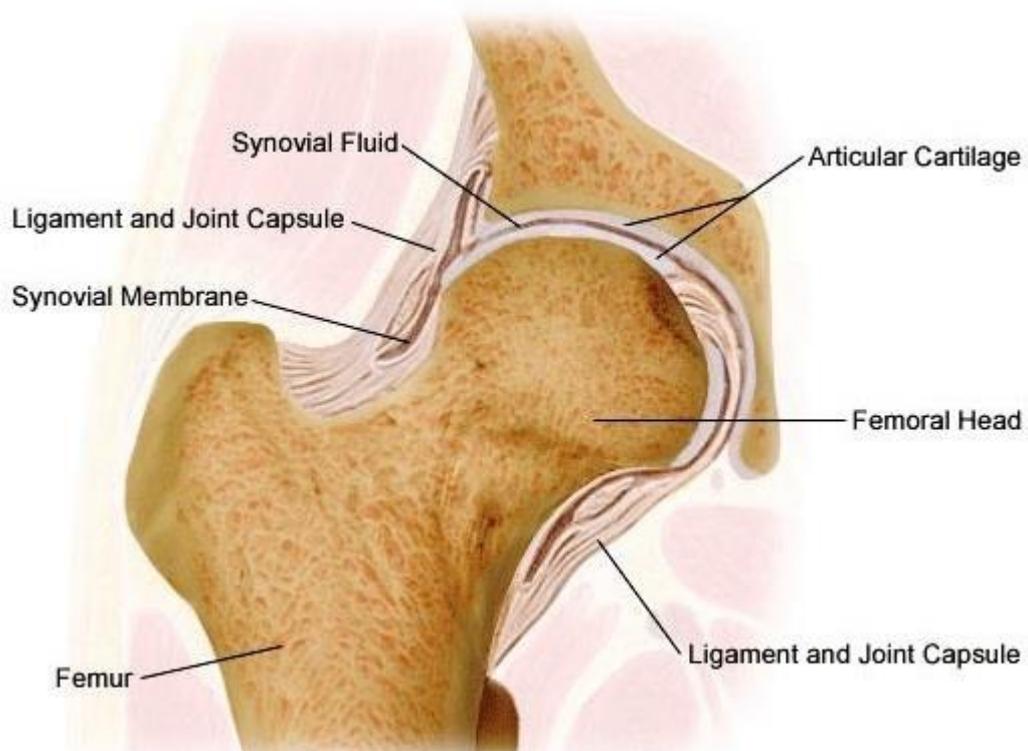


The Hip

A Comprehensive Overview



Laurence Hattersley 2016
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The Hip

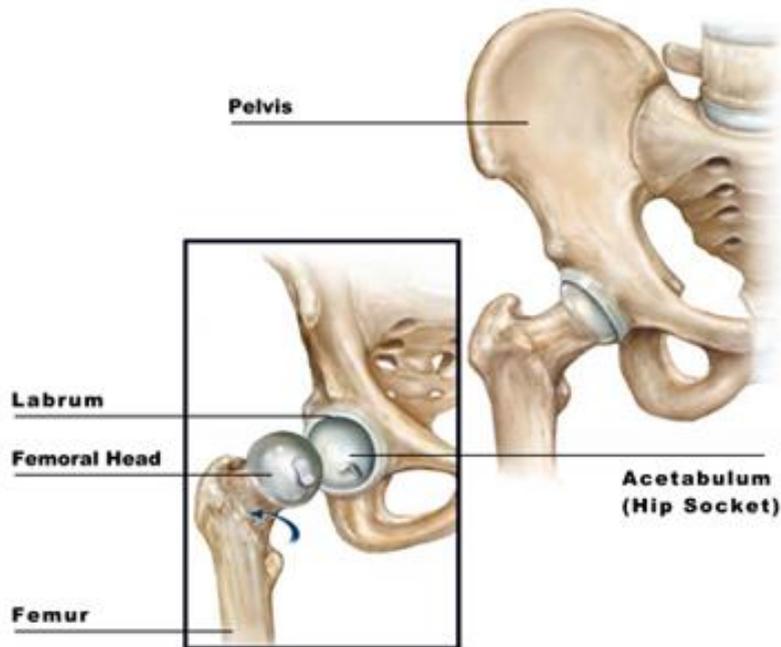


Figure 1 The Hip Joint

The terms 'hip' and 'pelvis' are frequently used interchangeably, but strictly speaking the pelvis is a ring of bones, along with their attachments, and the hip is a joint. The bones of the pelvis (in the hip joint) and the upper end of the femur grow together and develop with each other, with both of them being subjected to increasing forces (e.g. sitting, standing, walking etc). These forces are unavoidable but desirable for sculpting normal bony tissues. In this respect, many things can go wrong in the journey from birth to adulthood and even then, there is old age.

Bony Apparatus

The hip, or **Coxofemoral joint**, is formed from a number of bones: the ilium, the ischium and the pubis. These form a bony apparatus (the acetabulum), which collectively hold the head of the femur.

In children the acetabulum is shallow to the point of non-existence and so can dislocate very easily. Babies are usually checked at birth for hip stability and then again at 6 months if there is a history of congenital dislocation of the hip (CDH; see page 3) in the family.

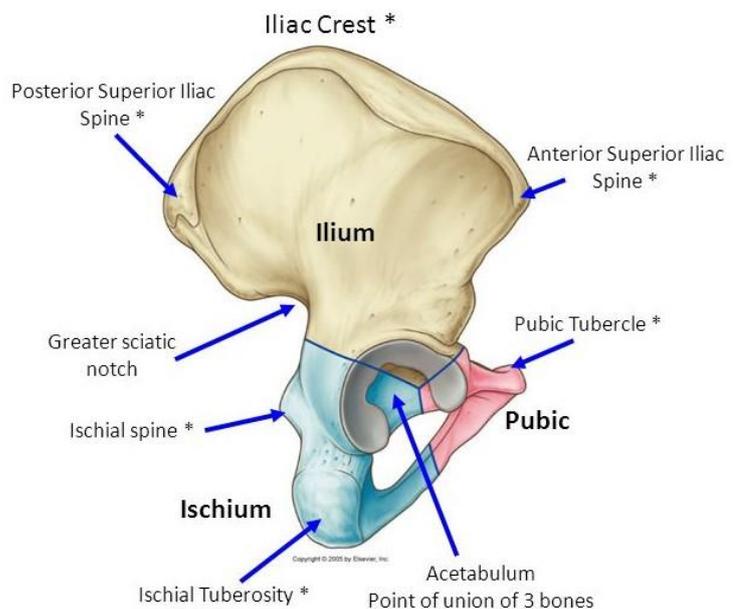


Figure 2 Bones of the pelvis seen

in the acetabulum

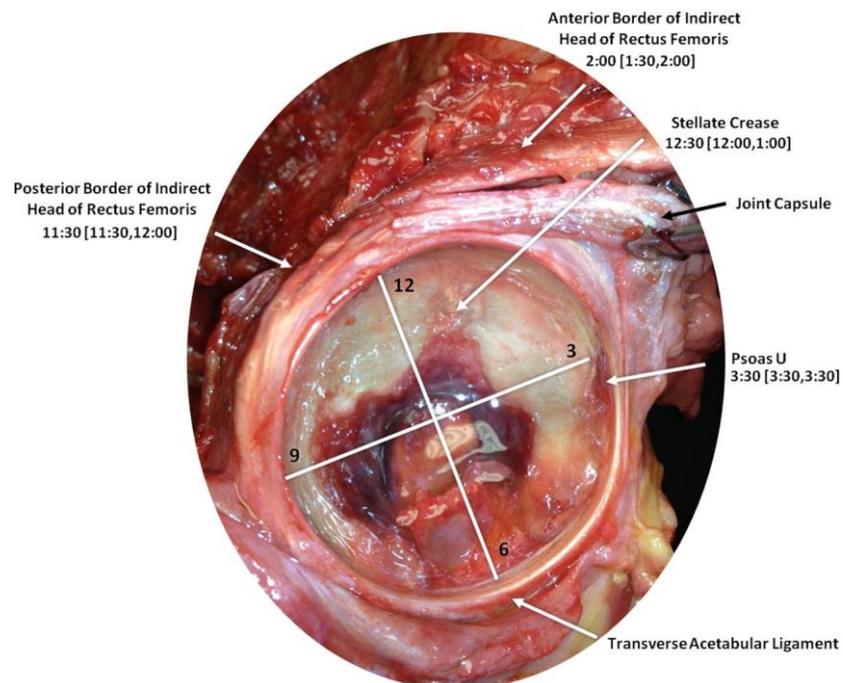


Figure 3 Acetabulum on dissection showing articular surface



Figure 4 Coxofemoral joint, showing both femoral head and acetabulum with ligamentum teres cut

Ossification in the Hip Area

At a very early age, the bones of the pelvis and femur have several parts: the **Ilium**, the **Ischium** and the **Pubis**, them forming from their centres of ossification.

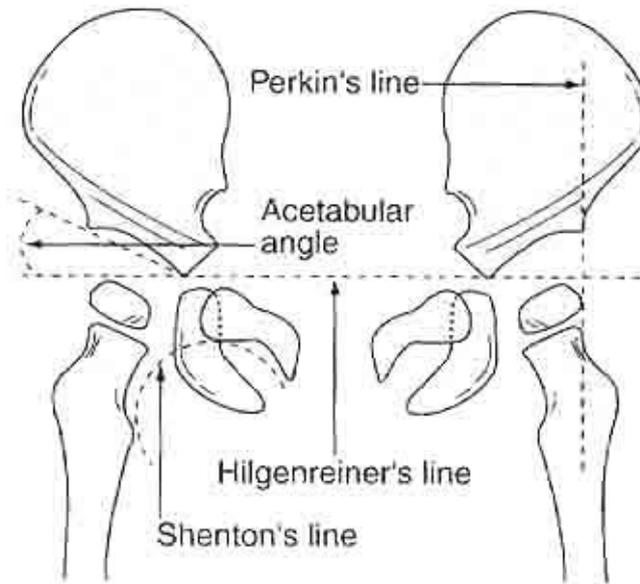


Figure 5 Schematic of pelvic and femoral epiphyses

Similarly, the head of the femur also has several centres of ossification

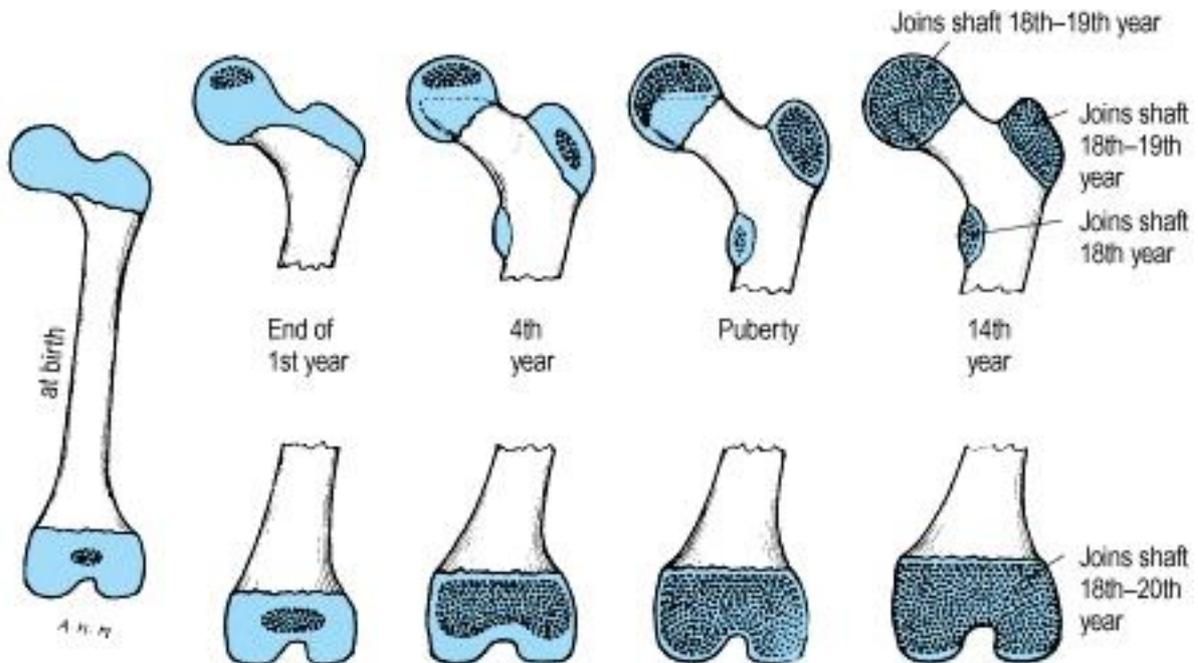


Figure 6 Centres of Ossification in Femur

These centres of ossification of the top of the femur are found in:

- The femoral head
- The greater trochanter
- The lesser trochanter

Of these, the epiphysis of the head of the femur is the biggest. It is joined to the neck of the femur by cartilage (the growth plate) and this line of cartilage can present itself as a line of *potential* weakness and the hip can slip

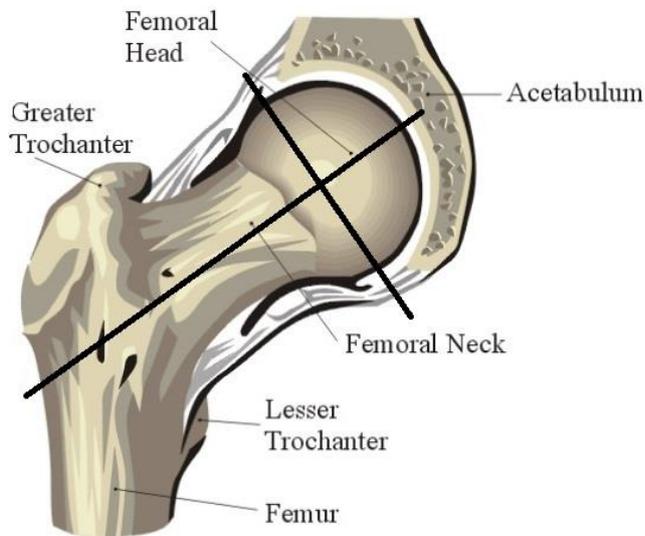


Figure 7 Head of Femur in Acetabulum

The diagram on the left shows the head of the femur in the acetabulum. Note:

- The femoral head is about 2/3 of a sphere
- The neck of the femur is about 240° to the shaft
- The femoral neck is presented 180° to the acetabulum

In a normal pelvis, there is a labrum; a lip consisting of a horseshoe shaped lip of cartilage around the upper edge of the acetabulum. The function of the labrum is to increase the depth of the acetabulum and articular surface.

The labrum and atmospheric pressure contribute to the stability of the joint; if the joint was stripped of all of the surrounding tissues (muscles, ligaments and capsule), it would still require 22Kg of traction to dislocate the joint. This is rare but it can happen.

Femoral Head and Neck

The bony structure of the head of the femur is designed to bear weight and distribute it and the trabeculae of the femoral neck and head are aligned to facilitate this

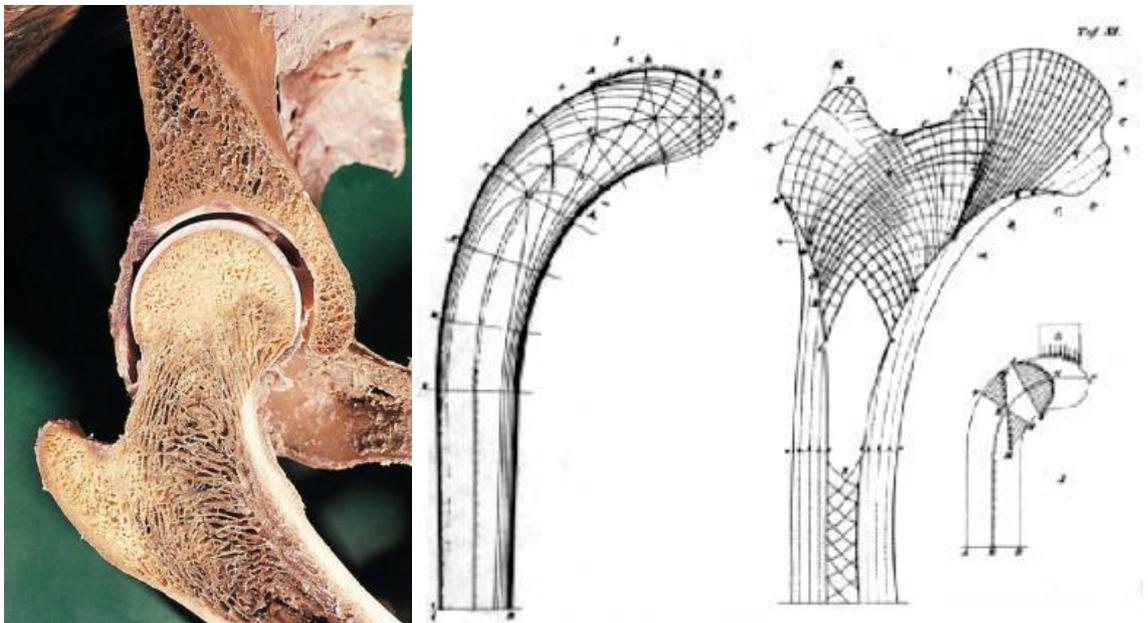


Figure 8 Section of femoral head showing trabeculae

The femoral neck, normally, forms an angle of **120-135 degrees** with the shaft of the thigh bone. This acts as a lever in easing the action of the muscles around the hip joint. An increase or decrease in this angle beyond the normal limits causes improper action of the muscles and interferes with walking. An increase in the angle beyond 135 degrees is called as **coxa valga** or outward curvature of the hip joint. A decrease in the angle below 120 degrees is called as **coxa vara** or inward curvature of the hip joint.

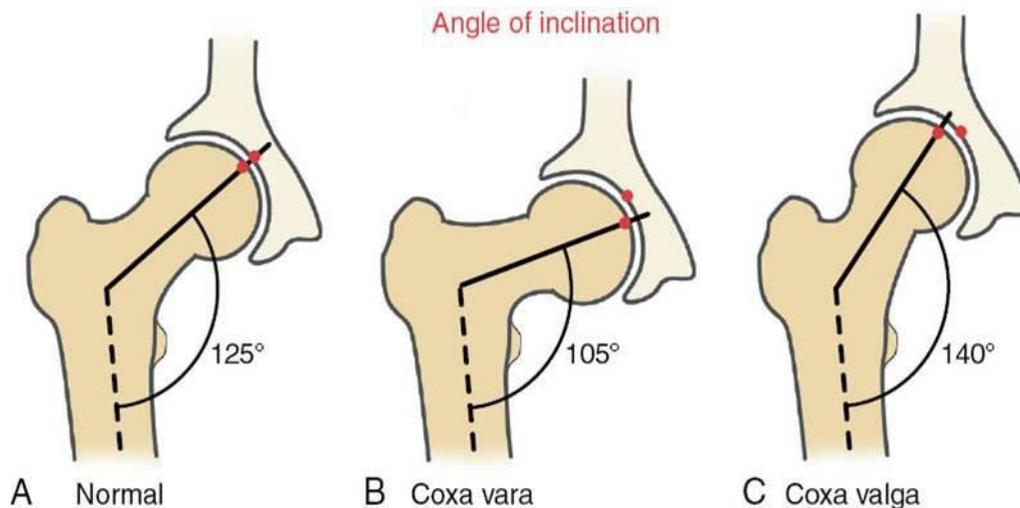


Figure 9 Angle of Femoral Neck indicating Coxa Vara and Valgus

The angle of the femoral neck is important in terms of centre of gravity and lines of force through the hip and leg to the foot.

Figure 10 Centre of gravity and line of force through hip to foot

Due to the low incidence of coxa vara which is even lower for coxa valga little recent literature is currently available. Therefore, this page will predominantly describe the complete pathology of coxa vara.

Coxa vara is as a varus deformity of the femoral neck. It may be defined when the angle between the neck and shaft of the femur is less than 110 – 120 ° (which is normally between 135 ° - 145 °) in children.

Coxa vara is classified into several subtypes:

- **Congenital coxa vara** is present at birth and is caused by an embryonic limb bud abnormality.
- **Developmental coxa vara** occurs as an isolated deformity of the proximal femur. It tends to go unnoticed until walking age is reached, when the deformity results in a leg length difference or abnormal gait pattern.
- **Acquired coxa vara** is caused by an underlying condition such as fibrous dysplasia, rickets, or traumatic proximal femoral epiphyseal plate closure.



Clinically relevant anatomy

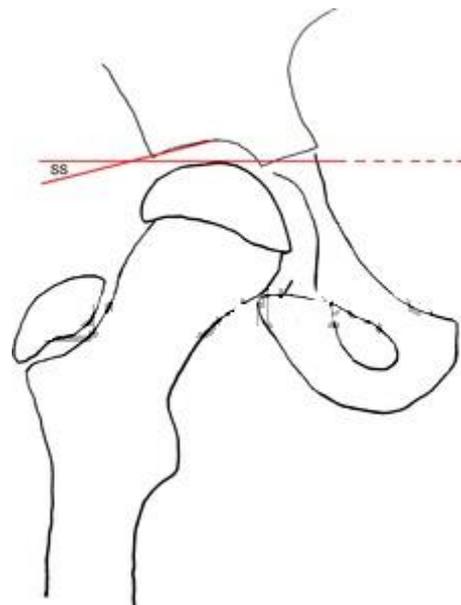
Congenital coxa vara results in a decrease in metaphyseal bone as a result of abnormal maturation and ossification of proximal femoral chondrocyte. As a result of *congenital coxa vara*, the inferior medial area of the femoral neck may be fragmented. A progressive varus deformity might also occur in *congenital coxa vara* as well as excessive growth of the trochanter and shortening of the femoral neck.

A review on the development of coxa vara shows an association with spondylometaphyseal dysplasia, demonstrating that stimulated corner fractures were present in most instances.

A case study shows also that a varus position of the neck is believed to prevent hip subluxation associated with femoral lengthening. An associated dysplastic acetabulum can lead to a hip subluxation.

This case study shows also that the acetabulum is abnormal in coxa vara. Acetabular index (AI) and Sourcil slope (SS) are significantly different than in the normal acetabulum.

Figure 11 Sourcil line



Variations in the femoral neck angle away from 135° can result in problems, certainly locally and possibly distally. This is primarily due to the increased shearing forced experienced by the neck and head of the femur.

Both the deformities cause early hip joint arthritis, whereas,

- **Coxa valga** can also cause frequent hip dislocations.
 - Treatment is by a surgery known as **femoral neck osteotomy**, in which the bone is cut, realigned, and fixed, to bring the angle in the normal range.
- Coxa vara can be a significant indicator in cases of slipped epiphysis (SUFE - see later)

Femoral Neck Fractures

Road accidents are a major cause of fractures of the femoral neck in adults. In children, however, injury to this region causes bending of the bone, leading to a deformed femoral neck, rather than a break (fracture). A femoral neck fracture is one of the commonest fractures occurring in the elderly. This is because of the weakening of the bone due to osteoporosis (bone loss). Hence, a **trivial fall** is all that is needed for breaking the femoral neck

A fracture of femoral neck causes the femoral head to lose its blood supply or become avascular. This causes death of the femoral head tissue (necrosis) in as less as 6 hours. The **avascular necrosis** (AVN) of femur (death of the femoral head due to loss of blood supply) is an irreversible process and it has to be replaced with a prosthetic head. Hence,

any injury to the hip joint should always be taken as an emergency, and prompt medical treatment should be taken.

Epidemiology/aetiology

Femoral neck fractures (< 1 % of all paediatric fractures in children) are associated with a high incidence of complications. The most serious ones with high and long-term morbidity being osteonecrosis and coxa vara.

A retrospective study of femoral neck fractures in children show the following complications:

- Avascular necrosis (14.5%)
- Limb shortening in seven (11.3%)
- Coxa vara (8%) and premature epiphysis fusion (8%)
- Coxa valga (3.2%), arthritic changes (3.2%).
- Non-union in one (1.6%)

Premature epiphyseal closure is described as one of the aetiological factors of coxa vara. Incidences of premature epiphyseal closure reported in the literature range from 6 % to 62 %.

Another possible explanation for the high occurrence of coxa vara is the loss of reduction after initial fracture reduction of implant failure in unstable fractures.

A study shows that developmental coxa vara is a rare condition with an incidence of 1 in 25,000 live births.

Recent reports show that the incidence of coxa vara can be decreased by using internal fixation such as pins or screws.

Clinical Presentation

Clinically the condition presents itself as an abnormal but painless gait pattern. A Trendelenburg limp is sometimes associated with unilateral coxa vara and a waddling gait is often seen when bilateral coxa vara is present. Patients with coxa vara often show:

- Limb length discrepancy
- Prominent greater trochanter
- Limitation of abduction and internal rotation of the hip.

Differential Diagnosis

Radiography (AP view of the pelvis) can be utilized to determine the HEA (Hilgenreiner's Epiphyseal Angle - Fig 9). Signs to look out for are as follows:

- The neck – shaft angle is less than 110 – 120°.
- The greater trochanter may be elevated above the femoral head.
- A growth plate with an overly vertical orientation.

Hilgenreiner's Epiphyseal Angle

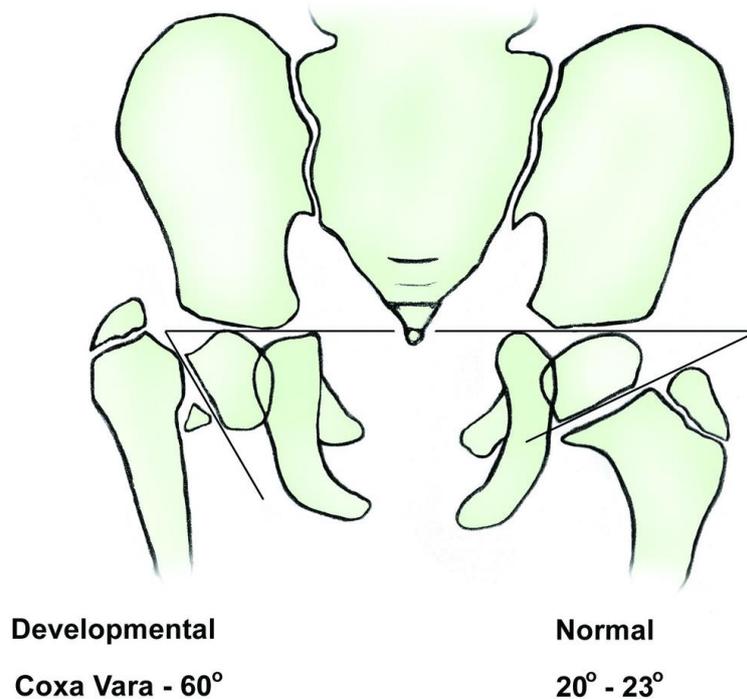


Figure 12 Hilgenreiner's Epiphyseal Angle

Medical management

According to a case study, the objective of medical interventions is to restore the neck-shaft angle and realigning the epiphyseal plate to decrease shear forces and promote ossification of the femoral neck defect.

This is achieved by performing a valgus osteotomy, with the valgus position of the femoral neck improving the action of the gluteus muscles, normalising the femoral neck angle, increasing total limb length and improving the joint congruence.

The following are indications for surgical intervention:

- Neck – shaft angle less than 90 °.
- Progressive development of deformity.
- Vertical physis and a significant limb.

Other indications are based on the HE – angle.

- HE – hoek > 60 ° is an indicative for surgery.
- HE – hoek 45 – 60 ° warrants close follow – up.
- HE – hoek < 45 ° warrants spontaneous resolution.

Except when the neck–shaft angle is less than 110°, progression of the varus angulation takes place, gait pattern abnormalities or degenerative changes take place.

The treatment of femoral neck fracture depends on the time elapsed after the injury and the age of the patient.

If this is less than 6 hours, then the fracture can be fixed with **metallic screws** passing through the femoral neck, which restores the blood supply of the femoral head (as the displaced fragments of bone are brought in place). After this treatment, the patient has to take bed-rest for 3 weeks followed by graded exercises. This surgery is highly **affordable**, and is frequently recommended for young and active individuals. Elderly individuals, with weak osteoporotic bones, are not suitable candidates for this surgery.



Figure 13 Surgical fixation of fracture of neck of femur

Hip joint growth and deformity

Normal hips will grow as per the forces going through them. If these forces are changed, there will be change in the structure of the joint, e.g. an amputated thigh or leg during the growth years leads to atrophy of the pelvis, resulting in a smaller ilium.

There are various bony aspects that can be seen for normal parameters to be defined

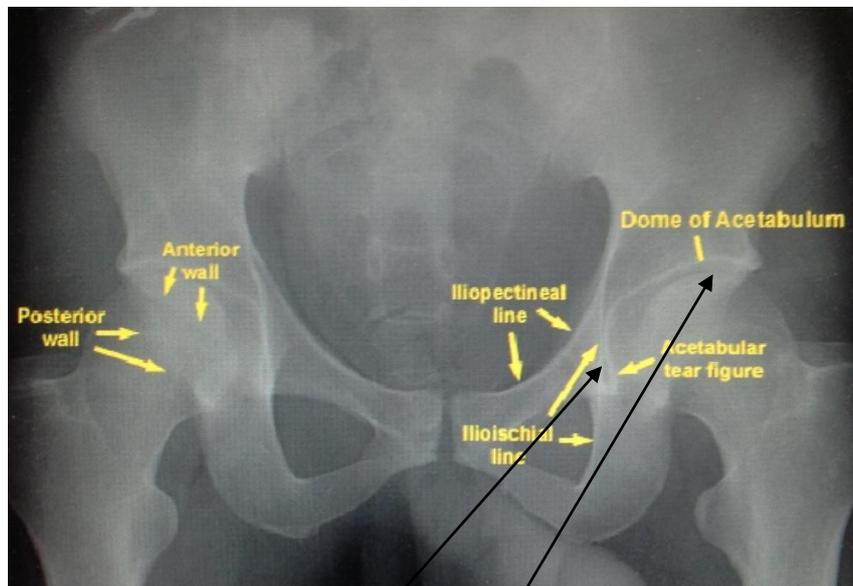


Figure 14 X-Ray Showing bony parameters of reference

From these points of reference, various lines can be drawn:

- **Radiographic teardrop**
- **Acetabular roof (dome) or Sourcil**

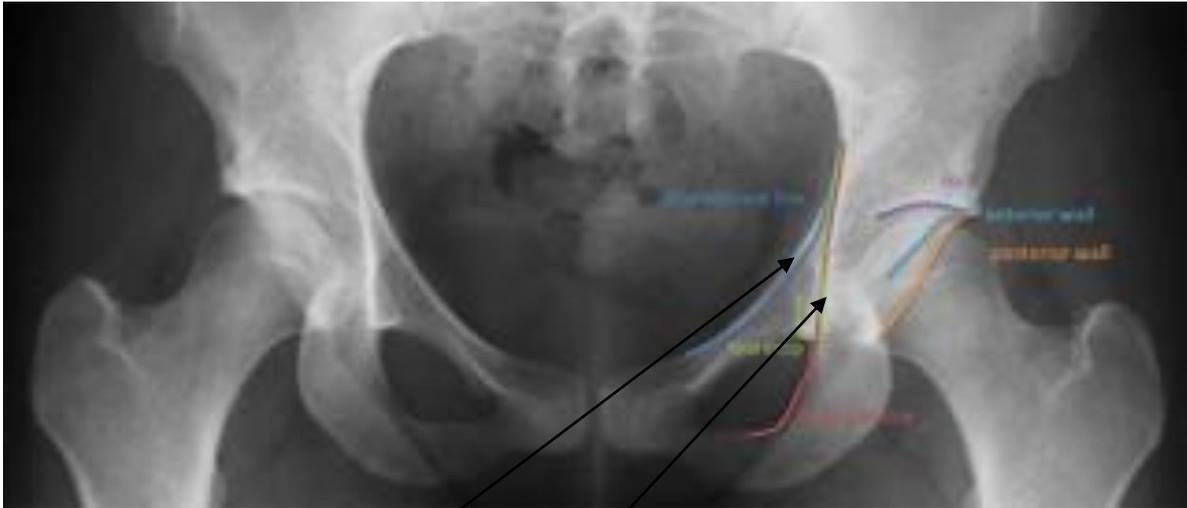


Figure 15 X-Ray showing Kohler's line and Iliopectineal line

- Iliopectineal line
- Ilioischial line (Kohler's line)
- Anterior wall
- Posterior wall

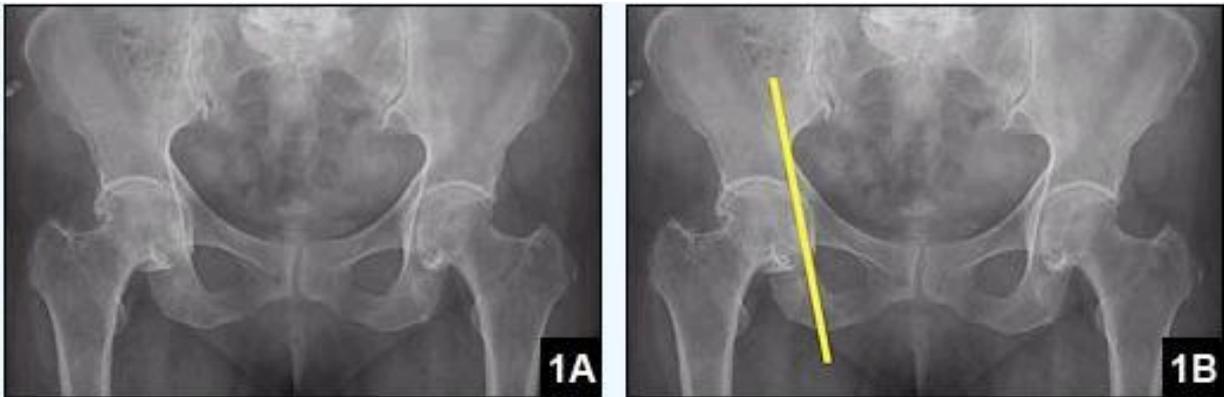


Figure 16 X-Ray using Kohler's Line as diagnosis of protrusio acetabuli

AP pelvic radiograph demonstrating a mild protrusio deformity

(1A) Kohler's line is illustrated takes its course along the lateral border of the obturator foramen to the medial border of the iliac wing and passes through the acetabular teardrop in normal hip joints.

(1B) The medial border of the acetabulum is medial to Kohler's line in protrusio acetabuli

Capsuloligamentous apparatus

The joint has four ligaments contributing to its stability

There are three external ligaments (and one internal ligament)

- **Iliofemoral** - from the ilium to the femur
- **Ischiofemoral** - from the ischium to the femur
- **Pubofemoral** - from the pubis to the femur

These completely encapsulate the joint. Their fibres are directed down and laterally, permitting the greatest range of movement in flexion, abduction and external rotation. In addition to these is the ligamentum teres, which passes from the head of the femur to the acetabulum.

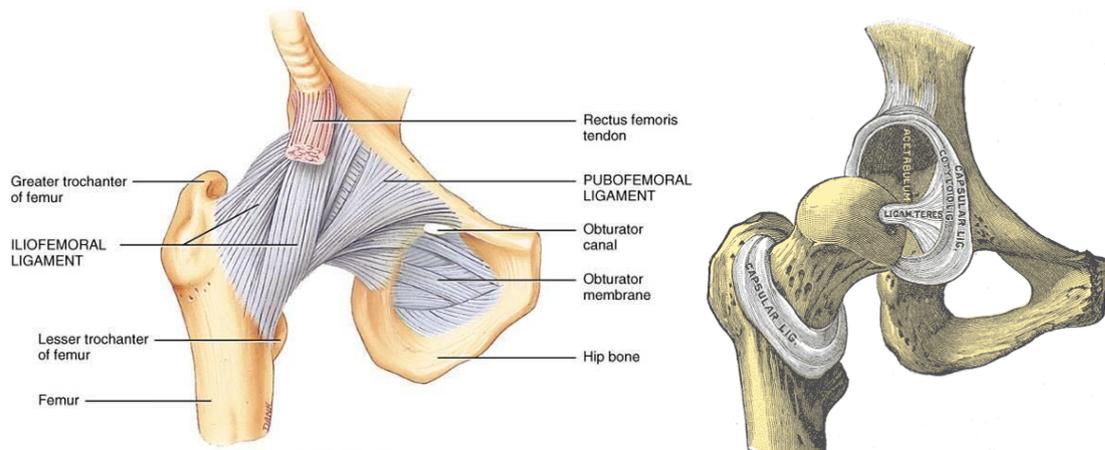


Figure 17 Ligaments of the hip



Figure 18 Section through the hip joint showing the attachments of the ligaments

The joint can be looked at from several viewpoints:

- Range of movement (variations and limitations)
- Capsular ligaments and their possible lesions (e.g. arthritis, trapped ligamentum teres)
- Muscles: the different groups and their actions, causing:
 - Gluteal bursitis
 - Gluteal claudication

- Ischial claudication
- Psoas strain
- Psoas bursitis
- Psoas haemorrhagic bursitis

The 4 ligaments of the hip are;

- **Pubofemoral**
- **Ischiofemoral**
- **Iliofemoral**
- **Ligamentum teres**

Of these, the ligamentum teres passes from the head of the femur into the acetabulum. It can get caught, usually in children after twisting suddenly and causing an awkward movement in the hip. It is possible that the child will not be able to bear weight on that leg, or stand up. As it is cartilaginous, X-rays wouldn't show anything. It is very rare, but can be treated with traction with a little adduction (i.e. distraction), leading to a complete recovery.

Ranges of Movement of the Hip

The range of movement of the hip should always be checked by assessing the good hip first (if they have one):

Extension

- 10 - 15° in adults
- 40° in 2-year-old

The action of extension winds up the fibres of the capsule, which run in a spiral (Fig 6) (via the vertical fibres of the iliofemoral ligament that passes from the AIIS to the intertrochanteric line) and forces the femoral head into the acetabulum. The range of movement therefore becomes self-limiting. At extremes of extension, the tension of the ligaments prohibits any rotation. As the hips are normally in extension in the standing position, this ligament assists posture, affording a strong element of stability. This also has a knock-on effect in the knees; as the hips are in extension, the knees will also tend towards extension, allowing the quads to relax and the patella will become mobile.

Rotation

- Internal - 45°
- External - 30°

[* Note the direction of the spiral fibres of the iliofemoral ligaments, passing anterior and medial around the capsule. Medial rotation will result in a *tightening* of these fibres, pushing the femoral head into the acetabulum and increasing compression. Lateral rotation will *unwind* them. Hence any pathology, sinusitis, reaction to trauma, haemophilia and degeneration will cause the person to adopt a position of greatest ease: *flexion, lateral rotation and adduction*]

Flexion in the hip can be with the knee straight or bent.

- a. Knee flexed: the abdominal wall limits flexion of the hip.
 - This (asthenic) people can go all the way without gluteus maximus being a limiting factor
 - 120° active
 - 140° passive
- b. Knee Straight: about 90°, usually limited by the hamstrings

Adduction

Here the range is small - about 35°. if the other leg does not obstruct it, the lateral band of the iliofemoral ligament limits it.

Abduction

Range:

- 45° in the standing position
- 80 - 90° from full adduction

The pubofemoral ligament is the primary structure that limits movement here, however it is also the spiral fibres of the iliofemoral ligament and by the adductors. The range of movement increases if the hip is semi-flexed; this relaxes off the ischiofemoral and the iliofemoral ligaments.

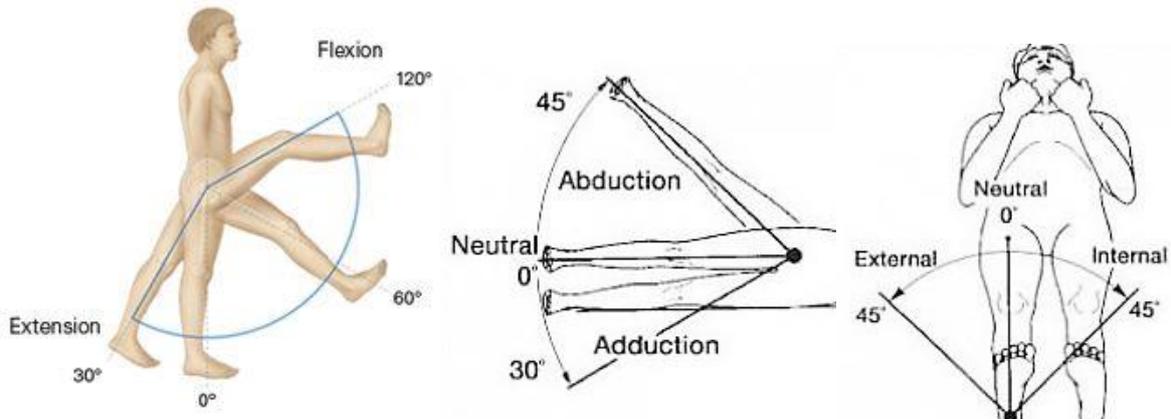


Figure 19 Diagrams showing the ranges of movement of the hip

Muscles of the Hip

Extensors: Adductor Magnus, Gluteus maximus, Hamstrings

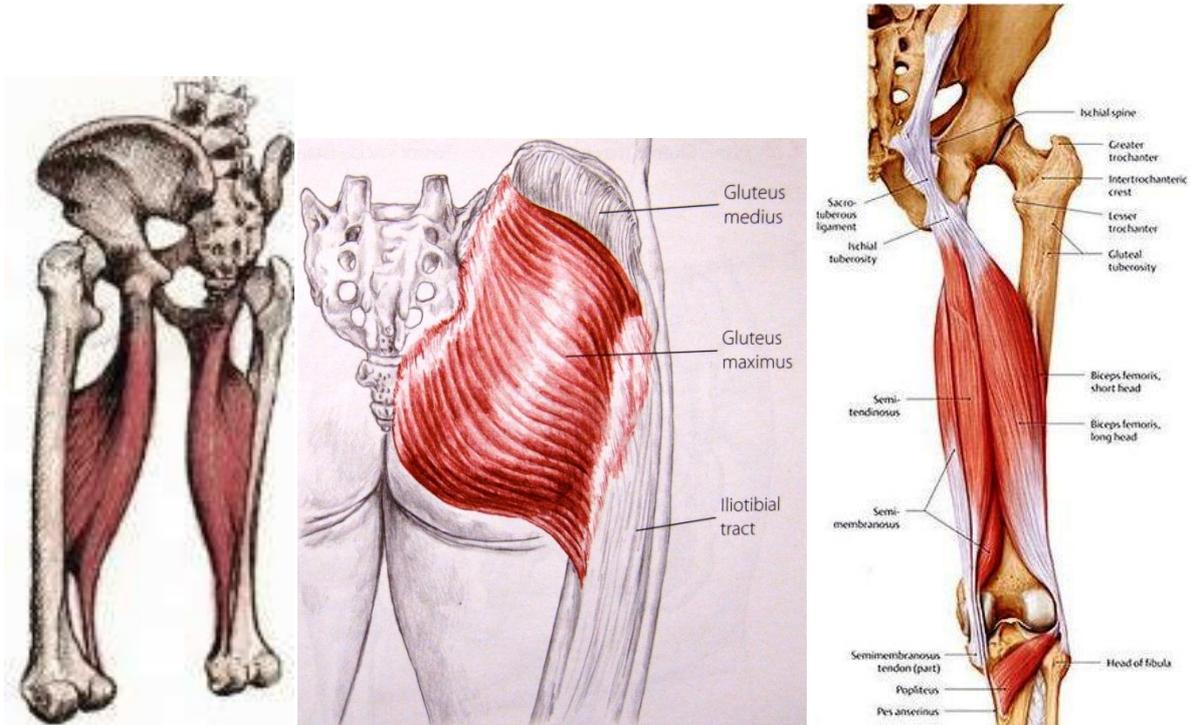


Figure 20 Hip Extensors

Gluteus Maximus

- Largest muscle in the body

- Strongest extensor of the hip joint

Origin

- Iliac crest
- External iliac fossa
- Posterior sacrum
- Sacrotuberous ligament
- Gluteal fascia

Insertion

- Gluteal tuberosity of femur

The superficial fibres of the upper muscle form a thick tendinous lamina, passing across the greater trochanter to the Iliotibial tract (ITT) [in some people, with active flexion/extension and passive internal rotation with a flexed knee, this can roll and 'snap' around the head of the greater trochanter; it causes 'snapping hip' but without pain. No treatment]

Functions

- Locomotion
- Lateral rotator
- Extends hip
- Balances weight of the trunk, especially on hills and stairs
- Changes in position (sitting to standing)

Muscular dystrophy (pseudohypertrophy) affects the extensors of the hip (and sometimes knee). The person has palsy/paralysis of the extensors, causing the characteristic movement of 'climbing up the legs' when standing up from the ground and a waddling gait whilst walking.

Lesions

- **Gluteal bursitis** - the bursa hurts on compression, but not on traction, therefore is difficult to diagnose.

Pain is felt:

- In the posterolateral trochanteric region, with full passive flexion of the hip
- At the end of movement
- Full lateral passive hip rotation
- Passive abduction
- Resisted passive abduction
- With direct pressure

Claudication in gluteal (mid-buttock) region

- Pain, causing a 'limp', in the buttock. The patient can walk 100yds, then gets a pain in the buttock and needs to stop and rest, then can go on, then rest, then go on etc.
- The main characteristic is that the distance is the same
- It is caused by intermittent claudication because of muscle ischaemia via partial occlusion of the artery, or because of arteriosclerosis, or it is occurring at the level of the cord [causing pins and needles via a reduced blood supply at the base of the spinal cord]
- The main cause is ischaemia. With this, the pulse at the ankle will be normal, however the pulse at the femoral will be reduced.

Differential diagnosis between glutei and cord (spinal claudication)

- With spinal claudication, will get neurological symptoms bilaterally

- Get mushrooming of L-disc, causing pain with sitting or standing for a while
- Pain with general weight bearing and increased with the day; no pain with walking

Treatment of claudication

- Rest
- If you doubt because the ROM is normal, ask the patient to lie prone and contract their glutei for a few minutes; will reproduce symptoms and pain

Ischial bursitis (Weaver's bottom)

- Pain on the ischial tuberosity
- Can reproduce this pain with local pressure and with excess sitting
- The pain stops immediately on getting up

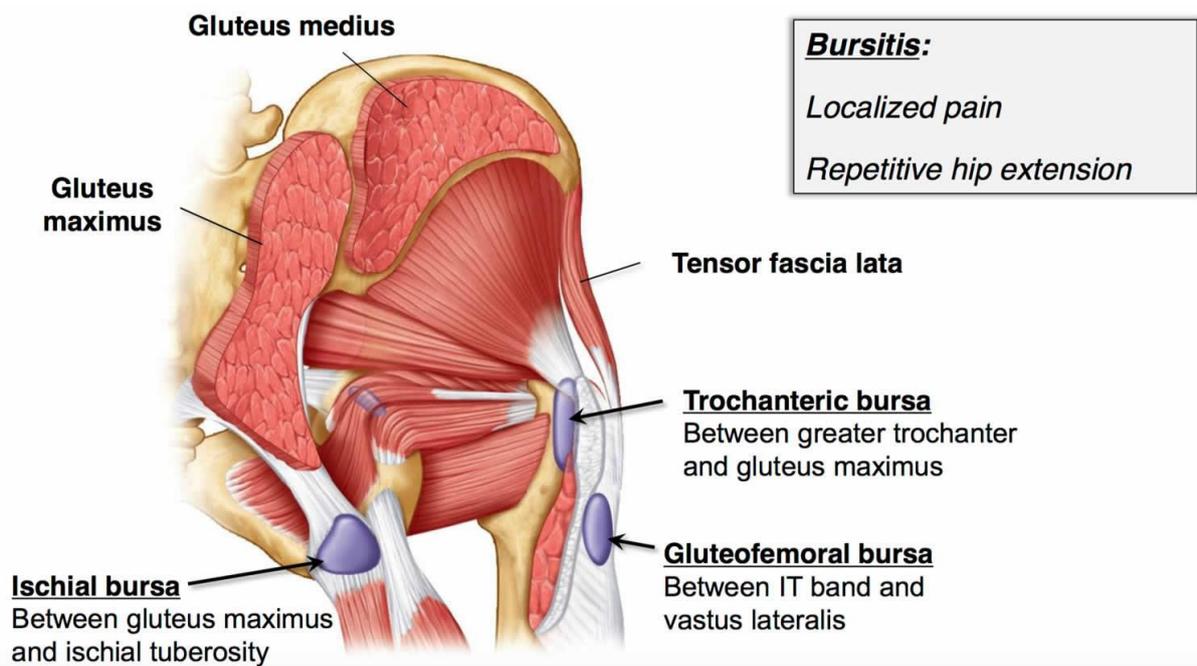


Figure 21 Ischial Bursitis - Weaver's Bottom

Hamstrings

- These are the primary extensors of the hip, but also have a postural function, ensuring stability of the pelvis of the femur.
- Normally, the centre of gravity tends to fall forwards; here the hamstrings tend to pull you back.
- They are very strong muscles
- They can tear (usually in the lower regions)
- Tearing frequently occurs when there is a sudden contraction when the muscle is in a lengthened position (e.g. cricket, football, rugby, hurdles, cross-country running)
- Tearing of the hamstrings can occur when it is fully extended; this can be seen in a lot of body building.

Flexors

- Psoas
- Iliacus
- Rectus femoris
- Tensor fascia lata
- Adductor longus
- Sartorius

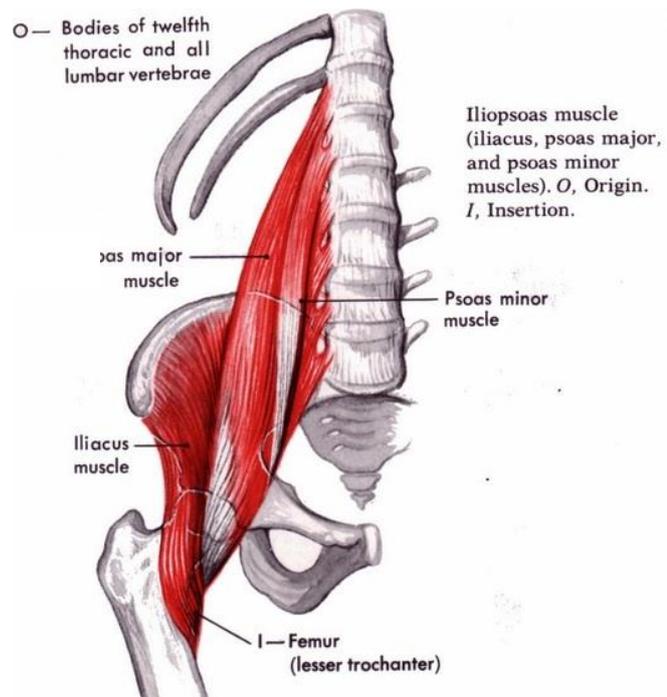
Psoas

Of all the hip flexors, Iliacus and Psoas (iliopsoas) are the most important ones, not only for their bulk and volume, but also for their attachment on the lumbar spine.

Whenever Psoas is in spasm as part of a 'pelvis imbalance', visceral dysfunction, or spinal or abdominal disease, both flexion and extension will be limited. It can cause

- Low back pain
- Groin pain
- Testicular pain
- Muscular pain in the quads and adductor groups

Figure 22 Iliopsoas



Attachments

Medial

- Attaches to T12 and the vertebral bodies and the intervertebral discs of the lumbar spine

Lateral

- It blends with the anterior layer of the Thoracolumbar fascia
- Inferiorly it blends with the fascia of the Iliacus muscle

Inferiorly

- It extends behind the femoral artery into the thigh and attaches to the lesser tuberosity via the Psoas tendon

Superiorly

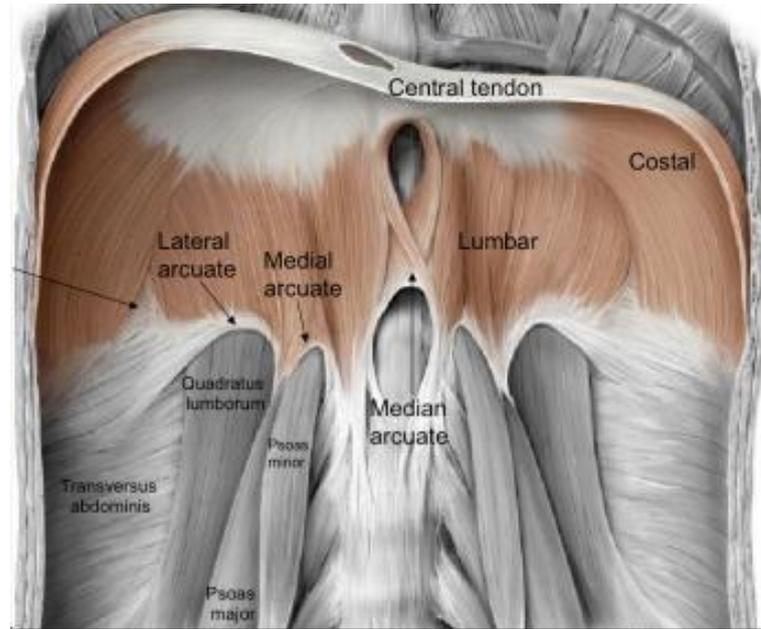
- The TP of L1 laterally
- The body of L2 medially
- The upper edge of this connection forms the medial lumbar costal arch (median arcuate ligament). It forms a funnel shaped aperture that opens into the posterior mediastinum

Figure 23 Arcuate Ligaments

[NB the lower thoracic and lumbar spine. Any disease at this level with abscess formation, the pus from it will track down following Psoas, underneath the fascia of the muscle itself. This can appear as a swelling in the groin, or the lesser trochanter]

Symptoms/signs

- Painless
- Lateral to femoral artery
- Impulse when patient coughs



Differential diagnosis between abscess and local cyst

- A cyst will not increase in volume and push the fingers away with coughing
- There may be another swelling above the inguinal ligament and have a pressure link between it and the inferior one; press one flat and the other one increases, hence there is a fluctuation between the two

Psoas dysfunctions

- Strained
- Bursitis
- Haemorrhagic

Strain

Strains are very uncommon. If you find one, rejoice as they respond well to treatment. They may accompany O/A of the hip or the spine.

Clinical presentation

- Pain at the front of the upper thigh, radiating down the patella area
 - This is because it develops from L2 and L3 segments and retains that innervation
 - The patient complains of pain on walking
 - Onset is gradual. NAR. Can be going on for years

O/E

- Pain with lateral rotation
- Medial rotation and flexion are pain free
- The bursa is stuck between the stretched muscle and the rotating head of the femur
- All active resisted movements are pain free (pain only when the bursa is put under pressure)
- Must check rectus femoris (via hip extension)

Treatment

- Often, sadly, only responds to local injections of hydrocortisone; all other work is no help, though ultrasound may help

Haemorrhagic bursitis

With haemorrhagic bursitis there is enlargement of the bursa with bleeding into it.

- Very sudden onset:
- The patient will report a jarring of the thigh, with onset of pain within one minute
- Pain with hip extension
- No pain or weakness with all active resisted movement (no muscle is involved)
- Passive flexion is limited to 90°
- Weakness here is always serious (especially in the C-Spine as it could indicate a tumour)

Weak and painful

Abdominal neoplasm infiltrating Psoas

Metastases in upper femur - can't stabilise
Psoas in contraction

Traction (avulsion) fracture of lesser trochanter; or traction fracture to AIIS [onset is never sudden and symptoms may vary.

Occurs in adolescents

Psychoneurotic patients

Weak and pain free

Neoplasm of spine

Congenital dislocation of hip

Herniation of lumbar disc

Possible diagnosis for pain in the groin

Partial ureteric obstruction

- Caused by retroperitoneal fibrosis
- Very rare
- Occurs with certain drugs, especially methylsergide (anti-migraine/vascular headaches)

Arterial blood supply to Hip

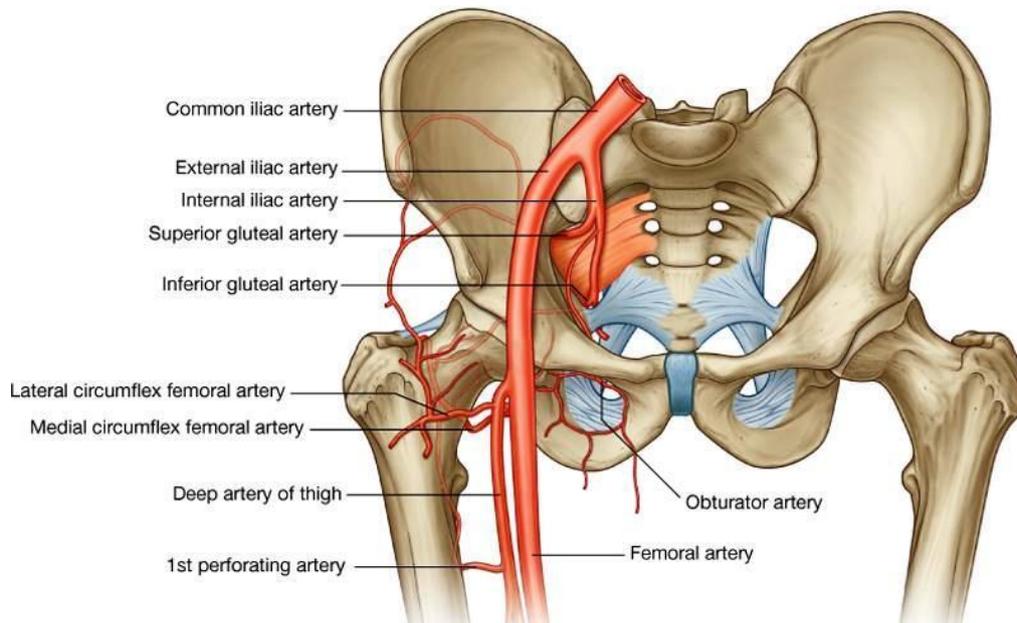


Figure 24 Arteries supplying Hip

Normal supply to hip:

- **Obturator artery**

A branch of the internal iliac artery

- **Femoral artery** - give off branches
 - **Profunda femoris**
 - **Circumflex arteries**
 - **Medial femoral circumflex** - passes back between Pectineus and Psoas. When it reaches the upper border of adductor magnus, it splits and gives off articular branches to the hip
 - **Lateral femoral circumflex** - passes laterally, deep to sartorius and rectus femoris and gives off articular and muscular branches to the hip (any swelling of the synovial membrane in the hip can obstruct the circumflex arteries; more below)
 - **Metaphyseal arteries** (metaphysis - region of growth of bone between the epiphysis and diaphysis during bone growth). These are terminal branches of the arteries of the shaft. They are the predominant blood supply to the head in the first 4 years of growth. As they have to cross the growth plate, past 3 - 4 years of age the plate blocks the supply
 - **Ligamentum teres artery** - this is a branch of the obturator artery and passes along and within the ligamentum teres into the epiphysis of the femoral head. However, it does not supply the head until about 10 years of age. It grows into the ligamentum teres and the acetabulum needs to be ossified for this to happen (else no blood supply). It persists until the person is 20 - 30 years of age (all growth has stopped by 25 years of age) when there is negligible blood supply. In 20% of the population, it doesn't form at all.
 - **Epiphyseal Arteries** - These are the medial and lateral circumflex arteries
 - **Medial ascending cervical artery**
 - **Posterior ascending cervical artery**
 - **Lateral ascending cervical artery**
 - Gives off the **anterior ascending cervical artery**

- All these are crawling between the bone and the synovial membrane and are, therefore, very vulnerable. Any swelling, trauma or increase in synovial fluid can lead to ischaemia.



Figure 25 Epiphyseal Arteries of Hip

Changes in arterial blood supply

- At birth - cartilaginous head
 - Metaphyseal vessels
 - Medial circumflex
- 6 months
 - Metaphyseal artery
 - Medial circumflex, especially its lateral branch
- Critical age of 3-4 years to 8-10 years
 - The hips are very vulnerable as far as blood supply is concerned, as it is dependent entirely upon the epiphyseal vessels
- Over 10 years of age, things improve. The ligamentum teres artery penetrates the femoral head anatomising with the ascending cervical artery.
- 14 years of age female
- 17 years of age male } The shaft and the epiphysis fuse
- Old age
 - The vascular supply reduces and can be absent
 - Poor callous formation in fractures
 - Possible idiopathic avascular necrosis in elderly

Head of Femur - Pathologies of blood supply

- Intrinsic supply to the joint
 - a. Head of femur
 - i. Perthes disease
 - ii. Avascular necrosis
 - iii. Slipped epiphysis (page 32)
 - b. Shaft of femur
 - i. Paget's disease
 - ii. Osteomyelitis
- c. Extrinsic supply
 - Syndrome of Leriche, arteriosclerosis obliterans

Perthes Disease (aka Legg Calve Perthes Disease)

- This is a self-limiting condition of the hip caused by a varying degree of ischaemia and subsequent necrosis of the femoral head.
- There is avascular necrosis of the proximal femoral epiphysis, abnormal bone growth of the epiphysis and eventual remodelling of regenerated bone
- Loss of blood supply to the epiphysis is thought to be the essential lesion
- It is usually seen in 4 - 8 years old boys with delayed skeletal maturity
- Male: female ratio is 4-5:1
- Rare in blacks
- There is an increasing incidence with a positive family history, low birth weight and abnormal pregnancy/delivery
- Up to 12% of cases are bilateral, but can be at different ages and can be asymmetric
- Age is the key to a poor prognosis; after 8 represents a poor prognosis

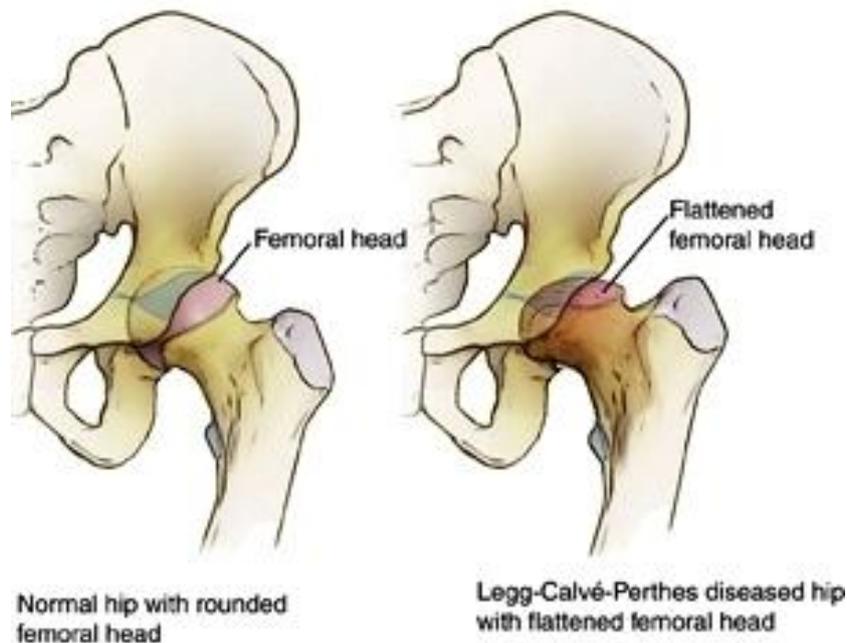


Figure 26 Perthes Disease

Pathogenesis

- Avascular necrosis of the femoral epiphysis, which results in a delayed ossification nucleus
- The articular cartilage is nourished by synovial fluid, so continues to grow
- The cartilage columns become distorted with some loss of their cellular components, they do not undergo normal ossification, which results in excess calcified cartilage in the primary trabecular bone
- The revascularisation proceeds from peripheral to central
- Symptoms can occur when there is subchondral collapse and fracture

Avascular Necrosis of the Hip

This is a condition that only occurs in adults. It is a deprivation of blood supply resulting in bone death. It involves the head of the femur and is likely to develop after a severe injury to the region. Necrosis is probable in:

- Posterior part of the capsule, from a local tear; possibly from a dislocation
- A fracture of the neck of the femur, especially if the line of the fracture is intracapsular and is vertical or subcapital
- Inadequate reduction of a fracture an predispose to avascular necrosis

Other causes:

- Caisson's disease (decompression sickness) causing massive bony infarction
- Prolonged doses of steroids (e.g. after organ transplant)
- A black person with sickle cell anaemia (it can affect many tissues)
- Idiopathic avascular necrosis

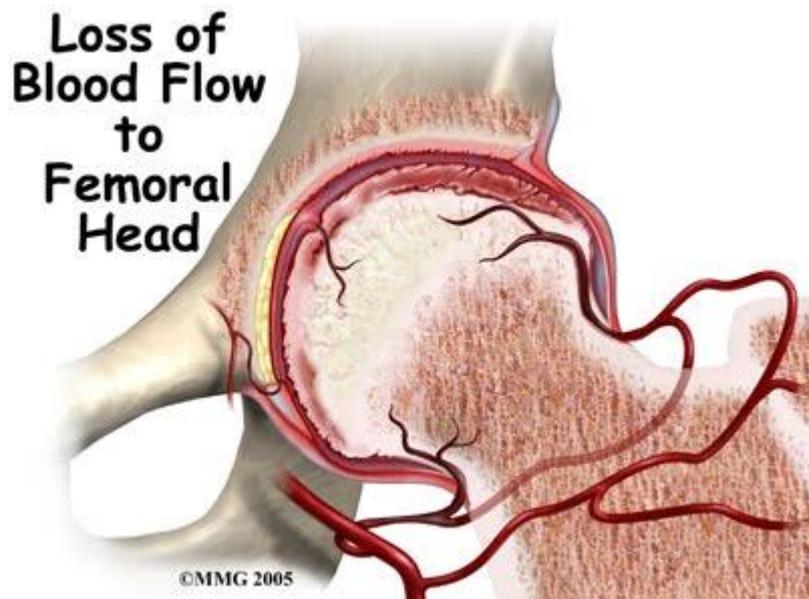
Anatomopathology

It is similar to Perthes in that there are three stages:

1. Necrosis
2. Deformity
3. Revascularisation and repair

However, it leaves behind a joint that is weak

Figure 27 Avascular Necrosis of Femoral Head



Idiopathic Avascular Necrosis (AVN)

Here the patient presents with no obvious cause, but with positive X-Rays.

One theory

- Associated with a subchondral fracture (i.e. under the articular cartilage) or osteochondritis dissecans
- The transarticular forces and muscled forces across one hip can make the weight on it three times the weight on the other
- Then the patient slips, and a small sub-trauma can occur without the patient realising it
- It can cause a minute subchondral fracture, or a gradual roughing of the cartilage, with a flake splitting off
- Also, a sudden muscle contraction, especially is the bone is decalcified, can also cause a fracture and a AVN

Figure 28 Flaking of the femoral head

The dissected fragment can 're-glue' itself if it remains vascular at its base.



X-Rays



Figure 29 Avascular Necrosis of Femoral Head

- A wedge-shaped area of necrosis situated at the superior, weight bearing, portion of the head, this experiences the maximum forces
- Below the outline of that is the fracture line
- Below that is the sclerotic bone, which is a reaction of living bone to compensate (i.e. thickened)
- At first the articular cartilage width remains the same. Later, if the person keeps weight bearing, the cartilage width will decrease as the necrotised area softens
- As the area gets its nutrition from the synovial fluid, the shape will remain unaltered. However, if weight bearing is permitted, the cartilage will become fissured and the underlying bone disintegrates

From this it can be seen that early degenerative osteoarthritis changes can take place

Clinical presentation

- Males: females 4:1
- Ages - 20 - 50 yoa
- 20-30 yoa usually associated with AVN and osteochondritis dissecans
- 40-50 yoa associated with subchondral fractures

Why?

- 20-30 yoa are much more active and the cartilage is much 'juicier' and can take trauma
- 40-50 yoa - the bone is much more fragile and is associated with subchondral fractures

Pain

- Pain can be referred to the knee
- It is usually low grade, more anterior and medial (it refers via the obturator nerve)
- A slight limp can be present

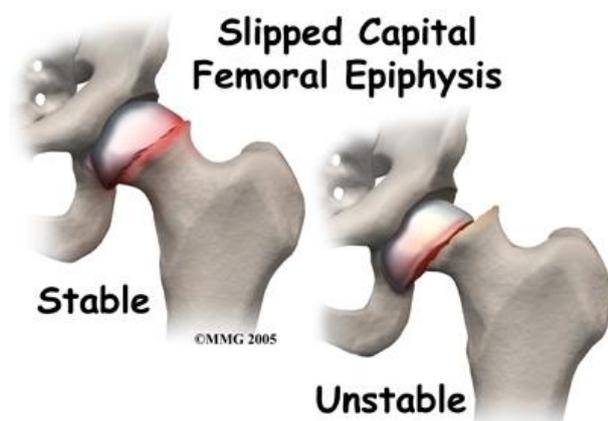


Figure 30 Perthes Disease of the Hip - here seen on the right

Slipped Epiphysis

Slipped epiphysis (aka Slipped Upper Femoral Epiphysis - SUFE or slipped Capital Femoral Epiphysis) is a displacement of the head of the femur at the level of the epiphyseal line.

Figure 31 Slipped Epiphysis



Characteristics of a slipped Epiphysis

- It is a displacement of the head of the femur at the level of the epiphyseal line
- The displacement is always down and backwards
- It is always associated with a varus deformity (the neck is more horizontal)
- The slip takes place through the substance of the growth plate
- After the slip, some cartilage is left on both sides of the line
- It carries its blood supply with it, creating a kink in the blood vessel and can lead to AVN
- It strips the periosteum from the back of the neck
- The body can compensate by forming a bony beak, or ridge, at the back of the metaphysis, an exostosis

Causes

3 theories:

- **Traumatic** } These are accepted totally
- **Hormonal** }
- **Dietary** - certain causes incriminate this

Hormonal imbalance

Here there is a discrepancy between the level of growth hormone and the sex hormones in adolescents of that age. The ratio is important, as it should be equal.

The level of growth hormone increases in adolescence, and it stimulates the growth disc, causing additional cartilage to be laid down. If this proceeds too quickly, the cartilage can become too soft.

Sex hormones convert the additional cartilage to bone. Hence if there is an imbalance, the sex hormones can't keep up. It results in too much unossified cartilage at the growth plate. It becomes too soft and cannot withstand the stress of the body weight (which is also increasing at this age). Thus, generally:

- Growth hormone decreases the shearing strength of the epiphyseal plate
- Sex hormones increase the shearing strength at the epiphyseal plate

There are three typical types of person to suspect of a hormonal cause of slipped epiphysis

- 1) **Adipose Genital** - commonest cause
 - Usually teenage males; little fat boy
 - Knees have slight valgus
 - No secondary sexual characteristics present
 - Body hair absent
 - Possibly undescended testes, or underdeveloped
 - Body size normal

All these indicate:

- A lack of growth hormone, possibly due to an underactivity of the anterior pituitary/hypothalamus
- Frohlick's syndrome
- Tendency to lethargy

2. Opposite

- a) Adolescent with too much growth hormone
- b) Tall, thin, adolescent who is growing too quickly; long limbed
- c) Due to anomaly (over activity) of anterior pituitary, therefore growing too fast

- d) Bright. Alert, normal sexual characteristics
- e) Possible underlying pathology - adenoma (tumour) in anterior pituitary
- f) May lead to gigantism/acromegaly

3. Normal - nothing to be seen

- a) No special characteristics
- b) No underlying pathologies
- c) No cause for potential hormonal imbalance
- d) Idiopathic

Dietary - not well-known theory

- Lack of vitamin A, D incriminated
- Amino-nitril is thought to be responsible. These are contained in sweet peas family of vegetables. They are also fed to cattle. Switzerland and Sweden have noticed 70% increase in slipped epiphyses at the end of the summer

Trauma

Why are adolescents most vulnerable at the level of the hip?

- 1) Increase in body weight
 - a) Increase in muscle bulk
 - b) Therefore, increase in intra-articular forces put on the hip
- 2) Increased activity and sports during adolescence
- 3) Epiphyseal plate running out of blood
 - a) Supplied by metaphyseal arteries
 - b) As growth occurs, there is an increase in the length of the terminal arterial branches from the primary source

Clinical presentation

- 1) Sudden
- 2) Slow, chronic, insidious
- 3) Acute on top of chronic

Acute - sudden slip, like a trauma

- Sudden onset
- Patient feels something 'give'
- Weight bearing becomes impossible
- In theory, it is possible to reduce the slip if it is done immediately with flexion and internal rotation
- Even with reduction, it needs to be pinned surgically. This, in practice, is not as good as it seems

Chronic slip - slow slide

- Usually from repetitive, minor, stress fractures
- A very severe deformity can be reached without the patient being aware of any symptoms, it being very insidious
- The patient keeps walking with an increased limp
- Shortening of the leg is present - as much as 2 ins
- Can have trivial aches in the front of the thigh, or the superior patellar region

Acute on a chronic slip

- A sudden trauma precipitates an acute slip
- The leg is:
 - Shortened
 - Adducted (for protection)

- Externally rotated
- Hyperextended (this is the only time this occurs)

Silent symptoms

- Age group - 10 - 20
- Males - mostly 14-16; ends with closure of epiphyseal plate
- Females - mostly 11-13; almost impossible to slip after started menstruating

Symptoms and signs of Slipped Epiphysis

- Shortening of the limb
- Varus
- External rotation - standing or lying, there is one foot out
- Abduction decreases
- Adduction increases (due to coxavaram)
- Loss of internal rotation
- Hyperextension with the loss of flexion
- If there is any muscle spasm around the joint, it suggests a discontinuity between the head and the neck
- May be bilateral
- An accident can precipitate a slip as there is an underlying condition

X-Rays

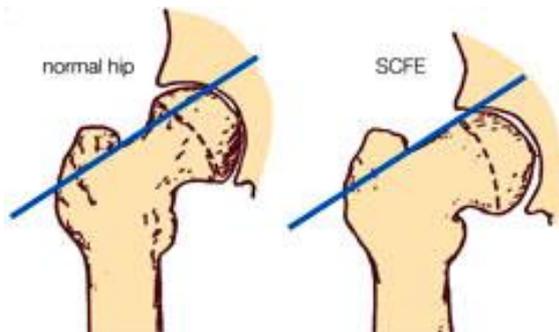


Figure 32 SUFE demonstrates a flattening of the femoral head



Figure 33 X-Ray SUFE A/P view

- X-Rays are always taken in A/P and lateral
- It shows a flattening of the head in adults
- An early slip will only show on the lateral view

Figure 34 SUFE lateral view

- It can be easily missed
- There are 1st, 2nd and 3rd degrees
- Calculated by the diameter of the metaphysis



- X-Ray evidence appears 2-6 months after onset



Figure 35 Fixation of SUFE, right hip fixed with two screws and the left with one prophylactically

Prognosis

- X Rays are repeated 6 months later
- If weight bearing has started before complete repair, the head will collapse, leading to early degeneration and severe O/A changes
- If it is spotted early and weight bearing avoided, there can be a creeping substitution/repair; if the bony architecture can remain intact, no deformity will occur. This will take 18 months - 2 years (not as long as Perthes disease)
- Generally, the outlook is not very good
- It will never be the same size or shape
- The patient should be content that their functioning is satisfactory until middle age (i.e. 40-45 yoa) then hip replacement

Complications

- Avascular necrosis
- Chondrolysis - destruction of cartilage via pathological process, leading to thinning of the cartilage

Treatment

- Avoid weight bearing. The younger the patient, the better the outlook, as the area of necrosis is smaller
- In the 4th and 5th decades, a larger portion of the femoral head is involved
- Surgery is almost always the rule to **fix** the epiphysis; reduction is not enough
- Surgery may include correction of the deformity of the neck, in reduction of the exostosis

Cord Claudication at the spinal level

The patient always has the same symptoms picture: the patient walks for 5 minutes, then gets crippling low back pain. Then they stop and bend forwards for relief. Then they straighten up and resume walking.

Presentation of hip conditions versus age

0-5	CDH
5-10	Perthes, Pseudo-coxalgia
10-15	Slipped epiphysis
20-40	O/A due to the above
Over 40	Degenerative changes Fractures Avascular necrosis

Shaft of Femur – Pathologies of Blood Supply

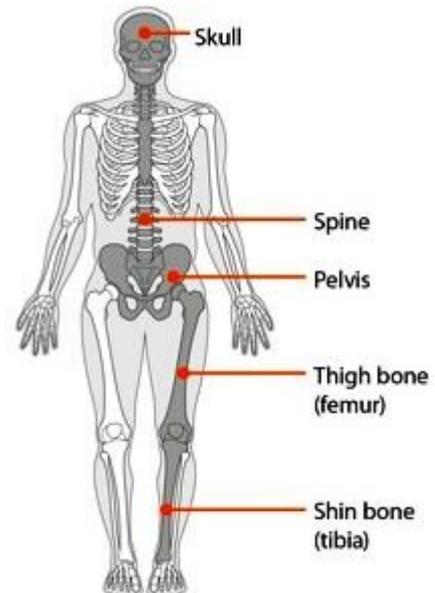
Paget's Disease (osteitis deformans)

Normal bone undergoes a continual process of remodelling. This involves **osteoclast** activity, breaking down bone, and **osteoblast** activity, forming new bone. Paget's disease causes a malfunction in this process, with the new bone that is laid down being soft and porous. Soft bones can be weak and bend easily, leading to a shortening of the affected part of the body. The bone replacement takes place very quickly and an excess may be formed. This may cause the bone to get large, painful and may break easily. The bone affected by Paget's tends to have more blood vessels, resulting in the area feeling warmer than usual.

Paget's can affect any bone, but most commonly:

- Spine
- Pelvis
- Femur
- Tibia

Figure 36 Paget's distribution



Paget's can also predispose to other conditions, such as O/A, kidney stones and heart disease

Paget's:

- Affects more men than women
- Usually people over 40 yoa
- No apparent cause
- 30% sufferers have other family members with it
- It is a worldwide condition, but more prevalent in Europe and Australia; sufferers more likely to have an Anglo-Saxon descent, hence it may have a genetic factor
- The symptoms may be so mild that the person has no symptoms at all
- It may manifest as cutaneous rash in genital area
- No known cure

Warning Signs

- As it has a very slow onset, many people do not know they have it
- The first symptom may be pain in or over a bone
- The affected area may feel warm
- Tiredness
- If it affects the leg, it may change its shape such that it bows outwards

Figure 37 Paget's bone scan showing bowing of the leg

- If the head is affected, it may start getting bigger and the hearing may be affected (fig. 38)
- Usually only affect one or two bones

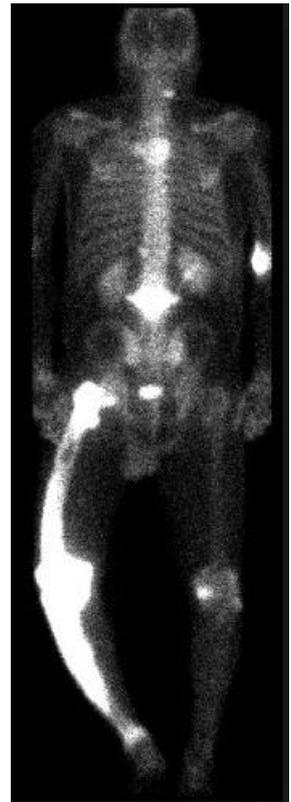


Figure 38 Paget's in upper femur

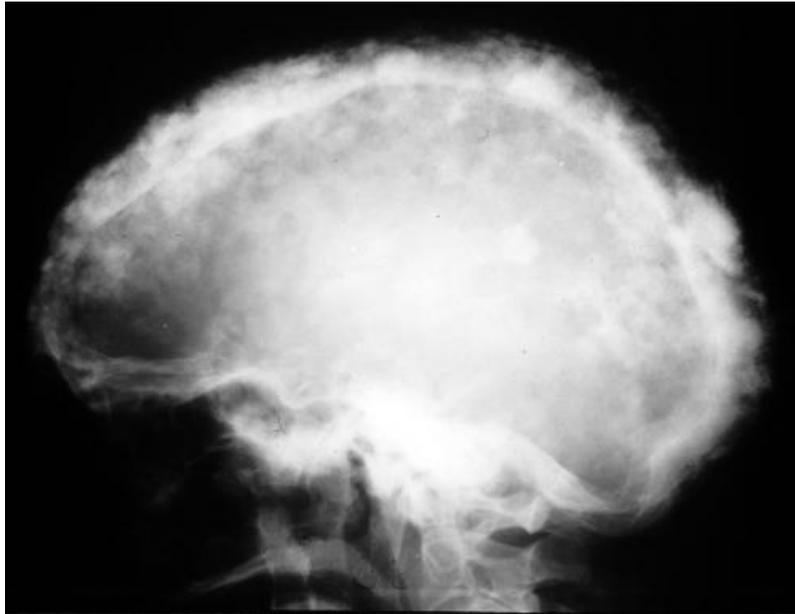


Figure 39 Paget's Disease of the skull

Medicines Prescribed

- Bisphosphonates
 - These have been shown to be helpful in rebuilding bone, in reversing bone loss and causing the body to produce normal bone
- Calcitonin
 - This hormone decreases blood calcium and increases bone density. It reduced bone destruction and can also reduce pain. Often Calcitonin from eels and salmon are used; it is many times more powerful than the human form
- Calcium supplements are recommended
 - Milk, cheese, yoghurt, salmon, sardines, dark green leafy vegetables, almonds, and broccoli

Exercises

- The pain and swelling of Paget's can make the joints stiff. Not using it will cause them to become weak; exercise will help reduce the stiffness and keep the joint moving
- The wrong type of exercise can make the condition worse; some may even cause the bones to break

Hot and cold can give temporary relief symptoms

- Heat helps to reduce pain and stiffness via increasing circulation
- Cold helps numb the area via reducing blood flow; it can help reduce inflammation

Advice

- After heavy or repetitive activity; **rest**
- Use the back and limbs wisely to avoid putting stress on them (e.g. use a trolley for shopping)
- Maintain an ideal body weight to minimise the strain on the bones

Surgery may be necessary if a joint becomes too damaged, though this is rare

Osteomyelitis

Osteomyelitis is an infection of the bone. This is usually around the cortex, medulla, and periosteum. The main causative agents are:

- Staphylococcus aureus
- Escherichia coli (especially in the very young and elderly)
- Salmonella (particularly in sickle cell patients)
- Mycobacterium tuberculosis

In osteomyelitis, the infective organism gains access to the medullary cavity of the bone via two main routes:

- Direct access through an open wound
 - This is an important cause after trauma, particularly an open fracture, can could lead to failed or delayed fracture repair
- Blood-borne spread
 - Following bacteraemia from a focus of sepsis elsewhere, e.g. acute pyelonephritis

Occasionally, bone infection can be a complication of sepsis in adjacent tissues or organs e.g. mastoiditis complicating bacterial middle ear infection

In all forms of osteomyelitis, (except TB) the marrow cavity becomes filled with purulent, acute inflammatory exudates, leading to necrosis of bone trabeculae. The destruction of cortical bone tissue may lead to a discharge of pus into extraosseous connective tissue and the infection may track through to the skin surface (through the easiest route, not the shortest), producing a chronic discharging sinus.

Because the infection is localised to the confinement of the marrow cavity, the pus has little chance to drain without surgical intervention; the inflammation tends to be chronic, with organism remaining viable in the marrow cavity for many years.



Chronic osteomyelitis

Chronic osteomyelitis results with extensive bone destruction, marrow fibrosis and recurrent focal suppuration. With this there is reactive new bone formation, particularly around the inflamed periosteum, leading to a thickened and abnormally shaped bone. Predisposing conditions to this are:

- Sickle cell anaemia
- Septic arthritis
- Diabetes

X-Rays

X-Rays will be positive after 3 days of symptoms

Newborn



Figure 40 Osteomyelitis in newborn

In infants below 1 yoa, some metaphyseal vessels may traverse the epiphyseal plate to the epiphysis and adjacent bone, this occurring mainly in the hip

Child



Figure 41 Osteomyelitis child

In the child the bones are developed and now enveloped in a tough periosteum and this sleeve may be lifted away by the dissecting pus. As the periosteum is firmly attached to the bone is the region of the epiphyseal plate, it forms a barrier, preventing the spread of the infection to the adjacent bone.



Figure 42 Osteomyelitis Adult

Clinical findings:

- Possibly high fever
- Metaphyseal tenderness is most often present
- May be an uncommon cause of a limp in children
- Look for subtle extremity swelling and/or subtle loss of ROM
- X-Ray may show a well circumscribed subchondral lesion with well-defined trabecular margins
- Bone scan is often positive



Figure 43 Osteomyelitis showing subchondral lesion

Pathology of external blood supply

Syndrome of Leriche (Arteriosclerosis Obliterans)

Symptoms

- The symptoms are of arterial insufficiency in the lower limbs (but can happen at any level of the spinal cord)
- It is characterised by cramp-like pain in the calf (mostly) during walking (i.e. using the muscles)

The aetiology is ischaemia; there is insufficient oxygen to the muscles and the metabolites cannot be removed quickly when the muscles are exercised. The patient rests, the metabolites are drained (via the blood supply being restored) and the pain abates.

Underlying pathology

The cause is arteriosclerosis, (though the term atherosclerosis is also used) involving local thrombosis in the main vessel. Here low-density lipoproteins are taken up by macrophages under the endothelium.

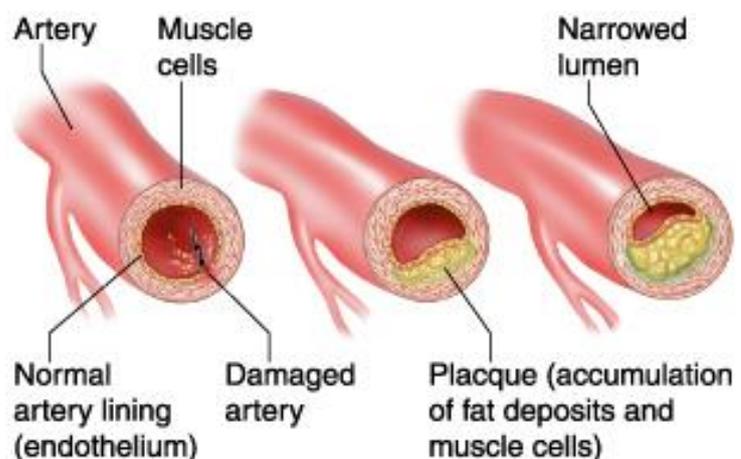


Figure 44 Arteriosclerosis

This accumulation of fatty material leads to a narrowing of the diameter of the blood vessel. This can lead to an upset of laminar flow, with local turbulence and possible thrombus formation.

Atherosclerosis affects arteries throughout the body (i.e., arteries in the heart, brain, kidneys, and extremities). It is the underlying cause of the majority of cardiovascular events (Roger, Heron). It is the leading cause of morbidity and mortality worldwide in most industrialized countries.

Incidence and Prevalence: Thirty-seven percent of men and 35% of women were living with cardiovascular disease in 2008. Over 800,000 people in the US died that year of cardiovascular disease (CVD), 52% were women. Men aged 35 - 75 are more likely to have a heart attack or fatal coronary heart disease, but after age 75 women outpace men in fatal events. At any age, women are more likely to die of their condition and less aware of their risk compared to their male counterparts (Roger). The use of hormone replacement therapy to reduce the risk of heart disease is controversial and complex. Initiating HRT at or shortly after the onset of menopause may reduce the risk of atherosclerosis over time in some women.

Causation and Known Risk Factors

Conventional risk factors for atherosclerosis are well known and include increased plasma cholesterol, cigarette smoking, hypertension, diabetes, obesity, age, sedentary lifestyle, and heredity.

Diagnosis

History: Individuals with atherosclerosis may have symptoms associated with reduced arterial blood flow and oxygen delivery to one or more organs (ischemia). If ischemia is prolonged, it may result in death (necrosis) of cells. When an area of tissue is affected, cell death is commonly known as infarction. Ischemia may be an acute or chronic condition, whereas infarction is only acute

Fundamental to the treatment of atherosclerosis and its consequences is risk factor modification

Differential Diagnosis

- Monckeberg's sclerosis
- Non-infectious arterial inflammation (Takayasu's arteritis, Kawasaki's disease)
- Polyarteritis nodosa

Other causes:

- Thromboangiitis obliterans (Buerger's Disease)
 - Occurring in Jewish males, predominantly in USA
 - Acute inflammatory occlusion of small to medium sized arteries
 - Affects upper and lower limbs
 - Extends to adjacent veins and nerves
 - Segmental inflammatory infiltration of walls of arteries and veins with secondary thrombosis
- Acute case (unlikely to see this)
 - Arterial embolism in vessel

Clinical presentation of Syndrome of Leriche:

The symptoms correspond to which vessel has the partial obstruction

- Femoral } Mostly
- Popliteal }
- Iliac
- Bifurcation of the aorta

It can be unilateral or bilateral

The patient complains of:

- Low back ache
- Fatigue in lower extremity
- Subjective paraesthesia at the level of the lower extremity
- Intermittent claudication
- Males find it impossible to maintain an erection (be careful to differentiate with psychological problem)
- Loss of hair on the skin of the leg
- Nails brittle and irregular
- Skin thickened and shiny

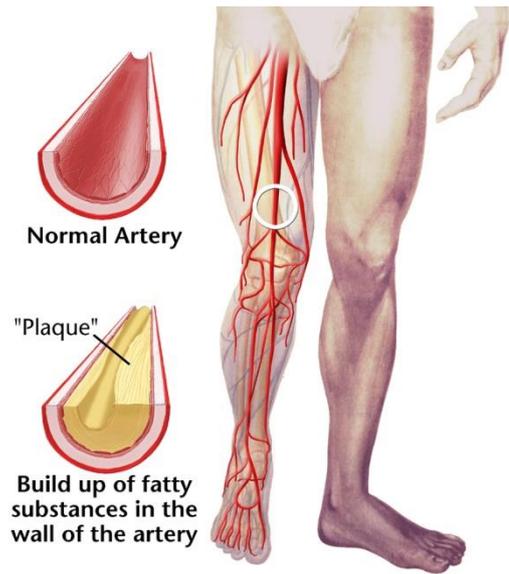


Figure 45 Arteriosclerosis of Femoral Artery

Sites	Pain
Abdominal aorta	Low back
Iliac	Buttock
Femoral	Thigh
Popliteal	Down leg

On examination:

- 1) Weak or absent pulses in lower extremity. If suspected, test all 4 pulses
- 2) Elevation of lower limb
 - a) Elevate on affected side- pallor very quickly on that side
 - b) If it is the lower limb, get the patient to sit/stand (i.e. the limb is in a dependent position), on the affected side there will be a brick red rubor
- 3) Temperature can be lower on affected side (though not reliable)
- 4) There is evidence of widespread arterial disease
- 5) There may be widespread trophic changes (though often not)
 - a) Here there is a variety of opinions, depending upon the extent of the arterial pathology. A collateral circulation will only establish itself if there is significant obstruction

Physical exam may reveal a whistling sound (bruit) heard with a stethoscope placed directly over a narrowed, but not completely closed artery. A diminished pulse may be felt in an artery beyond a narrowed segment of the vessel.

Examination of the small blood vessels (arterioles) in the retina of the eye with an ophthalmoscope can be valuable for diagnosis. Atherosclerotic arterioles reflect light

(emitted by the ophthalmoscope), giving them a "silver wire" appearance.

Tests: Tests done on individuals with suspected or known atherosclerosis include measurement of blood lipids.

Plain X-rays may show calcium deposits in the walls of affected blood vessels that correspond to a diagnosis of atherosclerosis. X-rays with contrast material (angiography) allow visualization of the interior of arteries and permit both a definitive diagnosis of the disease and an assessment of its severity.

Doppler ultrasound, CT, and MRI are other (non-invasive) methods used to diagnose and assess the extent of atherosclerosis.

Treatment

- Stop cigarette smoking,
- Treating high blood pressure
- Controlling diabetes mellitus
- Doing exercise; moderate to the point of comfort - stop before it hurts
- Keep warm
- Attaining an optimal weight
- Lowering plasma lipids are key

Intensive treatment and lifestyle changes can retard or reverse the progression of atherosclerosis. Six to 18 months of such lifestyle changes are necessary to assess whether or not the risk factor modification program is effective.

The three major sources of dietary cholesterol are egg yolks, animal fat, and red meats; however saturated fat intake is more important in determining lipid levels, since humans convert saturated fat to cholesterol. In addition to reduction of dietary cholesterol intake, a cholesterol and LDL lowering drug belonging to a class of drugs known as statins is often prescribed if an individual has a total cholesterol above 200 mg/dL and an LDL cholesterol ("bad" cholesterol) level above 130 mg/dL. Many individuals with atherosclerosis also have a low HDL cholesterol ("good" cholesterol) level. HDL cholesterol may be increased with exercise and small amounts of alcohol, although niacin may also be administered.

A small dose of aspirin (one-half adult aspirin or less daily; e.g. 81mg) may also be given to individuals with atherosclerosis. They act as a platelet antagonist, inhibiting their aggregation. The American Heart Association recommends that men 40 and over with two or more risk factors for atherosclerosis should take a low dose of aspirin daily.

Among individuals with atherosclerosis with localized obstructions that are potentially or actually causing ischemia, surgery may be considered:

- The obstruction may be removed surgically (endarterectomy)
- Bypassed with another blood vessel (bypass surgery)
- Displaced or "crushed" into the wall of the artery with a balloon-tip catheter (angioplasty), with or without stent placement, which serves as a brace to keep the artery open after it has been widened

Prognosis

Atherosclerosis is a progressive disease. It is frequently associated with and complicated by several clinical problems from ischemia or infarction. It carries a high morbidity and mortality with coronary heart disease being most frequent. However, some individuals may have regression of atherosclerosis related to lipid lowering associated with dietary changes or drugs. Several studies have shown that morbidity, progression, and mortality can be slowed through use of lipid lowering drugs for as little as 18 months of therapy

Capsular Lesions

When a person has any lesion of the capsule, both active and passive movements have a 'capsular pattern' (which will be the same for all movements), i.e. a **decrease in medial rotation**. The patient may present with a foot turned out, this following the general picture of **flexion, adduction and external rotation**.

The hip can be a site of psychogenic pain. Here the patient will find relief by internally rotating it. Any condition causing the hip to be fixed in internal rotation is **anomalous**, as it is not consistent with the anatomy of the joint.

Transient Synovitis (aka traumatic Synovitis)

- Only occurs in children under 10 yoa; more frequently in females
- Cause and pathology unknown
- Shows itself as a pain or a limp, with limitation of all ROM
- X-Rays normal
- Short lived condition; 4-6 weeks
- Responds well to bed rest
- Often followed later by Perthes disease

Traumatic Arthritis

- Young adults
- Frequently due to repetitive traumata (often joggers, especially road runners)
- Find capsular pattern (and some pain in all ROM)

Treatment

- Responds well to soft tissue work
- Traction
- Ultrasound
- Rest (i.e. stop jogging)

Congenital Dislocation of the Hip

Congenital dislocation of the hip (CDH) (aka Developmental Dysplasia of the Hip - DDH) is a condition that affects very young children and disrupts the normal development of the joint. CDH is an aplasia of the hip joint, resulting in the lack of development of the posterior/superior aspects of the acetabular ring. It occurs in 1 - 1.5 of every 1000 births, or even up to 5 per 1000 if aggressive screening system (ultrasound) is in place. In some sub-populations (e.g. North American Indians) it can be as high as 35 per 1000 births.

It may be due to a shallow acetabulum or loose ligaments. It may be even due to the mother's own hormones in the last trimester of pregnancy, affecting the baby's ligamentous integrity, causing them to be looser.

More broadly, CDH may be defined simply as abnormal growth of the hip. Abnormal development of the hip includes the osseous structures, such as the acetabulum and the proximal femur, as well as the labrum, capsule, and other soft tissues. This condition may occur at any time, from conception to skeletal maturity. The author prefers to use the term hip dysplasia, considering it both simpler and more accurate. Internationally, this disorder is still referred to as congenital dislocation of the hip

Normal hip development

- At 4-6 weeks gestation, the hip joint develops from the cartilaginous anlage

- By 7 weeks a cleft appears between the precartilaginous cells that are programmed to form the acetabulum and the femoral head
- By 11 weeks, the formation of the hip joint is largely complete; the femoral head is completely encircled by the acetabular cartilage
- At late gestation, the femoral head grows more rapidly than the articular cartilage, so that at birth at birth the femoral head is less than 50% covered
- At birth, the acetabulum is at its most shallow and most lax in order to maximise hip ROM, which facilitates the delivery process; the hip is uncontained in extension and adduction reflecting the hip's shallowness
- After several weeks, the articular cartilage develops faster than the femoral head, allowing more coverage
- Normal occurrences of hip shallowness and capsular laxity in the neonatal period are initial factors involved in CDH

Associated factors

- Left hip is more involved (?due to foetal positioning?)
- 80% of cases are female
- Breech presentation at delivery (may occur for 24-45% of CDH cases)
- Most frequent in first born
- More frequent if the hip is held and fixed in extension and adduction
- Down's Syndrome

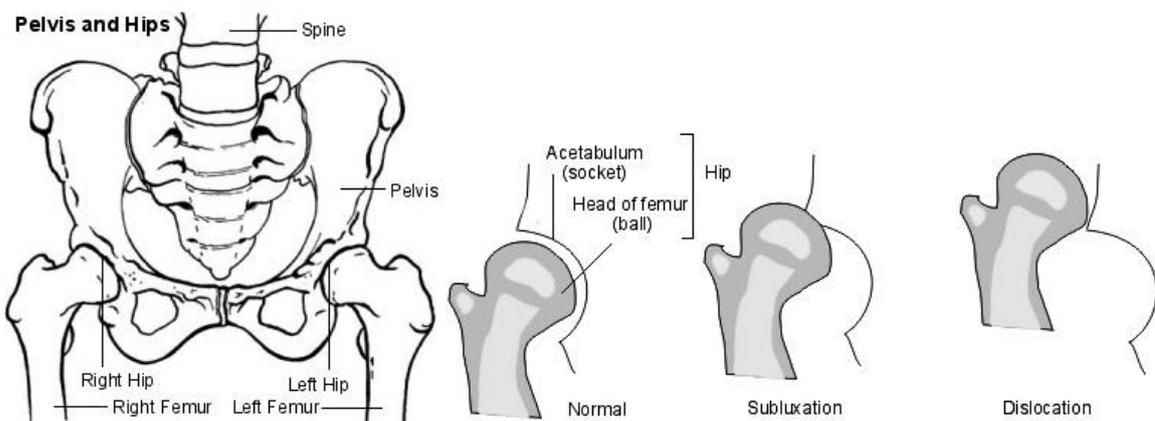


Figure 46 Normal hip and dislocated hip

- All this results in a shallow acetabulum
- Here the head of the femur tends to slip up and back
- Most frequently found in females (5:1 cf males)
- 1/3 of cases are bilateral (it is easier to miss a bilateral as the patient does not look asymmetrical)

More specific terms are often used to describe the condition more precisely; these are defined as follows:

- **Subluxation** – Incomplete contact between the articular surfaces of the femoral head and acetabulum
- **Dislocation** – Complete loss of contact between the articular surface of the femoral head and acetabulum
- **Instability** – Ability to subluxate or dislocate the hip with passive manipulation

- **Teratologic dislocation** – Antenatal dislocation of the hip

Early clinical manifestations of developmental dysplasia of the hip (DDH) are identified during examination of the new-born. The classic examination finding is revealed with the Ortolani manoeuvre, in which a palpable "clunk" is present when the hip is directed in and out of the acetabulum and over the neolimbus. A high-pitched "click" (as opposed to a clunk) in all likelihood has little association with acetabular pathology. Ortolani originally described this clunk as occurring with either subluxation or reduction of the hip (in or out of the acetabulum). More commonly, the Ortolani sign is referred to as a clunk felt when the hip



Figure 47 CDH on X-Ray

Unilateral dislocations result in significant leg-length inequality, with a gait disturbance and possibly associated hip and knee pain. In addition, hip pain commonly manifests as knee or anterior thigh pain as a consequence of the innervation of the hip joint (obturator and femoral nerve distribution). Typically, true hip pain is identified as groin pain.

The development of a false acetabulum is associated with a poor outcome in approximately 75% of patients. Bilateral hip dislocation in a patient without false acetabula has a better overall prognosis. In fact, a case was reported of a 74-year-old man with no history of hip or thigh pain whose dislocated hips were only discovered shortly before his death

With a bilateral CDH:

- The sacrum tends to be more horizontal and concave than normal
- The ilium is more anteriorly rotated
- The transverse diameter is increased
- The A/P diameter is decreased
- The lumbar spine has an exaggerated lordosis; as the hip is rotated anterior to the leg, so the foot can contact the ground
- This mechanical presentation puts more strain on the posterior S/I ligaments and increased muscular work, especially on the ischial tuberosities (from the hamstrings)

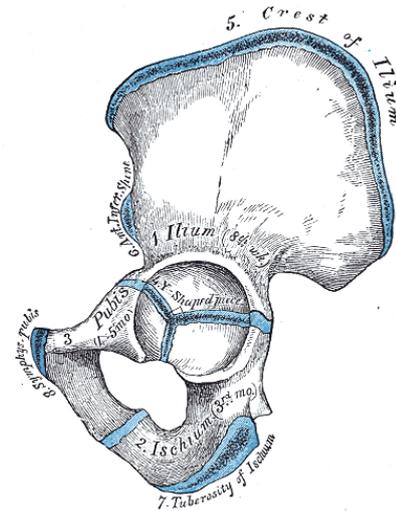
Clinical presentation of CDH

Generally speaking, the older the child, the worse the prognosis

Sometimes the child maybe older before symptoms are noticed that may be indicative of a CDH:

- Late walker
- Walking not symmetrical
- 'Funny waddle'; waddling gait
- Widened perineal gap
- Increased lordosis
- Prominent abdomen

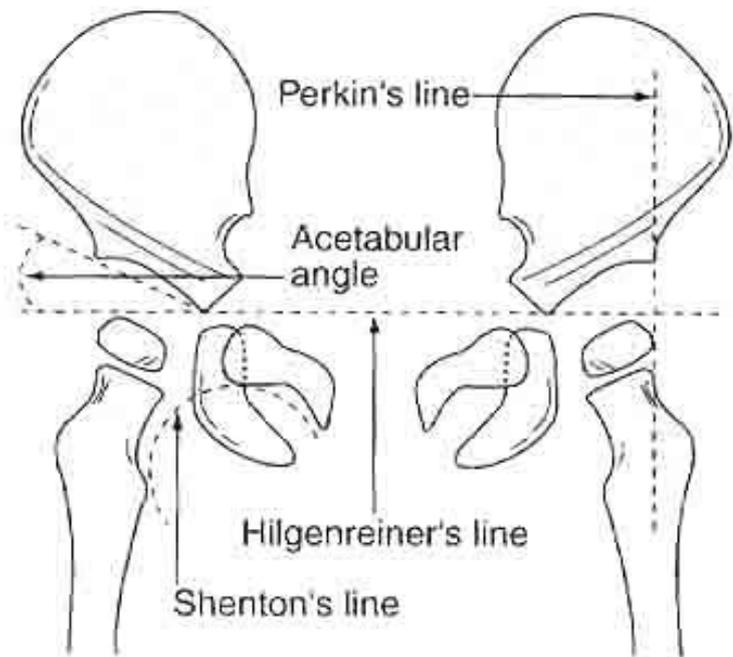
Figure 48 Acetabulum formed by fusion of 3 pelvic bones



The floor of the acetabulum corresponds to the fusion of the three pelvic bones

This junction is not completed until 16 yoa. Until then the cartilage presents as a potential point of weakness. With pressure, trauma, disease etc. the femur can be forced into the acetabular cavity and can be pushed in and even bulge into the pelvis cavity (see Otto's Pelvis)

Figure 49 Diagnostic lines for CDH



There are other parameters of assessment to check for CDH. These are **Shenton's lines**, **Perkin's lines** and **Hilgenreiner's line**

- **Shenton's lines** are lines drawn from the inner surface of the top of the femur; these should be continuous with the upper edge of the obturator foramen
- **Perkin's line** are vertical lines drawn through the AIIS; if the femoral head is superolateral, it is seen as dislocated
- **Hilgenreiner's line** is drawn across the inferior edge of the ilia

If cases are missed, they can lead to life-long problems



Figure 50 CDH - late diagnosis, shown here on X-Ray

Tests for CDH

Barlow's Test

Barlow's test identifies the unstable hip in the reduced position (appears normal), but can be passively dislocated and hence unstable.

- Less than 2% will have a positive Barlow's test
 - 60% will normalise after 1 month
 - 88% will normalise after 2 months

Technique:

- Hip is flexed, and the thigh is adducted while pushing posteriorly in the line of the shaft of the femur, causing the femoral head to dislocate posteriorly from the acetabulum
- The dislocation is palpable as the femoral head slips out
- This diagnosis is confirmed by Ortolani's test

Ortolani's Test

Strictly speaking, the Ortolani's test is the palpable sensation of the gliding of the femoral head in and out of the acetabulum.

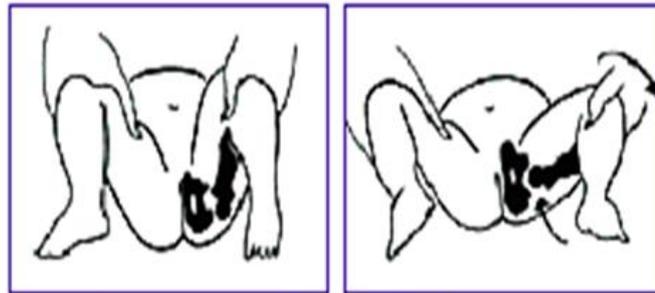
Technique

- The child's hips are examined one at a time
- Flex the infant's hips to 90°
- The thigh is gently abducted and bringing the femoral head from its dislocated position to opposite the acetabulum, hence reducing the femoral head into the acetabulum
- In a positive finding, there is a audible clunk as the hip reduces



Barlow Test

Figure 51 Barlow's and Ortolani's tests



Ortolani Test

Discussion

- The femoral head glides back and forth over a ridge of hypertrophic acetabular cartilage as the femoral head enters and leaves the acetabulum
- Others have interpreted Ortolani's test as a palpable reduction of the child's dislocated hip, whereas Barlow's test is used to describe the provoked dislocation of an unstable hip
- Ortolani's test identifies a dislocated hip that can be reduced in the early weeks of life; a positive test requires *active treatment*
- If the hip remains dislocated (for weeks), limitation of abduction becomes a more consistent finding
- With time it becomes more difficult to reduce the femoral head into the acetabulum and Ortolani's test become negative

Telescoping

- This is pulling and pushing the femur in relation to the pelvis
- One hand stabilises the pelvis and palpates the greater trochanter and the other pulls and pushes the femur
- Any abnormal to and fro motion is called telescoping
- It indicates CDH

Adduction contracture

- Flex the hips to 90° and abduct them
- There should be a normal ROM of 90°
- CDH will appear a 20° or less
- If the condition is unilateral, a direct comparison can be seen

Trendelenburg test

- This is only used in older children and adults
- This assesses the strength of the gluteus medius muscle
- Stand behind the patient and observe the dimples overlying the PSIS
- They should appear level on weight bearing
- Ask the patient to stand on one leg
 - On the normal side, the contralateral side of the pelvis will be elevated
 - If it remains position or descends, it is a positive Trendelenburg test, suggesting the muscle is weak

Treatment

This frequently can be conservative. It was once a plaster of Paris 'brace', keeping the legs in abduction (as a baby, the hips will normally be flexion), holding the femoral heads medially in the acetabula. In milder cases it may be a removable plastic brace worn over the nappies.

In any case it does indicate a weakness of sorts and re-dislocation is possible

Teratogenic dislocations

These are a distinct category, which represent a true dislocation of the hip

- It occurs in about 2% of prenatal hip dislocations
- It is associated with arthrogryposis (a congenital defect of the limbs characterised by contractures in flexion and extension), chromosomal abnormalities, Larsen's syndrome and others
- Typically, the hip is not reducible at birth and is equivalent to an untreated hip in a 3-4-year-old
- Operative treatment is controversial and is associated with multiple procedures to keep the hip reduced

Otto's Pelvis (Protrusio Acetabuli)

This is a deepening of the acetabulum, leading to a flexion deformity. It is idiopathic and is most common in adolescent women. It is often unrecognised and undiagnosed until middle age. X-Rays show deepening of the acetabulum with a consequent thinning of the bone between the acetabulum and the pelvic bowl. Its diagnosis depends upon an X-ray demonstrating Kohler's line

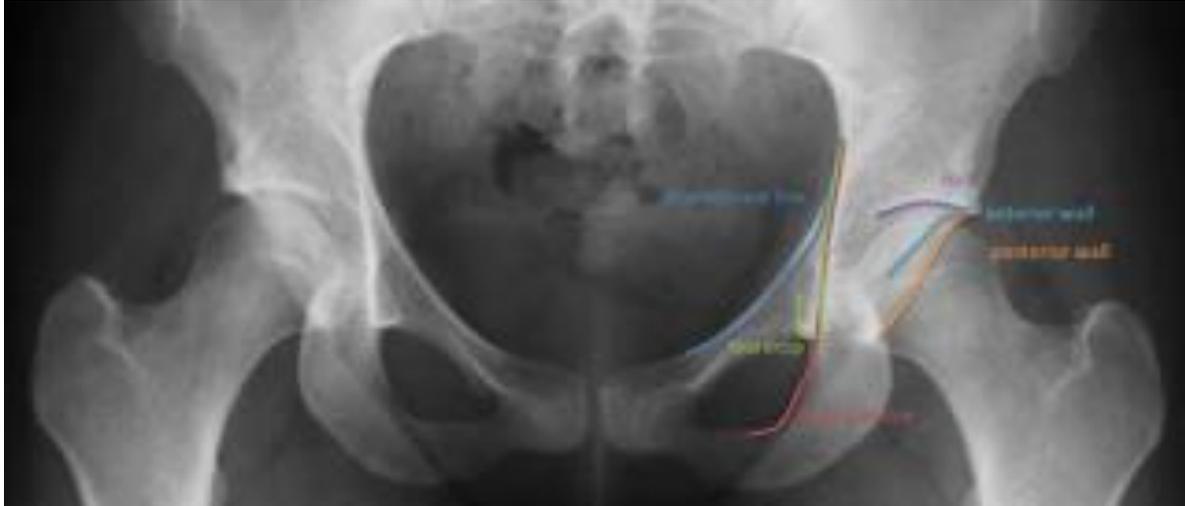
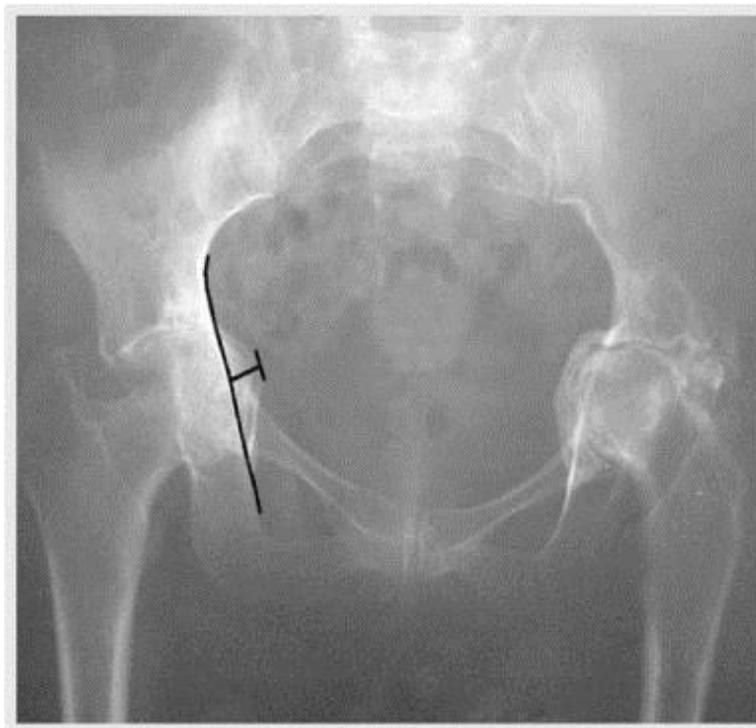


Figure 52 X-Ray showing Kohler's Line

- Iliopectineal line
- Ilioischial line (Kohler's line)



Sotelo-Garza and Charnley method.

Figure 53 Otto's Pelvis - Protrusio Acetabuli using Kohler's line as reference

Clinically:

- The patient is frequently middle aged
- They complain of stiffness of the limb and pain in the groin; worse with weight bearing
- There is a limitation in abduction, external rotation and extension (hence a flexion deformity)
- If unilateral, there will be asymmetry that will easily be picked up at the level of the buttock fold (higher on one side)
- Tests will show positive telescoping and Trendelenburg

Differential diagnosis:

- Bilateral Perthes
 - Hypothyroidism
 - Multiple epiphyseal dysplasia
 - Spondyloepiphyseal dysplasia tarda
 - Sickle cell anaemia
- Unilateral Perthes
 - Septic arthritis
 - Sickle cell anaemia
 - Spondyloepiphyseal dysplasia tarda
 - Gaucher's disease
 - Eosinophilic granuloma
 - Transient synovitis

Clinical presentation

- Pain, often in the knee, effusion (from the arthritis) and a limp
- In the early phase, there is limited abduction of the hip and limited internal rotation in both flexion and extension.
- There will also be an antalgic gait (due to the pain)
- In the later stages there is a Trendelenburg gait

Prognosis

- At least 50% of hips do well with no treatment
- Many do well up to their 5th decade of life when anatomic asphericity leads to degenerative joint disease
- Age is key to prognosis:
 - Less than 6 yoa - outcome is good, regardless of treatment
 - Between 6-8 yoa - results not always satisfactory with containment
 - Over 9 yoa - questionable benefit from containment. Children at this age at initial onset will have poor prognosis and may be expected to have significant symptoms and a restricted ROM

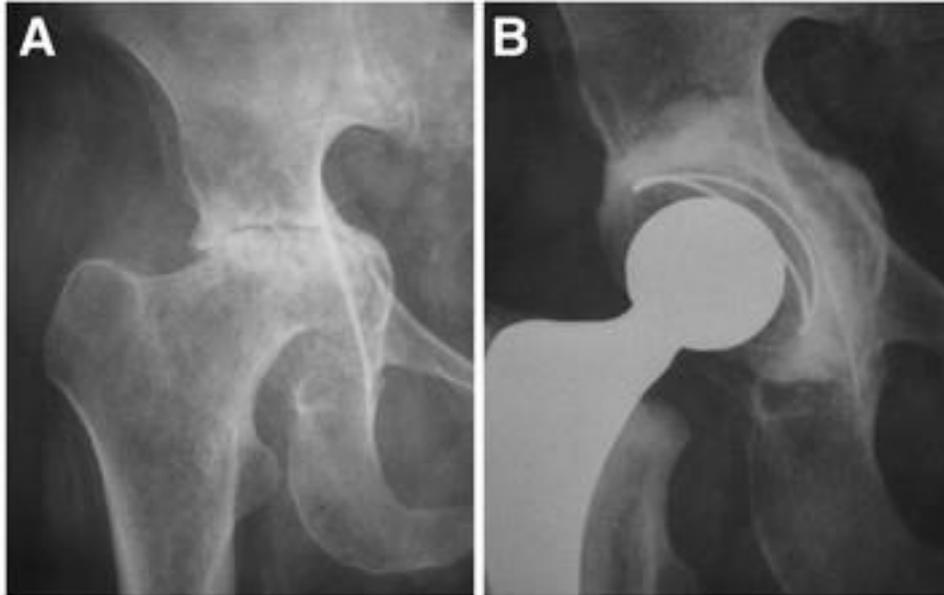


Figure 54 Joint replacement with bone graft

Congruency

- A flat-topped femoral head that is incongruent with the acetabulum has the worst prognosis; the shape of the femoral head after healing
- The degree of epiphyseal involvement
- The ability to maintain the hip motion
- Can predispose to subluxation of the joint

Treatment

- Non-operative
 - Generally, the principles of treatment are maintenance of the ROM and containment of the femoral head through the evolution of healing of the epiphysis
 - Improve the ROM
 - Massage and stretching to reduce muscle spasms and regain abduction
 - In younger children (under 5 yoa) this goal maybe achieved by relief from weight bearing and supervising ROM exercises
 - Some patients may require several weeks of abduction traction
- Containment
 - Theoretically, this is containment of the femoral head in the acetabulum during the repair process leading to a more spherical femoral head and a more congruous joint
 - Containment is not clearly defined, but generally implies 80% coverage of cartilage
 - Prevents extrusion and compression by the acetabular rim
 - Bracing and surgery can achieve containment

Osteogenesis Imperfecta

Osteogenesis imperfecta is also known as: Van der Hoeve syndrome, trias fragilitas osseum, Eddowe"s syndrome, osteopsathyrosis ideopathica of Lobstein, Ekman-Lobstein disease, osteogenesis imperfecta congenita, osteogenesis type III lethalis, brittle bone disease, Vrolik disease

Osteogenesis imperfecta is a group of genetic disorders that mainly affect the bones. The term "osteogenesis imperfecta" means imperfect bone formation. People with this condition have bones that break easily, often from mild trauma or with no apparent cause. Multiple fractures are common, and in severe cases, can occur even before birth. Milder cases may involve only a few fractures over a person's lifetime.

- This is an autosomal dominant disease
- Carriers have a 50% chance of passing OI onto their children

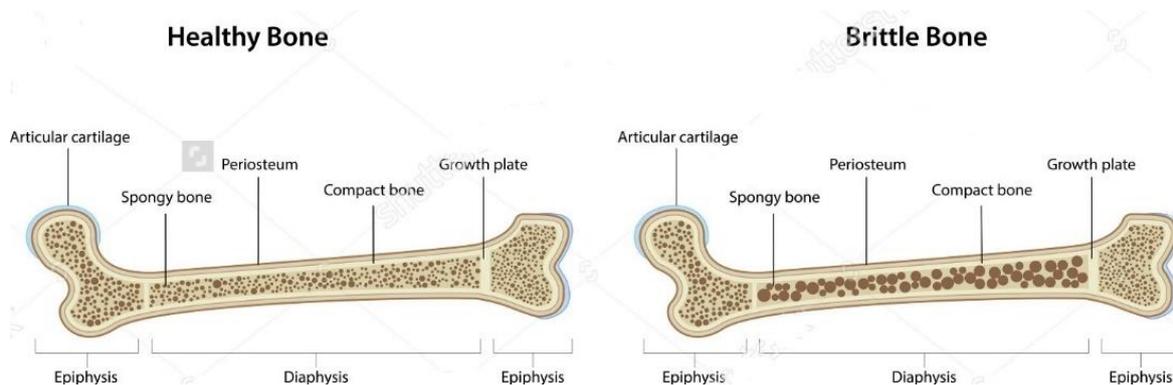


Figure 55 Diagram showing differences between normal bone and that of Osteogenesis Imperfecta

There are at least eight recognized forms of osteogenesis imperfecta, designated type I through type VIII. Although specialists in this, most OI diagnoses are categorized as one of the following types.

- **Type 1:** the mildest and most common form of OI
 - This is the most common form. People with this type can live a normal lifespan.
- **Type 2:** a severe form of the disease, often affecting a foetus in utero
 - This is a severe form that often leads to death in the first year of life.
- **Type 3:** a progressive, deforming type of OI,
 - People with this type have many fractures starting very early in life and can have severe bone deformities. Many people need to use a wheelchair and often have a somewhat shortened life expectancy.
- **Type 4:** a moderate form of the condition
 - This is a moderately severe OI, is similar to type 1, although people with type 4 often need braces or crutches to walk. Life expectancy is normal or near normal.

There are other types of OI, but they occur very rarely, and most are considered subtypes of the moderately severe form (type 4)

The milder forms of osteogenesis imperfecta, including type I, are characterized by bone fractures during childhood and adolescence that often result from minor trauma. Fractures occur less frequently in adulthood.

More severe affects

- Bowed legs and arms
- Kyphosis
- Scoliosis
- Hearing loss
- Heart failure
- Spinal cord problems
- Brain Stem problems
- Permanent deformity



Figure 56 Blue Sclera

People with mild forms of the condition typically have a blue or grey tint to the part of the eye that is usually white (the sclera) and may develop hearing loss in adulthood.

Affected individuals are usually of normal or near normal height.

Other types of osteogenesis imperfecta are more severe, causing frequent bone fractures that may begin before birth and result from little or no trauma. Additional features of these conditions can include blue sclerae, short stature, hearing loss, respiratory problems, and a disorder of tooth development called dentinogenesis imperfecta. The most severe forms of osteogenesis imperfecta, particularly type II, can include an abnormally small, fragile rib cage and underdeveloped lungs. Infants with these abnormalities have life-threatening problems with breathing and often die shortly after birth.

Symptoms

All people with OI have weak bones, and fractures are more likely. People with OI are most often below average height (short stature). However, the severity of the disease varies greatly.

The classic symptoms include:

- Blue tint to the whites of their eyes (blue sclera)
- Multiple bone fractures
- Early hearing loss (deafness)

Because type I collagen is also found in ligaments, people with OI often have loose joints (hypermobility) and flat feet. Some types of OI also lead to the development of poor teeth.

Symptoms of more severe forms of OI may include:

- Bowed legs and arms
- Kyphosis
- Scoliosis (S-curve spine)

Figure 57 Marked deformation of arm, with evidence of fractures in the mid-shaft of the radius and ulna. The elbow is relatively spared, but there is flattening of the head of the humerus



Figure 58 Evidence of marbling of humerus



Figure 59 Scoliosis with osteogenesis imperfecta

Exams and Tests

OI is most often suspected in children whose bones break with very little force. A physical exam may show that the whites of their eyes have a blue tint.

A definitive diagnosis may be made using a skin punch biopsy. Family members may be given a DNA blood test.

If there is a family history of OI, chorionic villus sampling may be done during pregnancy to determine if the baby has the condition. However, because so many different mutations can cause OI, some forms cannot be diagnosed with a genetic test.

The severe form of type II OI can be seen on ultrasound when the fetus is as young as 16 weeks.

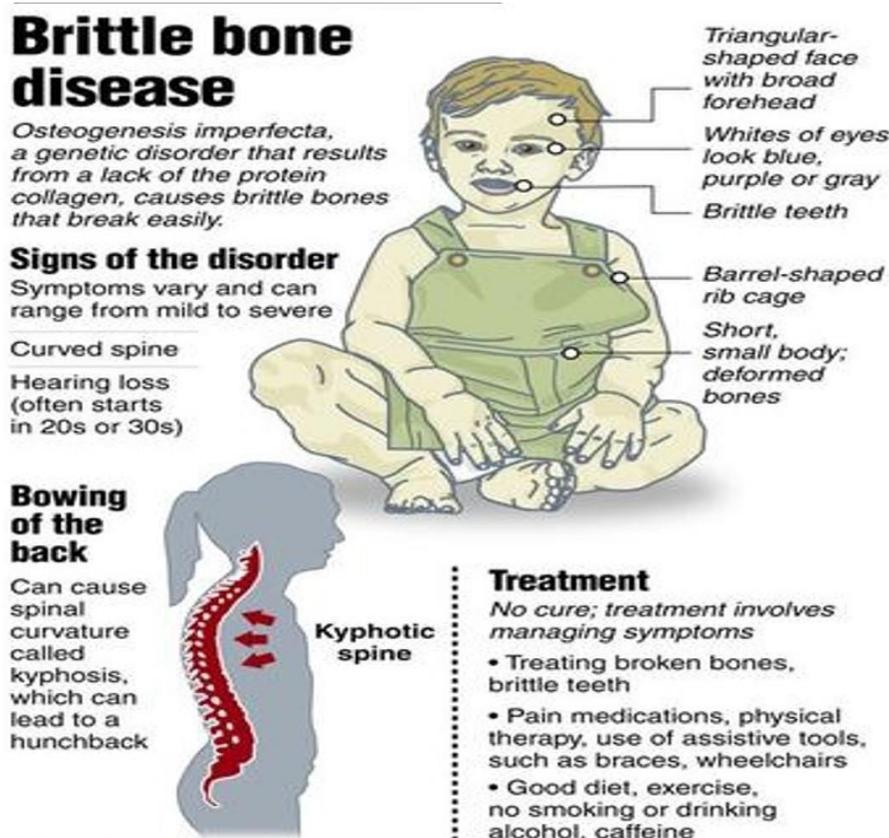


Figure 60 Overview of Osteogenesis Imperfecta

Treatment

There is not yet a cure for this disease. However, specific therapies can reduce the pain and complications from OI.

- Specific therapies reduce pain
- Low impact exercises
- Surgery such as rod implants
- Drugs that help your bones build more calcium

Drugs that can increase the strength and density of bone are used in people with OI. They have been shown to reduce bone pain and fracture rate (especially in the bones of the spine). They are called bisphosphonates.



Figure 61 X-ray showing 'zebra lines' of bisphosphonate treatment. When the drug is delivered, dense bone is formed

Low impact exercises, such as swimming, keep muscles strong and help maintain strong bones. People with OI can benefit from these exercises and should be encouraged to do them.

In more severe cases, surgery to place metal rods into the long bones of the legs may be considered. This procedure can strengthen the bone and reduce the risk for fracture. Bracing can also be helpful for some people.



Figure 62 Intramedullary rod insertion, such that bones grow in a straight line

Surgery may be needed to correct any deformities. This treatment is important because deformities (such as bowed legs or a spinal problem) can interfere with a person's ability to move or walk.

Even with treatment, fractures will occur. Most fractures heal quickly. Time in a cast should be limited, because bone loss may occur when you do not use a part of your body for a period of time.

Many children with OI develop body image problems as they enter their teenage years. A social worker or psychologist can help them adapt to life with OI.

Possible Complications

Complications are largely based on the type of OI present. They are often directly related to the problems with weak bones and multiple fractures.

Complications may include:

- Hearing loss (common in type I and type III)
- Heart failure (type II)
- Respiratory problems and pneumonias due to chest wall deformities
- Spinal cord or brain stem problems
- Permanent deformity

Prevention

Genetic counselling is recommended for couples considering pregnancy if there is a personal or family history of this condition.

Osteoarthritis (O/A)

Osteoarthritis is also known as

- Degenerative joint disease
- Osteoarthrosis
- Hypertrophic osteoarthritis

It is an arthropathy with altered hyaline cartilage and is characterised by loss of articular cartilage and hypertrophy of bone, producing osteophytes. It is the most common articular, beginning asymptotically in the 2nd or 3rd decades in life when almost all people have some pathological change in weight bearing joints and is extremely common by 70 yoa.

Men and women are equally affected, but it appears earlier in men. O/A affects almost all vertebrates, suggesting it appeared with the evolutionary arrival of the bony skeleton. It occurs in whales, dolphins and porpoises, which are supported by water, but not in bats and sloths, which hang upside down. This suggests that O/A is an ancient Palaeozoic mechanism or repair and remodelling, rather than a disease in the usual sense.

Figure 63 Osteoarthritis of Fingers

O/A is classified as:

- **Primary** (idiopathic)
- **Secondary**, due to some cause

Primary, generalised O/A involves:

- Distal and proximal interphalangeal



- joints, causing Heberden's nodes and Bouchard's nodes respectively
- 1st metacarpophalangeal joint
 - Intervertebral disc and zygapophyseal joints in the cervical and lumbar spine
 - Hip
 - Knee

Secondary O/A appears to result from conditions that change the microenvironment of the chondrocyte. These include:

- Congenital joint abnormalities
- Genetic defects
- Infectious, endocrine and neuropathic diseases
- Diseases that alter the normal structure and functioning of the hyaline cartilage (e.g. gout, R/A, chondrocalcinosis)
- Trauma (including fracture) to the hyaline cartilage or surrounding tissue (e.g. from prolonged overuse of a joint, or group of joints, associated with foundry work, coal mining and bus driving)

These principles appear sound, but do not explain all cases of O/A. There some active people who do not have manifest symptoms, whereas there are sedentary people who do have them.

Normal joints have a low coefficient of friction and do no wear out with typical use. Hyaline cartilage is avascular, aneural and alymphatic. It is 95% water and extracellular cartilaginous matrix and only about 5% chondrocytes. Chondrocytes have the longest cell cycle in the body (along with CNS and muscle cells). Cartilage health and function depends upon compression and release in joint movement. The compression squeezes the synovial fluid out from the cartilage into the joint space, capillaries and venules, whereas the release allows the cartilage to re-expand, suck the fluid back in and hence absorb necessary nutrients.

The pathophysiological progress of O/A is progressive. Triggered by changes in the microenvironment, the chondrocytes undergo mitosis and increase synthesis of proteoglycans and type II collagen (the principal structural elements of cartilage). Then synthesis of bone by subchondral osteoblasts increases, presumably prompted by intercellular communication by cytokines between osteoblasts and chondrocytes. With this increased bone formation in the subchondral area, the physical properties change; the bones become stiffer with decreased compliance, and microfractures occur, followed by callus formation, more stiffness and more microfractures.

Metaplasia of the peripheral synovial cells results in periarticular formation of osteophytes or, more correctly, osteochondrophytes, consisting of bone and a mixture of connective tissues with a coating of Fibrocartilage and sometimes islands of hyaline cartilage within the osteophytes. The degree of formation of these spurs varies amongst joints.

Finally, bony cysts (pseudocysts) form in the marrow below the subchondral bone. Bony cysts form from the penetration of joint fluid through the clefts in the hyaline cartilage into the marrow, with a fibroblastic cellular reaction.

Gross pathology includes roughening, pitting and irregularities of the hyaline cartilage surface, proceeding to gross ulceration with focal, then diffuse, areas of complete loss of cartilage, leaving only eburnated (polished) bony surfaces. By the time symptoms appear, synovial proliferation and some mild synovitis are virtually always present.

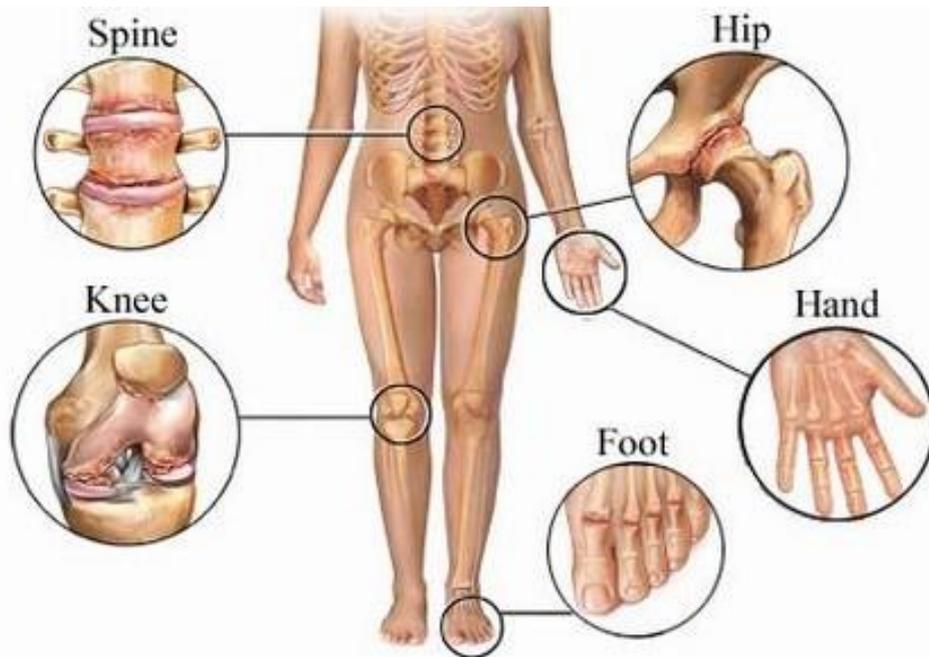


Figure 65 Joints affected by Osteoarthritis

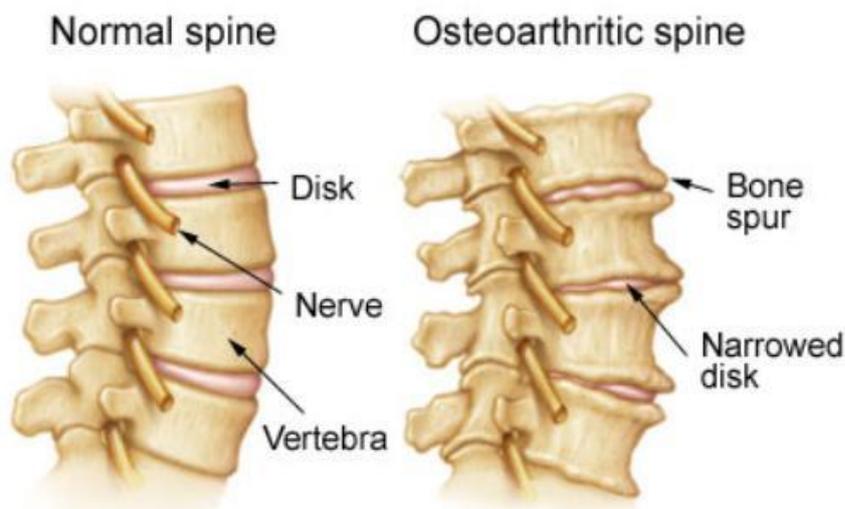


Figure 64 Osteoarthritis of lumbar spine showing osteophytes

Symptoms and signs

- Onset is gradual, usually involving one or a few joints
- Pain is the earliest symptom, usually worsened by exercise and relieved by rest
- Morning stiffness follows inactivity; it lasts 15-30 minutes and lessens with movement
- As O/A progresses, joint motion diminishes, tenderness and crepitus; flexion contractures may develop
- Proliferation of cartilage, bone, ligament, tendon, capsules and enlargement is characteristic of O/A
- Acute and severe synovitis is not expected, but may occur in patients with other diseases (e.g. gout, pseudogout), when they are the primary initiating mechanism of O/A
- O/A of the cervical and lumbar spine may lead to myelopathy or radiculopathy, however the symptoms of the former are relatively mild

- At the disc level, marked thickening and proliferation of the posterior longitudinal ligament results in transverse bars encroaching upon the anterior spinal cord
- Hypertrophy and hyperplasia of the ligamentum flava often compress the posterior spinal cord
- Radiculopathy is less frequent because of the anterior and posterior nerve roots and the common spinal nerves are well protected in the intervertebral foramina, where they occupy only 25% of the available and well-cushioned space

Hip O/A

- Is characterised by gradually increasing rigidity and decreased ROM
- Pain may be experienced in the inguinal area or even in the knee
- Tenderness felt on palpation and passive motion are relatively late signs
- Muscle spasm and contracture add to the pain
- Mechanical block by osteophytes or loose bodies can occur causing locking or catching
- Deformity or subluxation can result from lost cartilage volume, subchondral bone collapse, osteophytes and muscle atrophy

X-Ray

Diagnosis is usually by X-Ray



Figure 66 X-Ray of Osteoarthritis of Hip

X-Rays reveal:

- Loss of joint space
- Formation of osteophytes
- Increased density of subchondral bone
- Pseudocysts in subchondral marrow

Prognosis and treatment

Progression is usually the norm, but occasionally (for no known reason) can stop or reverse

- Rehabilitation techniques for preventing dysfunction
- Avoid soft, deep, chairs or recliners
- Avoid using pillows under the knees, they encourage contracture
- Sit in straight chairs without slouching
- Sleep on a firm bed, with a board

- Perform postural and mobility exercises
- Exercise within one's capacity, including ROM, isometric, isotonic, isokinetic, postural and strengthening. This will maintain healthy cartilage and ROM and help develop stress absorbing tendons and muscles

Glucosamine and Chondroitin

These nutritional supplements have been used with significant results. Glucosamine help rebuild the joint hyaline cartilage and chondroitin helps slow the enzymes causing its breakdown; as does Flexiseq.

Joint replacement

Joint replacements have changed such they are less invasive and less traumatic on insertion.



Figure 67 Hip resurfacing and hip replacement

Joint replacement consists of a prosthesis of a replacement femoral head (metal) and a replacement acetabulum (metal or plastic), they have a life span of about 10 years. Either way, those people have great fun at airports.

Total hip resurfacing arthroplasty is a bone-preserving procedure that helps restore comfort and function to patients' hips damaged by degenerative joint disease (osteoarthritis rheumatoid arthritis and traumatic arthritis) avascular necrosis or developmental hip dysplasia. It is viewed as an alternative to traditional hip replacements for helping patients return to their active lifestyles.

There has been a recent surge in interest in hip-resurfacing procedures. Advocates have pointed to the bone-conserving nature of the procedure and anticipated potential benefits related to post-operative activities and range of motion. Fig 49 illustrates the difference in the amount of bone removed for a resurfacing procedure versus a traditional total hip replacement procedure. In this procedure surgeons replace the acetabulum (hip socket) in much the same way as a conventional total hip replacement but the femoral head is resurfaced rather than removed.

Hip resurfacing is intended for patients with high functional demands for whom traditional total hip arthroplasty would be a poor option because of anticipated failure in the future and subsequent revision surgery.

In these patients the hip-joint surface – the socket side on the pelvis and the top end of the femur which fits into the socket – is destroyed by wear and tear inflammation or prior injury.

This leads to stiffness pain and decreased ability to perform at work or enjoy leisure activities such as sports.

Hip resurfacings may be easier to revise. Because the components (called implants) used in hip replacements and hip resurfacings are mechanical parts, they can — and do — wear out or loosen over time. This typically occurs between 10 and 20 years after the procedure, although implants may last longer or shorter periods of time

Decreased risk of hip dislocation. In hip resurfacing, the size of the ball is larger than in a traditional hip replacement, and it is closer to the size of the natural ball of your hip. Because of this, it may be harder to dislocate. This stance is controversial because several factors can affect the risk of dislocation, such as surgical approach, and the type and size of the implants used.

More normal walking pattern. Several studies have shown that walking patterns are more natural following hip resurfacing compared to traditional hip replacement. These differences in walking are quite subtle, however, and special instruments are needed to measure them.

Greater hip range of motion. Hip resurfacing patients are usually able to move their hips in a greater range of motion than total hip patients. However, certain total hip implants can achieve the same range of motion as hip resurfacings.

Disadvantages of Hip Resurfacing

Femoral neck fracture. A small percentage of hip resurfacing patients will eventually break (fracture) the thighbone at the femoral neck. If this occurs, it is usually necessary to convert the hip resurfacing into a traditional hip replacement.

A femoral neck fracture is not possible with a traditional hip replacement because the femoral neck is removed during this procedure. However, fractures around the implants can still occur with a traditional hip replacement.

Metal ion risk. In hip resurfacing, a metal ball moves within a metal socket. Over time, this leads to the production of tiny metal particles called ions. Some patients may develop sensitivity or allergy to the metal particles, which may cause pain and swelling. Also, there are concerns that the metal particles may increase the risk of cancer, although this has never been proven. Some types of traditional hip replacements also consist of a metal ball and a metal socket and these replacements run the same potential risks. Ask your doctor for more information about metal-on-metal implants.

Hip resurfacing is a more difficult operation. Hip resurfacings are more difficult that total hip replacements for surgeons to perform. As such, a larger incision is usually required for a hip resurfacing.