Pediatric Hip

- Hip joint: articulation between acetabulum and head of femur.
- Acetabulum: triradiate cartilage (3 ossifications centers: ischium, pubis, ileum).
- At birth, the hips are lax, head of femurs are cartilaginous, the acetabulum has more cartilage than bone.

❖ Developmental dysplasia of the hip (DDH)

Spectrum of:

1. Acetabular dysplasia
2. Hip instability
3. Hip subluxation
4. Hip dislocation

Pathology:

Acetabulum → shallow, anteverted deficient anterolaterally, may be deficient posteriorly, false acetabulum.

Hip → delayed ossific nucleus (normally appears at 4-6 months), deformed shape of the head, anteversion of the proximal femur, coxa valgus (neck-shaft angle >135).

Soft tissue:

extraarticular → Tight psoas tendon, Tight adductor muscles, Short abductors.

Intraarticular → Wide lax redundant capsule, Pulvinar (fibro-fatty tissue), Thick elongated ligamentum teres, Enfolded thick labrum inside (Inverted limbus), Thick transverse acetabular ligament, Narrow inferior capsule by the Psoas.

➲ Epidemiology:

- 1-20:1000 neonatal hip instability, 1-2:1000 treated for dysplasia.
- F>M (80% are females).
- Left hip 60%, 20% right, 20% bilateral
- 90% will become stable by the age of two months, leaving 10% with residual hip instability.

➲ Risk factors (9 Fs):

1. Frank breech presentation
2. Female (fetal estrogen increase ligament laxity)
3. First born (tight uterus)
4. Family history
   i. One child has DDH, risk of another child 6%
   ii. At least one parent involved: 12% risk
   iii. Parent and sibling involved: 36% risk

5. Fluid abnormality: Oligohydramnios
6. Feet deformity: metatarsus adductus, Calcaneovalgus feet, CTEV (associated conditions)
7. Facial asymmetry: torticollis (associated condition)
8. Fetal anomalies
9. Faulty habits: swaddling

➲ **Clinical presentation:**

History:

Acetabular dysplasia: asymptomatic

Incomplete dislocation:

Pre-walking → limitation of abduction while changing nappy.

Post-walking → unilateral: limping, tiptoe gait.

Bilateral: LLD, waddling gait.

Physical examination: (in frank dislocation)

Neonatal period (< 6 months) → Ortolani test (reduction test for dislocated hip, includes passive abduction of the hip).

Positive test: if the dislocated hip is reducible (clunk).

Ortolani test is not for dysplastic or subluxated hips (negative), only for completely dislocated hips, so it's a poor screening test (negative Ortolani test doesn't rule out DDH).

This test is negative in congenital hip dysplasia (the hip is irreducible).

Barlow test (down pressure on the hip in flexed and adducted position): harmful test because it induces dislocation. Shouldn't be done except during ultrasound examination.

The test is positive if the hip subluxate or even dislocates on stress.

Infants (>6 months) →

- walking
- limitation of abduction (>20 degrees): most sensitive test
- apparent limb shortening (if unilateral)
- deep long groin crease
- Galeazzi test (or Allis): positive only in unilateral dislocation. The hips and knees flexed to 90°; the test is positive if one knee (the involved side) is lower than the other.

Toddlers → - wide perineum, Trendelenburg gait, lumbar lordosis (in bilateral).

-Limping, LLD (in unilateral).

➲ **Diagnostic tests (confirmatory):**

Ultrasound

Normal α angle is >60°. (Acetabular roof angle).
Normal β angle is <55°. (Labral cartilage roof angle).

Plain radiograph

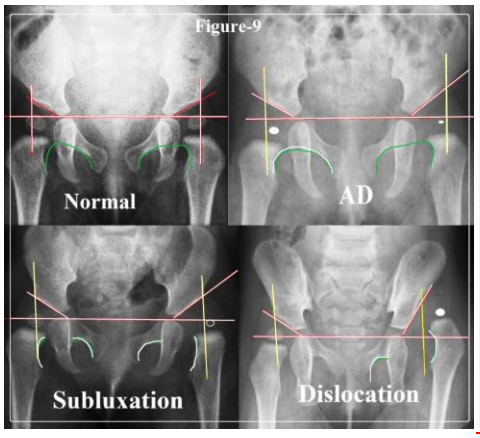
I. Hilgenreiner line: A line is drawn horizontally through each triradiate cartilage of the pelvis.

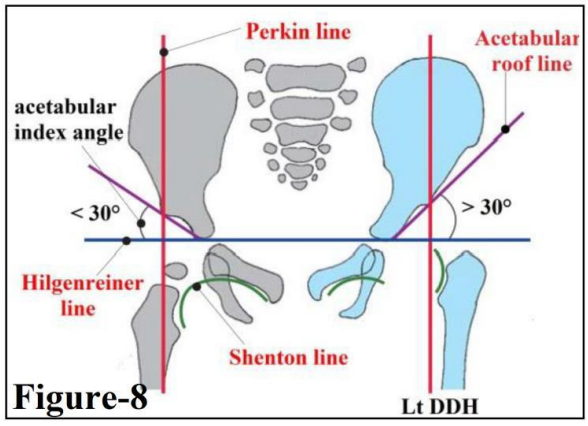
II. Perkin line: Perpendicular line to the H line at the lateral edge of the acetabulum. This will divide the hip region into four quadrants.

The normally ossific nucleus of the head in the inner lower quadrant.
in subluxation→ it is in the outer lower quadrant,
in a dislocation→ in the upper outer quadrant.

III. Shenton line: A continuous arch is drawn along the medial border of the femoral neck and the superior border of the obturator foramen.
Intact in acetabular dysplasia, interrupted in dislocation and subluxation.
Note: False positive in young children with high femoral anteversion.

IV. Acetabular index angle (AIA) (Mirror of DDH)
The angle formed by an oblique line (through the outer edge of the acetabulum and triradiate cartilage) and the H line.
(a) In the infant <3months, a normal value < 30°.
(b) By 6 months of age, the acetabular index decreases to 25°.
In acetabular dysplasia → AIA > 30°.





Screening for DDH:

- Clinical screening (thorough history taking and physical examination) of all newborn infants is necessary, without ultrasonography.

- Ultrasound for patients who are considered high risk (female, family history or breech presentation) despite a normal exam. At the age of 6 weeks, (to give a chance to neonatal unstable hips to be mature and stable (85-90%).
- If no proper ultrasound facility is available, do plain radiograph for the hips at the age of 3 months.

Notes:
- The maximum remodeling of the acetabulum occurs below the age of 18m.
- Dislocated hips per se do not develop AVN.
- In a dislocated hip the affected leg will become shortened, and this will put pressure on the back, increasing the risk of osteoarthritis to the spine.
- The false acetabulum is smaller than a true acetabulum and will develop osteoarthritis between 40-60 years of age if not treated.

➲ **Management**:

The basic principles of treatment are
•Concentric reduction of the dislocation or subluxation. • Hold the reduction until hip stabilizes. • Careful follow-up until skeletal maturity.
<6 months: **Pavlik harness** for 8-12 weeks until AIA < 30.
Position: The hips flexed to 90-100° with 45-60° abduction.
Excessive hip flexion → risk of femoral nerve palsy.
Excessive hip abduction → increased risk of osteonecrosis.
Success rates for Pavlik harness treatment in this setting have been reported at >90% if applied before 2m of age.

>6 months: **Spica casting**.

The recurrence rate of hip problems in the future is 10-20%; therefore, follow-up evaluation until maturity is necessary.

➲ **Complications:**

1-Joint stiffness after open surgery
2-Residual acetabular dysplasia, subluxation, and /or re-dislocation despite adequate treatment. (residual subluxation is ~20%) → FU till maturity.
3-Early osteoarthritis in the hip joint (the 30s)
4-Leg length discrepancy → back pain, functional scoliosis, and knee pain.
5-Genu valgum: Unilateral hip dislocations → fixed adduction deformity in the hip → increased medially directed stress on the knee joint.
6- **Trochanteric overgrowth** → abnormal gluteus function.

7-Avascular necrosis (AVN) of the femoral epiphysis, the most devastating. 0-73%. Extreme abduction, especially when combined with extension and internal rotation, results in a higher rate of avascular necrosis.

The potential sequel of avascular necrosis include:-

Femoral head deformity, acetabular dysplasia, lateral subluxation of the femoral head, relative overgrowth of the greater trochanter, and limb length inequalities, osteoarthritis is a common late complication.

Prognosis

Children with DDH who receive early treatment are generally good.

Teratologic (Congenital) dislocation of the hip (CDH):

Antenatal dislocation in utero and usually stiff irreducible on neonatal examination. Pavlik harness should not be used in CDH.

❖ Legg-Calvé-Perthes Disease

➲ **Definition:** a Non-inflammatory idiopathic AVN of the femoral head in a growing child.

➲ **Epidemiology:** M>F, 10-12% bilateral

➲ **Etiology:**

- idiopathic, **Disruption of the vascularity of the capital femoral epiphysis**.
- Hydrostatic pressure theory: Reactive synovitis → capsular distension → compression on retinacular vessels
- Thrombophilia in 50%.
- coagulopathy (protein C and S def.) in 75%.
- Microtrauma or passive smoking (affects fibrinolysis)

➲ **Risk factors**:

- Male gender (80%)
- Short stature with delayed bone age (usually by two years) (90%)
- Thin, very active child
- Smaller than his age group

➲ **Pathology**: The capital epiphysis and physis are abnormal histologically, with disorganized cartilaginous areas of hypercellularity and fibrillation.

➲ **Clinical presentation**:

Age of presentation: 4-9 years (range from 2 years - late teens)
Limp and pain in the groin, hip, thigh and knee (referred pain)

Physical examination:

- Antalgic gait (due to pain)
- Decreased abduction and internal rotation
- Mild, late LLD (due to femoral head collapse)

➲ **Diagnostic tests:**

Plain radiographs; AP and frog-leg lateral views of the pelvis

Radiographic features: (according to the stage)

- Widening of the joint space and minor subluxation.
- Sclerosis
- Caffey's sign (Salter), a **subchondral fracture (Crescent sign)** on lateral X-ray.
- Fragmentation and focal resorption of the epiphysis.
- Loss of epiphyseal height.
- **Widening of the femoral neck & head (Coxa Magna) (late).**
- Lateral uncovering of the femoral head.
- Metaphyseal cyst formation.
- **Sagging rope sign (Late**): curvilinear sclerotic line running horizontally across the femoral neck. a finding in mature hip with Perthe's disease.
- Acetabular remodeling

Radiographic stages: (Waldenstorm)

1. Necrosis (ischemic stage)
2. Fragmentation (revascularization)
3. Re-ossification (healing)
4. Final stage (remodeling)

Note: In general, necrotic and fragmentation stages last approximately six months each, the re-ossification stage 18 months and the remodeling stage 3 years.

➲ **Herring's (lateral pillar) classification: (most accurate)**

Based on the height of the lateral pillar of the capital epiphysis on the AP view of the pelvis

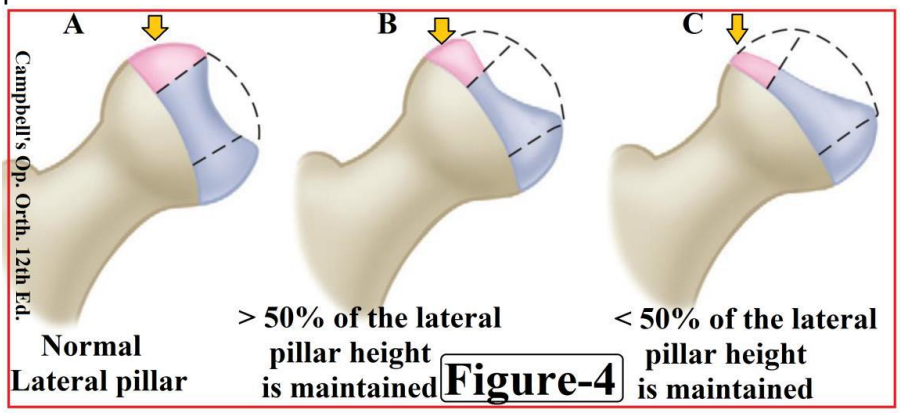


Figure-4

➲ **Poor prognostic factors in Perthes disease:**

1->6 years old
2-Female: mature earlier with less remodeling potential
3-Catterall's "head-at-risk" signs (clinical): Progressive loss of hip motion more so abduction, Fixed flexion deformity and adduction deformities of the hip.
4-Obesity
5-Adduction contracture
6-Progressive loss of hip motion
7-Flexion with abduction
8-Advanced stage of disease at diagnosis (B, C)
9-Advanced grade (loss of containment).
10-Recurrent episodes of stiffness

➲ **Treatment**

Conservative (this is the mainstay of management). NSAIDs, pain killers, physiotherapy.
Containment Surgery: for high riding greater trochanter at the age of >8y.

➲ **Complications of Perthes disease:**

Femoral head deformity:
* Premature physeal arrest patterns
* Osteochondritis dissecans,
* Labral injury
* Late osteoarthritis (5^{th} to 6^{th} decade)

The most important *prognostic factors:*
1- Shape of the femoral head and its congruency at skeletal maturity
2- patient age at onset of disease.

❖ **Slipped capital femoral epiphysis (SCFE)**

Physis layers:

A. **Reserve layer**: resting cartilage cells.
B. **Proliferative zone**: actively growing cartilage cells.
C. **Zone of hypertrophy**: chondrocytes become swollen & vacuolated.
D. **Zone of calcification**: chondrocytes become calcified **.**

➲ **Definition:** displacement through the growth plate in the hypertrophic zone during the rapid growth period of immature hip.

➲ **Epidemiology**

- Most common disorder of the hip in adolescents.
- M>F (12–14 years)
- Unilateral (80%),
- Obese hypogonadal male (adiposo genital syndrome), or excessively thin and tall.

➲ **Etiology:** Idiopathic, but in general, weakness of perichondral ring.

➲ **Conditions that weaken the physis (risk factors):**

- **Endocrinopathies** such as hypothyroidism, GH abnormalities hypogonadism, and hyper- or hypoparathyroidism.

(If the patient is <9 years or >16 years and has a retarded bone age or short stature, consider endocrinopathy).

- **Systemic diseases** such as chronic RF; radiation therapy to the pelvis.
- **Mechanical factors** increase the load across the physis:

(a) Overweight children
(b) Increase in femoral retroversion.
(c) Decreased femoral anteversion and femoral neck-shaft angle.
(d) Vertically oriented physeal plate.
(e) Thinning of the perichondral ring.

➲ **Clinical presentation:**

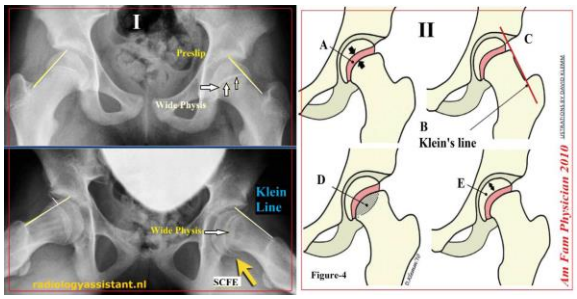
limp and pain in the groin, hip, thigh, or knee region. Symptoms are usually present for weeks- months before a diagnosis made.

Physical examination:

- Abnormal gait (antalgic), waddling gait in bilateral cases.
- decreased ROM (flexion and internal rotation).
- Obligatory external rotation (ER of the hip as the hip is brought into flexion, Walking with the externally rotated foot).

➲ **Diagnostic tests:**

Plain radiographs—Standard AP and frog-leg lateral views of the pelvis
Frog lateral radiographs are more sensitive in detecting an SCFE.
- Widening and irregularity of physis (appears woolly, earliest sign).
- Decreased epiphysis height (slipped posteriorly).
- **The Klein line**, a line tangential to the superior border of the femoral neck on the AP view intersects the proximal femoral epiphysis in a normal hip. In SCFE it fails to intersect the proximal femoral epiphysis (**Trethowan's sign)**
- **Metaphyseal blanch sign** (Steel sign) - Increased crescentic density in the metaphysis due to the overlapping of the metaphysis with the displaced epiphysis. (seen on AP view).



➲ **Classification of SCFE**

Loder classification (based on SCFE stability):

A. Stable (85%) if the patient can weight bear on the involved extremity (with or without crutches).

B. Unstable if the patient is unable to weight bear on the involved extremity→ Sudden onset of pain, often after a fall or injury, Inability to walk or bear weight on the affected leg, Outward turning (external rotation) of the affected leg, The discrepancy in leg length (the affected leg may appear shorter than the opposite leg).

*Risk of AVN in unstable hips 50%; in stable hips 0%.

➲ **Treatment: surgical**

The primary goals of management are stabilization of the slip to prevent further progression and promotion of physeal closure.

In situ screw fixation (The starting point should be positioned anteriorly)

*NO manipulation for reduction because it is associated with AVN.

*For a stable SCFE, a single-screw construct is usually adequate.

*Prophylactic pinning of the other hip in child <10 years, or with a known endocrinopathy

➲ **Complications of SCFE**

- Osteonecrosis (ON):

- 50% risk in unstable slips and 25% in severe slip.
- Screw placement in the posterior and superior femoral neck
- With open reduction.

- Chondrolysis: due to pin penetration of the joint and multiple screw fixation. If penetration is recognized during surgery and corrected, the hip will be safe. Diagnosis indicated by virtually nil range of hip movement, hip pain, and a narrowed joint space. Confirm with MRI.

- **Slip progression**: in 1% to 2%
- **Subtrochanteric fracture:** with entry sites through the lateral cortex and those at or distal to the lesser trochanter.
- **Degenerative joint disease**: 10% of patients with SCFE develop OA.

- **Residual leg length inequality and rotational deformity** (severe slips that may require late corrective osteotomy).

❖ Coxa Vara

Definition: abnormally low femoral neck-shaft angle (<120°).