilise tendons harvested from the patient's hamstrings or part of their patellar tendon. Tunnels are drilled in the tibia and femur at the sites of the native ACL attachments and the tendon graft secured at these points to form a substitute cruciate ligament. Prolonged rehabilitation is required following this surgery to allow return to sport.

Hallux Valgus/Bunions

Varus deviation of the hallux (big toe) metatarsal will lead to a valgus angulation at the metatarsophalangeal joint (MTPJ), termed hallux valgus. This angulation will result in a prominent bump (inflamed capsule, bursal tissue and joint osteophytes) on the medial side of the joint, which is referred to as a bunion, and may cause pain and rubbing on shoe-wear. After investigation with X-rays, treatment is decided on according to the patient's symptoms.

Inflammed bunion with hallux valgus



Hallux valgus may be treated non-operatively with accommodative wide shoes and avoiding tight shoes. Surgery corrects the underlying deformity of the great toe to alleviate symptoms and should never be performed for cosmetic reasons alone. There are many described procedures that involve correcting soft tissue balance around the MTPJ and excising the bunion in minor deformity. Correction of greater deformity involves cutting and realigning the metatarsal and sometimes the proximal phalanx, combined with soft tissue procedures. All of these procedures have a risk of recurrence of deformity and hallux valgus may eventually lead to MTPJ osteoarthritis, which if symptomatic, can be treated with fusion of the joint in the corrected position.

Chapter 7
Spine

Back pain investigations

A thorough history and clinical examination will provide the clinician with a diagnosis in most cases. History must include specific questions about the ‘red flags’ which, if present, warrant further investigation to ensure there is no serious underlying pathology.

Spine red flags:

1. New onset of pain age <15 or >50 years of age
2. Unexpected weight loss
3. Recent illness or systemic infection
4. Previous long term steroid use
5. Previous history of cancer
6. Fever and malaise
7. Pain at rest/night pain
8. Urinary retention and constipation
9. Saddle paraesthesia

Back pain presenting with any of these signs requires urgent investigation with plain X-rays of the spine (which could show evidence of fracture and infection and possibly metastases), bloods (including U&E, CRP, ESR, FBC, Ca^{2+}) and myeloma screen (urine protein electrophoresis looking for Bence-Jones proteins and serum protein electrophoresis). An MRI is a sensitive investigation that can be used to evaluate for important anatomical variations, fractures, tumours, infection and disc pathology that may require urgent treatment.

Lumbar herniation plus radiculopathy (sciatica)

With age, the intervertebral disc becomes dehydrated and the strength and resistance to compressive forces diminishes as the structural proteins and proteoglycans degrade. A tear in the outer fibrous lining of the disc will allow the gelatinous nucleus material to extrude out of the disc. This extruded material or 'herniation' may place pressure on the nerve roots crossing the disc, leading to pain radiating to the region supplied by that nerve root.

Most common in the lower lumbar spine due to the highest motion and load-bearing being through these segments, disc herniation is most often at the levels L4-5 and L5-S1. The radiation of pain down the affected limb in the distribution of a specific nerve root is referred to as 'radicular'. If the pain affects the lower nerve roots, it may be referred to as "sciatica'.

Clinical examination will elicit back pain and probable muscle spasm with limited motion in the lumbar spine combined with pain radiating down the leg to the specific dermatome supplied by the affected nerve root. The pain may be burning or pins and needles and may be accompanied by paraesthesia and weakness in the muscles in that myotome. Nerve root tension signs are elicited by the straight leg raise test (L4-5, L5-S1) or the femoral stretch test (L2-3, L3-4) and indicate that the affected nerve root is tethered by the herniated disc material.

After initial presentation, management with analgesia and a maximum of a week of rest followed by a graduated exercise program under physiotherapy guidance will see 90% of patients recover within 3 months. If there is no improvement, an MRI is required to rule out more sinister causes of pain and to determine the site of herniation. This will allow for treatment with lumbar epidural or X-ray guided nerve-root steroid injections.

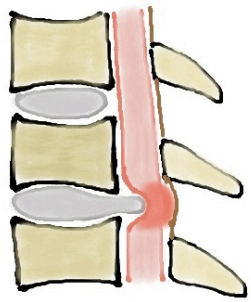
With continued symptoms, in carefully selected patients, surgical discectomy has been shown to give a more rapid resolution of symptoms but similar long-term outcomes to non-

surgically treated patients. Surgery does have the rare but potentially fatal complication of injury to the aorta and risk of permanent nerve root injury, so must not be embarked upon lightly.

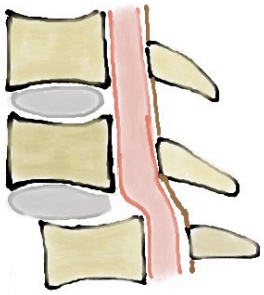
Cauda equina syndrome

This is an orthopaedic emergency. A large, central disc herniation at the level of the cauda equina (which are the nerve roots that continue in the spinal canal after the termination of the spinal cord at the level of the L1-2 disc) compressing these nerve roots, patients will usually present with bilateral buttock and lower limb pain (may be pain free) with urinary retention, saddle anaesthesia and possibly constipation. A vital part of the clinical examination must include digital rectal examination to assess for anal sensation and tone.

The sacral nerve roots are particularly sensitive to ischaemia and delayed decompression of more than 6-12 hours may lead to total loss of function. This can lead to lifelong disability with loss of anal sphincter and bladder control leading to incontinence. Urgent investigation with MRI and surgical decompression give the best prognosis. Delay to diagnosis and treatment is common and leads to a very high number of successful, costly medical negligence claims.



Lumbar disc prolapse - lateral position of displacement will cause compression of nerve roots. Central displacement may cause compression of the cauda equina



Spondylolisthesis - slip of one vertebrae upon another. May lead to central compression

Lumbar disc degeneration

Low back pain with limited leg pain is often caused by degeneration of the intervertebral discs with no herniation. Investigated with MRI, an abnormal disc will appear dehydrated and the disc space will be narrowed. Often multiple levels are involved. A discogram may be performed to confirm the causative disc - a high volume of saline is injected into the disc and the levels above and below. Pain upon injection of the suspected disc with an absence of pain upon injection into the other discs confirms the affected level and diagnosis.

Treatment is initiated with analgesia and physical therapy. If a prolonged trial of conservative treatment fails, removal of the pain-causing disc (discectomy), combined with fusion of the vertebrae on either side of it, may be considered.

Central canal stenosis

This is narrowing of the spinal canal producing a combination of back and leg pain, due to compression of nerve roots and the thecal sac. It usually occurs in late middle age due to a combination of central disc bulge, hypertrophied facet joints and ligamentum flavum decreasing the central volume of the canal. The canal volume is smallest with the spine extended but flexion increases the available space and patients often find flexing their backs relieves pain.

Symptoms are described as neurogenic claudication with pain worsening with walking but progressing from proximal to distal, pain relieved by sitting or bending and lack of symptoms with cycling (compare with vascular claudication with pain progressing distal to proximal, pain alleviated by stopping activity and pain with cycling).

Investigation is with MRI. Management initiates with analgesia and flexion exercises and may be followed with lumbar epidural steroid injections. Persistent pain impairing the patient's quality of life may lead to surgery, which involves complete decompression of the spine and may require fusion of the affected levels.

Spondylolisthesis

This is a forward slip of one vertebrae on another. It is caused by pathology at or around the facet joints that stabilise the spine, including abnormal development, stress fracture, degenerative disease and acute fracture. Young patients with mild slips require no treatment but should avoid contact sports. If the slip is more severe, there is a risk of progression and it may cause radicular symptoms or secondary central canal stenosis with corresponding symptoms. These patients may benefit from surgical fusion *in situ*. Symptomatic degenerative spondylolisthesis requires surgical decompression and fusion in situ.

Scoliosis

A curve in the spine, which although a complex 3 dimensional deformity, appears as a lateral deviation with an S- or C-shaped curve. Scoliosis is divided into adult and childhood.

Childhood scoliosis is commonly idiopathic and adolescent but may be related to cerebral palsy and other neuromuscular disorders or congenital spinal abnormalities. The age of presentation, skeletal maturity and rate of progression are the most important factors when deciding upon treatment. Lesser curves and slower progression may be treated conservatively with regular monitoring and intermediate curves with custom-made thoracolumbosacral braces. The younger the patient is, and the quicker the curve is progressing, the higher the chance of a poor outcome and greater likelihood of requiring surgical correction and fusion.

Adult scoliosis is usually more symptomatic and idiopathic (other causes include degenerate, neuromuscular, post-traumatic and post-surgical). Patients will usually present with pain and a cosmetic deformity. Most are managed non-operatively with analgesia and physical therapy and possibly facet joint injections. Surgery is usually in young, symptomatic adults with progressive, large curves and requires multi-level realignment and fusion.

Infections of the spine

Disc space infections are commonly seen in children, with *Staphylococcus aureus* being the usual organism. Back pain and tenderness, restricted range of motion and poor walking, sitting or standing represent the clinical picture. Blood tests with raised inflammatory markers and an MRI are diagnostic. Treatment is with appropriate antibiotics.

Vertebral osteomyelitis is becoming more prevalent, typically in elderly, debilitated patients and intravenous drug abusers. Other risk factors include pneumonia, urinary tract infection, diabetes and HIV. The organism is usually seeded via a haematogenous route. Pain, localised tenderness and muscle spasm are usually evident. Plain X-rays may show erosion of vertebral end-plates and disc destruction but MRI and CT-guided aspiration are diagnostic. 6-12 weeks of intravenous antibiotics is the treatment of choice.

Spinal tuberculosis can lead to destruction of several adjacent vertebrae. Abscess formation, neurological compromise due to spinal canal compression (by abscess or bone fragments), meningitis and progressive deformity may occur. Multi-drug therapy is the mainstay of treatment.

Fractures

Cervical spine injuries carry risk of spinal cord injury, so the first priority is immobilisation using a hard collar and blocks as per the ATLS protocol. Investigation is with CT to gain information on the extent of bony injury and MRI for information on spinal cord and ligament injury. Displaced cervical spine fractures and dislocations require reduction and, depending on stability, may require surgical stabilisation (especially if ligamentous disruption), fusion or immobilisation with specialised collars or braces.

Thoracic and lumbar spine fractures are investigated in a similar manner. Anatomically, the spine is divided from front to back into thirds, representing the anterior, middle and posterior columns. Compression fractures (often seen in osteoporosis) involve the anterior column and usually require a brace for 12 weeks. Burst fractures may involve all 3 columns and usually are managed in a brace for a similar period. However, a kyphosis deformity, neurological deficit, spinal canal compromise of > 50% or > 50% loss of anterior column height are indications for surgical stabilisation. Other injury patterns including fracture-dislocations and flexion-distraction injuries are routinely treated with surgical stabilisation.

Traumatic cord injury

Injury to the spinal cord may be complete or incomplete and usually occurs in young males involved in motor vehicle accidents, falls and sports related injuries. Initially spinal shock results in a 24-72 hour period of paralysis, hypotonia and areflexia. When it resolves,

spasticity, hyperreflexia and clonus progress and only after full resolution can the level of injury be determined. The functional level is determined by the most distal intact functioning dermatome and motor level (with a minimum of 'fair' motor power).

Cord injury occurs due to contusion and compression with resultant ischaemia at the time of a fracture or dislocation of the spine. Incomplete lesions include the central and anterior cord syndromes, Brown-Sequard syndrome and single nerve-root lesions.

Most cord injured patients require treatment in a specialist centre in an ITU setting for the first weeks to overcome the life-threatening medical conditions associated with cord injury. Surgical treatment is required to reduce dislocations and decompression may be indicated for incomplete lesions with persistent compression, which may lead to an improvement in level. Spinal fusion may be utilised to speed rehabilitation and prevent progressive deformity at the fracture level.

Rehabilitation is dependent on the level of cord injury and requires input from a multi-disciplinary team including physicians, surgeons, physiotherapists and occupational therapists.

Chapter 8
Paediatric orthopaedics

Bone basics

Normal bone may be either cortical or cancellous, both of which have a highly ordered macrostructure leading to the ability to withstand stresses. Mature bone is stress orientated, allowing it to resist forces in some directions but being weaker to other force directions. Immature bone is woven and has a less organised structure, leading to it being weaker and more flexible. Bone is made up of a matrix containing organic components (type I collagen, proteins, proteoglycans and growth factors) that provide some strength but also promotes bone mineralisation and cell activity; inorganic components (calcium hydroxyapatite and calcium phosphate) which provide compressive strength; and bone cells (osteoBlasts - BUILD new bone; osteoClasts - CHEW up/digest bone; osteocytes - maintain bone).

Bone growth

Long bones undergo longitudinal growth at the ends via the growth plate (physis). This is via a process called enchondral bone formation in which cells form cartilage that becomes mineralised. This is then replaced by bone when osteoclasts reabsorb it, closely followed by osteoblasts laying down new bone. Bone that has grown at the physis matures at the metaphysis and as the epiphysis grows further away, it will become the shaft of the long bone (diaphysis). The joint is formed by growth and development of special cartilage at the epiphysis.

Flat bones are formed via a process called intramembranous bone formation in which mesenchymal cells aggregate and differentiate into osteoblasts and deposit an organic matrix that mineralises into bone. There is no cartilage precursor in intramembranous bone formation.

Lower limb development

From birth through to full skeletal maturity, a child's lower limbs will change in alignment as they grow. When born, until up to 18 months of age, children will have genu varum (bowed legs). This progresses to genu valgum by 3 years of age and gradually settles to a physiological mild valgus by age 5 years. If the genu varum does not resolve by 2 years of age, the problem is unilateral or there is severe angulation, then a pathological cause must be considered, such as Blount's disease (growth disorder of the proximal medial tibia resulting in genu varum and internal tibial torsion).

As children start walking, parents may notice an in-toeing gait. The most common cause is internal rotation of the tibia (internal tibial torsion) due to intrauterine moulding that virtually always resolves spontaneously with growth. Similarly, children are born with their proximal femora naturally internally rotated due to their intrauterine position. This can also lead to an in-toed gait and sitting in a W position with legs behind them. This usually corrects spontaneously by adolescence and rarely requires treatment.

Many young children, when first starting to walk, will walk on tip-toes or with flat feet. Habitual toe walking may be due to contracture of the Achilles tendon and usually responds to stretching but occasionally requires serial casting or even Achilles lengthening. Flat foot deformity is seen when standing and is often associated with ligamentous laxity. Medial arch supports have not been shown to result in any correction in deformity and the majority of flat feet do not cause any disability.

During growth, stress from the patellar tendon upon the tibial tubercle can lead to fatigue failure, leading to pain and tenderness with localised swelling over the tubercle. This is termed Osgood-Schlatter's disease and is a self-limiting phenomenon only requiring activity modification when symptoms are present, usually during teenage growth spurts. A similar phenomenon is seen in the Achilles tendon leading to pain in the posterior heel (calcaneum) around the adolescent growth spurt and is known as Sever's disease.

Disorders of bone growth

Bone dysplasias (dwarfism)

These affect specific areas of the growing bone and have varying affects on different bones within the body. Involved bones are typically shortened - if all bones are affected equally, this is proportionate dwarfism. If there is greater involvement of the spine, resulting in a short trunk, or the limbs, resulting in short limbs, this is know as disproportionate dwarfism. Achondroplasia is an autosomal dominant condition and the most common form of dwarfism resulting in a disproportionate child with short limbs. Other examples of disproportionate dwarfs include mulitple ephiphyseal dysplasia, diastrophic dysplasia (both short limb) and spondyloepiphyseal dysplasia (short trunk). Examples of

proportionate dwarfisms include the mucopolysaccharidoses (Morquio's, Hurler's, Hunter's and Sanfilippo's syndromes). By the way, this is a very specialized area of study and knowledge of these syndromes is not expected at undergraduate level!

Osteogenesis Imperfecta (brittle bone disease)

A defect in volume and quality of type I collagen, leading to bone fragility, short stature, tooth defects, ligamentous laxity and hearing defects.

Treatment is management of acute fractures and prevention of deformity and further fractures. Bracing may be required to prevent deformity although this is commonly not successful for scoliosis. Fractures are often managed non-operatively or with minimal internal fixation and will heal well.

Osteopetrosis (marble bone disease)

Decreased osteoclast function leads to bony sclerosis and thickening with loss of the bone medullary canal. The bone is incredibly hard but can be prone to fracture because it is abnormally brittle.

Rickets

A decrease in calcium and occasionally phosphorous which affects the mineralisation occurring at the epiphyses of long bones. There are many different causes of rickets including dietary insufficiency, gastrointestinal malabsorption, kidney disease and genetic abnormalities of enzymes. Rickets leads to weak, brittle bones, bowing of the long bones, flattening of the skull, kyphosis and enlargement of the costal cartilages (rickety rosary). Radiographic examination reveals widening and cupping of the physes of long bones with marked bowing, particularly of the femur and tibia.

Treatment is according to the underlying cause, often requiring administration of calcium, phosphate and/or Vitamin D or one of its more active metabolites.

Cerebral palsy (CP)

CP is a non-progressive disorder, resulting from injury to the immature brain, leading to an upper motor neurone disorder of the neuromuscular system with age of onset less than 2 years of age. It may be caused by intrauterine, perinatal and postnatal problems, including infection, prematurity, anoxia and meningitis. It results in muscle weakness and spasticity which initially cause tightness around the joints, progressing to fixed deformity and contractures. This can eventually lead to bone deformity and joint subluxation or dislocation (typically of the hip). CP may involve different parts of the body, including scoliosis of the spine, and results in hemiplegia, diplegia or total body involvement.

Management involves a multi-disciplinary team involving paediatricians, teachers, physiotherapists, orthotists and surgeons. Treatment may be required to improve function by preventing development of contractures and deformity using splints, stretching

exercises and injections (often using selective Botulinum toxin to paralyse spastic muscles).

Abnormalities of gait may be documented using special gait analysis labs and patients may require tendon lengthening and transfers to improve their gait.

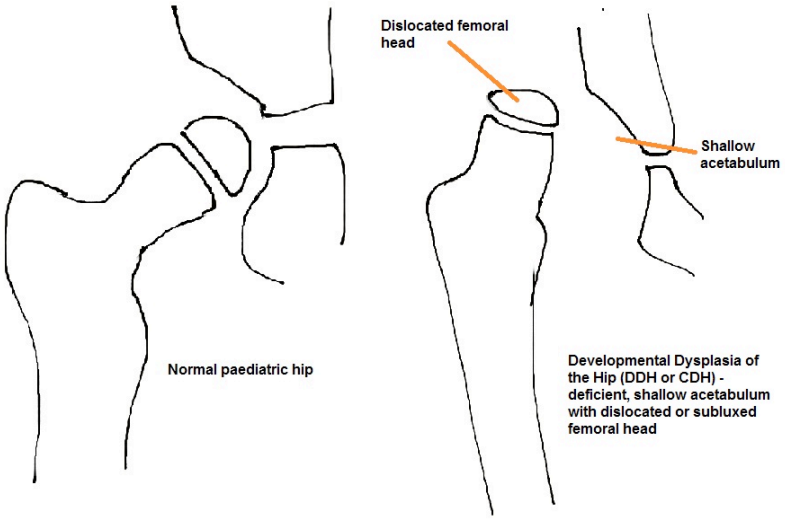
Scoliosis is common and may require operative correction and spinal fusion if bracing and custom moulded wheel-chair seats fail.

Hip subluxation and dislocation may initially be treated with tendon releases (often the adductor and psoas tendons are the major deforming forces and require lengthening or release) but may require femoral or acetabular osteotomies to improve stability.

Paediatric hip disorders

Developmental dysplasia of the hip (DDH)

DH is caused by abnormal development of the acetabulum and proximal femur leading to subluxation or dislocation of the hip. It is usually noted at birth during the new born check with the Ortolani and Barlow tests that will reveal a clicking, dislocated or unstable hip. The incidence is 1 in 1000. There should be a high index of suspicion in patients with risk factors such as family history, breech position, female sex and oligohydramnios, and should be followed up with specific hip examination and ultrasound scanning must be performed. Clinical examination may show positive Galeazzi test (affected thigh is shorter when both legs are held together with knees flexed) and decreased abduction on the dislocated side. Asymmetrical skin folds are a non-specific sign.



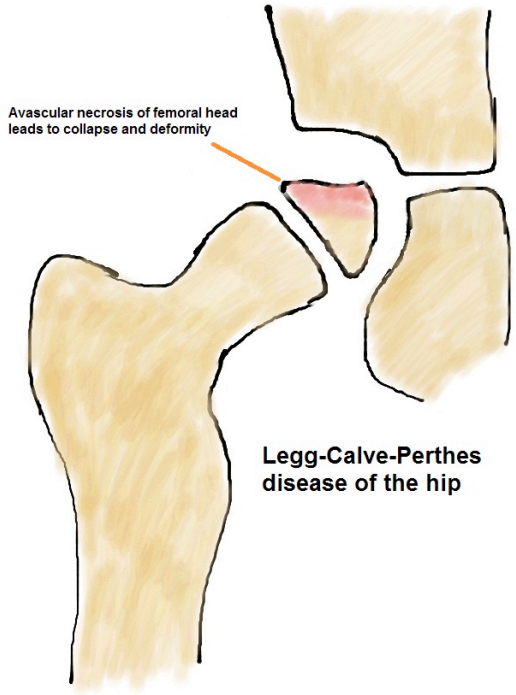
Early diagnosis and treatment of the hip leads to normal development and reduces the risk of early degeneration in the joint. A reducible hip in an infant less than 6 months old can be

placed into a Pavlik harness for up to 12 weeks, holding the hip in a safe, reduced position and allowing it to mould to a more normal shape.

After 6 months of age, or if Pavlik treatment fails, patients will undergo gentle closed reduction and adductor/psoas tenotomies under general anaesthetic and application of a hip spica cast for 12 weeks. If that fails, at close to walking age, an open reduction with hip capsule tightening and femoral osteotomies with subsequent application of a hip spica cast for 12 weeks. Some patients with continued instability or particularly malformed hip joints may require further surgery later in life with proximal femoral osteotomies or pelvic osteotomies to achieve and maintain a stable reduction and allow for more normal hip development. Complications of DDH include avascular necrosis of the femoral head (often related to treatment), leg length discrepancy and early development of hip osteoarthritis.

Legg-Calve-Perthes' Disease (Perthes)

Perthes' disease is due to disruption of the blood supply to the proximal femoral epiphysis leading to osteonecrosis followed by revascularisation and eventual remodelling. With osteonecrosis affecting the bone underlying the joint surface, there may be collapse, leading to the femoral head becoming malshaped and flattened. It is commonly seen in boys between 4-8 years of age with increased incidence with a positive family history. The incidence is 1 in 10,000. Patients will present with pain (sometimes knee pain) and a limp.

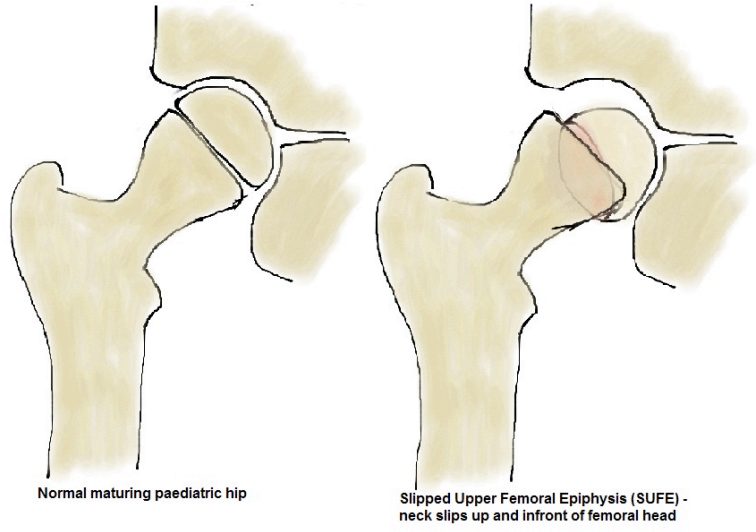


X-rays are diagnostic and prognosis is dependent on the amount of the femoral head involved and the age of the child (the older the child at presentation, the poorer the

prognosis due to less time available for remodeling during growth). Management is aimed at controlling pain, protecting range of motion and maintaining the sphericity of the femoral head and rarely requires operation (such as proximal femoral or pelvic osteotomy). The long-term risk of Perthes' disease is the development of early hip osteoarthritis.

Slipped Upper Femoral Epiphysis (SUFE)

This is due to weakness in the proximal femoral physis leading to the femoral neck displacing anteriorly and rotating externally relative to the proximal femoral epiphysis. The incidence is 1 in 100,000. It is often seen in overweight adolescent boys aged 10-16 years old and is more common in Afro-Caribbean and Pacific Islander populations. Up to a quarter have both hips affected and slips in younger patients may be associated with hormonal abnormalities (such as hypothyroidism). Examination reveals an antalgic (painful) gait, externally rotated leg and groin, thigh or knee pain.



Normal maturing paediatric hip

Slipped Upper Femoral Epiphysis (SUFE) - neck slips up and infront of femoral head

Slips may be acute or chronic and diagnosis is made on X-ray. Most slips require operative treatment with screws used to fix the epiphysis to the proximal femur and often the contralateral hip will be pinned prophylactically to prevent a slip. Complications include loss of the joint cartilage, avascular necrosis of the femoral head and early arthritis.

Transient synovitis of the hip

Any child presenting with a painful limp must have septic arthritis, DDH, Perthes' disease and SUFE ruled out with X-rays and blood tests (including inflammatory markers to rule out infection). Once these severe hip problems are excluded, the most likely diagnosis and most common cause of painful hip in childhood is transient synovitis. This is often related to a prior viral illness and the child presents non-weight bearing with limited painful hip motion. This is a self-limiting disorder and usually resolves over 24-48 hours, with anti-inflammatories used to ameliorate symptoms.

Osteomyelitis

Deep bone infection occurs most commonly in children in the metaphyses of long bones due to the extensive blood supply to these regions. Commonly caused by *Staphylococcus aureus*, haematogenous seeding of the bone occurs due to slow blood flow at the level of the physis and metaphysis, leading to the formation of an abscess in the bone.

Clinically the child will be tender, warm and swollen in the region of the infection, usually with fever. X-rays and MRI are helpful in evaluating the area of bone affected. Blood tests will show elevated C-reactive protein (CRP), white blood cells and erythrocyte sedimentation rate (ESR) and blood cultures and image guided aspiration should be taken to confirm the diagnosis.

Treatment of an abscess is surgical drainage and debridement with administration of appropriate antibiotics. If detected early and if an abscess has not developed, antibiotics may be the only treatment required. Antibiotics are usually continued for 4-6 weeks, until the bone is quiescent, inflammatory markers have returned to normal and the child is clinically well.

Congenital talipes equinovarus (clubfoot)

This is a foot deformity more common in males that is due to shortened muscles and tendons (including the calf complex/Achilles tendon, tibialis posterior, flexor hallucis and flexor digitorum longus), joint capsules and ligaments around the foot resulting in forefoot adduction and supination with hindfoot equinus and varus. The incidence is 1 in 1000. Noticed at birth, the most common treatment is via the Ponseti method which usually involves 6 weekly serial casts to improve the foot position and stretch the deforming structures. Often an Achilles tenotomy is also required. Following the casting, the patient has external rotation boots and bars applied for 2-3 years. Now that the Ponseti method is widely in use, surgical intervention is rare and is reserved for resistant, late presenting and stiff, syndromic clubfoot.

Paediatric trauma

Any child being treated for an injury must be assessed for non-accidental injury. Fractures in children under 3 years of age, multiple injuries of varied ages, burns and a history that does not explain the injury should raise suspicion and warrant further investigation by a combined team of paediatric specialists and social workers.

Paediatric fractures

These frequently occur through the growth plate (physis). They are described by the Salter-Harris (S-H) classification:

Type 1: fracture directly through physis (slip). Excellent prognosis following reduction under anaesthesia.

Type 2: fracture traversing through the physis that exits through the metaphysis. Excellent prognosis following reduction under anaesthesia. Sometimes requires temporary fixation with percutaneous wires.

Type 3: fracture traversing through the physis that exits through the epiphysis. Requires anatomic reduction and fixation because joint line is disrupted. Guarded prognosis due to risk of partial growth arrest and angular deformity.

Type 4: fracture traversing the epiphysis and physis, exiting through the metaphysis. Requires anatomic reduction and fixation because joint line is disrupted. Guarded prognosis due to risk of partial growth arrest and angular deformity.

Type 5: crush injury to physis. Rare. Poor prognosis due to high risk of growth arrest.

Bone remodels during growth and after injury according to the mechanical stresses applied to it (Wolff's Law). This means that increased mechanical stress will lead to an increase in bone density and diameter. If a fracture's deformity is close to a joint and in the plane of motion of that joint, it has a greater ability to remodel back to normal.

Common paediatric fractures

Supracondylar elbow fracture

Commonly seen after a fall onto an extended arm. Can result in a displaced fracture that requires manipulation under anaesthetic and pinning to hold the position. Pins are removed after 3-4 weeks. These fractures have a small risk of median nerve (especially the anterior interosseous branch) and brachial artery injury, both of which must be closely assessed. Ulnar nerve injury may occur with medial pin insertion during surgery and care must be taken to protect the nerve.

Both bone forearm fracture

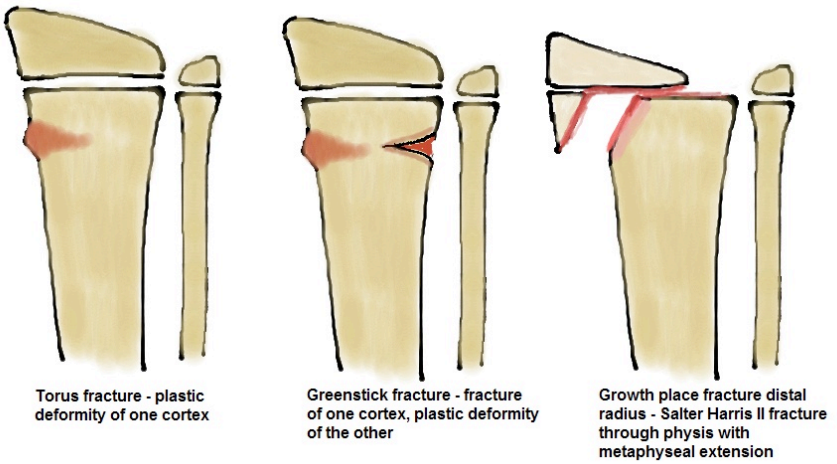
Seen after a fall with a direct blow to the forearm. Rotation and angulation must be corrected if present. Often stable, these fractures can usually be safely managed with manipulation and above elbow cast for 3-4 weeks. Unstable fractures may be treated with flexible intramedullary rods inserted at operation and removed when the fracture has healed.

Distal radius fracture

Four different types of fracture (applicable to all paediatric long bones), depending on the age of the patient and severity of injury:

1. Torus fracture - a buckle of one cortex due to compressive force. Treat in cast for 3 weeks.
2. Greenstick fracture - one cortex is fractured and one suffers plastic deformation. May require reduction if angulated. Treat in cast for 3-4 weeks.
3. Complete fracture - both cortices are fractured - potentially unstable. May require reduction if angulated or displaced. Treat in cast for 3-4 weeks.

4. Physeal fracture - Most common paediatric fracture is Salter Harris type II. Requires reduction if displaced. If S-H type III or IV, require reduction and pinning.



Torus fracture - plastic deformity of one cortex

Greenstick fracture - fracture of one cortex, plastic deformity of the other

Growth place fracture distal radius - Salter Harris II fracture through physis with metaphyseal extension

Femoral shaft fracture

Treated according to the age of the patient. Under 6 years old, a child can be placed into a hip spica cast for 4-6 weeks. This fracture may heal with the injured side 1-2 cm longer than the contralateral side due to fracture overgrowth. Over 6 years of age, most patients undergo operative fixation with flexible intramedullary nails or plates. It is important to ensure that any fracture rotational deformity is corrected. Patients over 14 years of age nd close to skeletal matutity may be treated with a rigid femoral intramedullary nail in the same manner as an adult.

Distal femoral and proximal tibial epiphyseal fracture

These are commonly S-H II injuries but any displaced fractures, especially those extending into the epiphysis, require perfect anatomic reduction and fixation with either wires or screws. Early motion is preferable.

Tibial spine avulsion

The equivalent of an ACL rupture in an adult, the stronger ligament pulls of its bony attachment. If displaced, requires arthroscopic or open reduction and fixation.

Tibial shaft fracture

If undisplaced, may be managed in an above-knee cast for 6-8 weeks until clinical and radiological union. If displaced, requires manipulation and if unstable can be fixed with flexible intramedullary nails.

Distal tibial fractures

Triplane fracture is a complex SH IV injury with fracture lines in all 3 planes. Requires investigation with CT scan to aid planning for open reduction and fixation if there is articular displacement.

Tillaux fracture is an isolated SH III fracture of the lateral tibial physis as the medial physis has closed. If undisplaced, can be treated in an above-knee cast but if displaced, requires open reduction and fixation.

Chapter 9
Pathology

Benign or malignant orthopaedic tumours may involve the bones or soft tissues of the skeleton. Primary bone tumours are relatively uncommon but bony metastases are and will often be referred to orthopaedic surgeons for stabilisation. Pain (especially night pain), swelling and a change in size of a mass are worrying features that require further investigation.

Benign bone lesions on X-ray have well demarcated margins with no associated soft tissue mass or periosteal reaction. These include simple bone cysts, enchondromas, osteochondromas (exostoses) and aneurysmal bone cysts. Malignant lesions have an aggressive appearance on X-ray with bone destruction, poorly demarcated margins and periosteal reaction, often with an associated soft tissue mass. These include osteosarcoma and Ewing's sarcoma.

Soft tissue tumours develop from the connective tissues other than bone, such as fat, muscle, tendon, nerve and blood vessels. Benign soft tissue lesions are usually present for many years in a superficial position and with no or minimal pain and no change in size. These include lipomas, haemangiomas and neurofibromas. Malignant soft tissue tumours are usually in a deep position, painful and increase in size rapidly. These include liposarcoma, fibrosarcoma, leiomyosarcoma and synovial sarcoma.

After taking a full history and performing a thorough examination, investigations include:

Blood tests (FBC, bone profile, U&E, protein electrophoresis, ESR, CRP)
X-rays
Further imaging (usually MRI)
Image guided biopsy

If the biopsy of a lesion indicates malignancy, the patient requires staging, including investigation for metastases with a CT chest (most common site is to the lung) and bone scan. Surgical excision is performed with concurrent chemotherapy and radiotherapy as guided by oncologists.

Simple bone cyst

Found in the growing skeleton, often in the proximal humerus and femur. They are usually asymptomatic until a fracture occurs through the weakened cortical bone surrounding the

cyst. A fracture usually stimulates healing of the cyst and apart from splinting in the form of casts or braces, simple cysts rarely require further intervention.

Osteochondroma (exostosis)

A benign bone tumour which is often solitary but may be inherited with patients having many lesions in hereditary multiple exostoses. Pedunculated lesions arise from the joint line and grow away from the joint. May be left alone unless they cause local irritation or become symptomatic (pain may indicate malignant transformation which occurs in about 1%) in which case, they may be excised.

Osteosarcoma

Commonly seen in childhood and adolescence with a second peak in 6th decade. Most often affects distal and proximal femur, proximal tibia and proximal humerus. Histology reveals a high-grade spindle-cell tumour. Treatment is neo-adjuvant chemotherapy (pre- and post-surgery) with tumour excision and reconstruction with custom designed prostheses. If this involves the joint, this will be excised and replaced with a prosthesis. Following treatment survival depends on whether the tumour had metastasised at presentation: 10 year survival 90% with no metastases but only 30% with metastases.

Ewing's Sarcoma

Occurs in children and adolescents, most often in the femoral shaft. Patients receive neo-adjuvant chemotherapy followed by surgical excision and reconstruction. Survival rate at 5 years is 60-70% with no metastases, dropping to 20% when metastases are present.

Metastatic disease

Malignancies that have a predilection for spread to bone are lung, breast, prostate, kidney and thyroid. A primary bone tumour must be excluded in all cases. Treatment revolves around avoiding a pathological fracture. Risk is calculated using Mirel's scoring system (increased risk with metastasis in the trochanteric region of hip, severe pain, lytic lesion on X-ray and involvement of >1/3 of bone cortex). Patients at high risk may undergo prophylactic fixation of bones to prevent fracture (most commonly the femur will be stabilised with an intramedullary nail).

Soft tissue tumours

Require investigation if any part of the history or examination point towards malignancy (rapid increase in size, pain, deep lying tumour) with MRI and biopsy. If benign, marginal excision is adequate. Malignant soft tissue tumours require a wide margin of excision, following by radiotherapy to the tumour bed to reduce the recurrence rate.

Chapter 10
Examinations

Orthopaedic examination of any bone or joint follow the same basic structure:

LOOK - gait (if lower limb), erythema, swelling, scars, deformity, muscle wasting
FEEL - warmth, tenderness, swelling, crepitus
MOVE - range of motion, power
SPECIAL TESTS - joint stability, isolated muscle power, provocative tests
JOINT ABOVE & BELOW - ensure pain not radiating from elsewhere
NEUROVASCULAR - examine neurological and vascular supply to limb

This is followed by INVESTIGATIONS - blood tests, X-rays, further imaging

This chapter will describe a basic technique to examine the joints regularly seen in the exams and clinics. By following the routine described above, most joints can be examined thoroughly. The text does not provide an exhaustive description of every test available but outlines the commonly used basic examinations.

Shoulder

LOOK

Scars - deltopectoral (from coracoid towards axillar), arthroscopy (anterior, posterior and lateral)
Deformity – chronic anterior dislocation (hollow under acromium and fullness anteriorly)
Muscle wasting - deltoid (axillary nerve), trapezius (spinal accessory nerve), supraspinatus (suprascapular nerve), serratus anterior with winging of scapular (long thoracic nerve)

FEEL

Tenderness - feel full length of clavicle, acromion, glenohumeral joint and scapular spine. Isolated tenderness over acromioclavicular joint (ACJ osteoarthritis)

MOVE

Range

1. abduction 0-170°
2. flexion 0-180°
3. extension 0-70°
4. external rotation 0-80°
5. internal rotation 0-90°

Painful arc - 40-150° abduction - impingement syndrome
150-180° abduction - acromioclavicular disease

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SPECIAL

Power - rotator cuff muscles:
Supraspinatus - resisted abduction with thumb point down
Infraspinatus & teres minor - resisted external rotation
Subscapularis - resisted internal rotation

Joint stability - Apprehension test - patient lying at 45°, shoulder abducted 90° and elbow flexed 90°, external rotation of shoulder causes apprehension of anterior dislocation.

Relocation test - apprehension test repeated with hand placed firmly at front of shoulder, blocking anterior instability and preventing apprehension.

Provocative - Empty can test - resisted abduction with thumb pointing towards ground causes pain = subacromial impingement

Scarf test (cross arm adduction) - hand is adducted towards opposite shoulder causing pain located at ACJ = ACJ pathology

Hip

LOOK
Gait - Trendelenburg (weak abductors allow pelvis to droop upon stance on the affected side, with reciprocal trunk lurch over weak hip to compensate and balance the pelvis; Antalgic (short stance phase due to pain)
Scars - Anterior (longitudinal inline with ASIS or parallel to groin crease - often from surgery as a child e.g. open reduction of DDH), lateral (longitudinal over greater trochanter - hip fracture or joint replacement), posterior (curved longitudinal behind greater trochanter - acetabular/hip fracture or joint replacement)
Muscle wasting - wasted gluteal muscles.
Trendelenburg test - with a single leg stance, the opposite hip sags due to weak abductors on the side of stance

Lie patient supine on examination couch.

FEEL
Tenderness - lateral tenderness indicates trochanteric bursitis
Leg length - Ensure the pelvis is level. Measure true length (ASIS to medial malleoli on both sides). Measure apparent length (xiphisternum to medial malleoli - difference indicates other cause e.g. scoliosis)

MOVE
Thomas' test - examiners hand under lumbar spine to ensure lordosis flattens. Flex one hip fully and assess other hip - if it has risen off bed, there is a fixed flexion deformity. Repeat with contralateral hip

Stabilise pelvis with free hand to ensure motion arises from hip and not pelvic movements

Range

1. flexion 0-120°
2. extension 0-15°
3. abduction 0-45°
4. adduction 0-30°
5. flexion & internal rotation 0-35°
6. flexion & external rotation 0-45°

SPECIAL
Power - hip flexor and extensor power
Straight leg raise - ensure hip pain not originating from spinal pathology

Knee

LOOK
Gait - Antalgic (short stance phase due to pain)
Scars - midline longitudinal (total knee replacement, ACL reconstruction with patella tendon), arthroscopy portals (anteromedial and anterolateral), posterior (PCL surgery or vascular surgery)
Muscle wasting - Quadriceps wastes after acute knee injury. Mark a point 10cm above tibial tuberosity (fixed bony point), measure circumference of thigh, compare to contralateral side
Erythema - infected pre-patellar bursitis presents with gross erythema over front of knee
Swelling - large effusion within knee joint may be visible. Pre-patellar bursitis has large fluctuant swelling over front of patella
Deformity - Va**L**gus (lower leg deviates **L**aterally), Varus (lower leg deviates medially), fixed flexion

Lie patient supine on examination couch.

FEEL
Warmth - inflammation or infection will lead to a warm joint – use dorsum of hand to feel the knee
Effusion - slide hand down over supra-patella pouch, milking fluid into knee joint and hold firmly above patella, "ballot" patella into tibia with large effusion (patella tap test), stroke fluid from medial side of knee to lateral side (bulge test) for smaller effusion
Tenderness - medial or lateral joint line tenderness (easier to palpate with knee flexed to 90°). Tenderness with pressure upon patella into trochlea (patella grind test) indicates patellofemoral disease
Crepitus - noted with hand over patella with range of motion tests - indicates possible patellofemoral disease

MOVE
Range
1. Extension-flexion 0-130°

Straight leg raise - extensor apparatus (quadriceps tendon, patella, patella tendon, tibial tubercle) intact

SPECIAL
Anterior drawer - knee flexed 90°, foot planted on bed and stablised. Examiner grasps patient's tibia, fingers around calf, thumbs on tibial tubercle and tugs tibia forward - anterior displacement implies ACL laxity
Lachman's test - knee flexed 30°, examiner grasps and stabilises thigh with left hand. Right hand grasps tibia, with thumb on tibial tubercle, and tugs tibia forward - anterior displacement implies ACL laxity

Posterior sag - both knees flexed to 90°, side by side. PCL deficient knee will have a posterior sag of tibia compared to contralateral side, showing an abnormal contour along line drawn from distal tip of patella through tibial tubercle and anterior border of tibia

Posterior drawer - knee flexed 90°, foot planted on bed with examiner sitting on it. Examiner grasps patient's tibia, fingers around calf, thumbs on tibial tubercle and pushes tibia backwards - posterior displacement implies PCL laxity

Valgus stress test - grasping shin and foot under right arm and using left hand to stabilise thigh, valgus stress applied to knee - opening of joint implies medial collateral ligament laxity

Varus stress test - grasping shin and foot under right arm and using left hand to stabilise thigh, varus stress applied to knee - opening of joint implies lateral collateral ligament laxity

McMurray's test - knee flexed with one hand to 90° whilst the sole of the foot is grasped with the other hand. The foot is internally rotated whilst knee is extended - medial joint pain or a click indicates a medial meniscal tear. The foot is then externally rotated whilst knee is extended - lateral joint pain or a click indicates a lateral meniscal tear

Hip examination - hip pain may radiate to the knee - isolate the hip and ensure movement does not mimic the patient's symptoms, indicating a hip-related cause of the pain

Foot and ankle

LOOK
Shoes - always examine a patient's shoes for signs of asymmetrical wear
Gait - antalgic (short stance phase due to pain)
Scars - medial and lateral longitudinal (fixation of fractures), anterior longitudinal (fracture fixation, ankle joint replacement), posterior (Achilles tendon rupture repair)
Ulceration - throughout the ankle, foot and between toes for evidence of vascular insufficiency and diabetes
Deformity - Va**L**gus (ankle/foot deviates **L**aterally), Varus (ankle/foot deviates medially), fixed plantar flexion
Hallux valgus and inflamed bunion
High arch of foot or flat foot
Clawing of toes

FEEL
Tenderness - feel for tenderness along lateral malleolus (fibular fracture), medial malleolus (fracture, deltoid ligament injury), anterior joint line (anterior talofibular ligament (ATFL) injury, ankle osteoarthritis), Achilles tendon (Achilles tendonitis)
Palpate midfoot for tenderness
Squeeze metatarsophalangeal joints looking for pain
Temperature - feel for warmth over ankle, mid foot and toes

MOVE
Foot and ankle have complex linked ranges of motion
Range

1. ankle plantar flexion 0-50°
2. ankle dorsiflexion 0-20°
3. ankle inversion 0-35°
4. ankle eversion 0-15°

SPECIAL
Ankle drawer test - grasp tibia with one hand and heel with the other. Attempt to sublux ankle joint anteriorly, identifying ATFL laxity
Syndesomosis compression test - squeeze distal fibular at level of syndesmosis onto tibia. Pain implies syndesmosis injury

Spine

LOOK
Deformity - scoliosis, kyphosis, lordosis, pelvic obliquity
Scars - longitudinal midline scar (discectomy, spinal fusion, scoliosis surgery - if longer scar)

FEEL
Tenderness - midline tenderness along spine. Tenderness in para-spinal muscles along whole length of spine. Sacroiliac joint tenderness
Temperature - feel for warmth over length of spine indicating infection
Abdominal examination - ensure no evidence of abdominal aortic aneurysm, pancreatitis. Perform digital rectal examination if necessary

MOVE
Range

1. flexion - bend and touch toes - hands to mid-shin is normal
2. extension - combined thoracic and lumbar is about 45° lateral flexion - slide hands down sides of thighs - should be equal
3. rotation - sit on couch, cross arms, then rotate - normal is about 40°

Hip - hip pathology may present as back pain - perform isolated hip motion to ensure this is not the source of symptoms.

Lie patient supine on examination couch.

SPECIAL
Straight leg raise.sciatic stretch test - flex hip with knee extended and foot dorsiflexed. Onset of back or leg pain is positive. Paraesthesia or pain in root distribution indicates nerve root irritation. Back pain indicates central disc prolapse. Lower leg slightly and dorsiflex foot more - aggravated pain is a positive sciatic stretch test.

Bowstring test - Once straight leg raise has caused pain, lower the leg slightly and flex the knee. Apply firm pressure in the popliteal fossa over the stretched tibial nerve. Resumption of pain and paraesthesia suggests nerve root irritation

Femoral stretch test - lie the patient prone. Flex each knee. If pain occurs in anterior thigh and is worsened by hip extension, this is a positive femoral stretch test (irritation of L2-4 nerve roots)

Neurological examination - thorough examination of tone, power by myotomes, sensation by dermatomes and reflexes is vital.

Myotomes:

- L2: hip flexion
- L3: knee extension
- L4: ankle dorsiflexion
- L5: big toe extension
- S1: ankle plantar flexion

Dermatomes:

- L2: anterior thigh
- L3: anterior knee
- L4: medial aspect of the lower leg
- L5: lateral aspect of the lower leg, medial side dorsum of the foot
- S1: lateral aspect of the foot, the heel and most of the sole
- S2: posterior aspect of the knee
- S3-S5: concentric rings around the anus, the outermost of which is S3

Reflexes:

- L3,4: patellar
- S1,2: ankle jerk